Varney's Midwifery

SIXTH EDITION

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Preface

What is the purpose of a clinical textbook today? Current research findings, national guidelines, and evidence-based recommendations can be found online and information needed to practice midwifery changes daily, so why read a book?

Midwives, like all healthcare providers, need some core knowledge about anatomy, physiology, the natural course of physiologic events, and signs or symptoms that indicate deviations from normal. Much of this core material is timeless, and it is valuable to have this information gathered together in one text, available for all of us. Providing an overview of this core knowledge in one place is the first purpose of any textbook.

Occasionally, new research and scientific techniques shed light on old knowledge, and then new understandings and clinical applications ensue. For this reason, it is also important to periodically review the basic knowledge and revisit the evidence that supports practice on a regular basis. Updates of the content in this text found in each new edition serve this second purpose as well. Every 5 years or so, the authors who contribute to this text methodically review midwifery practice in light of current scientific findings, and revise this compendium for the profession.

Over time and through subsequent editions, this text documents the growth of the knowledge base that underlies midwifery practice. Thus, the evolution of this text has a third purpose—it provides documentation of how midwifery practice changes over time.

The first edition of this text was written more than 30 years ago. Prior to publication of this text, midwifery students obtained core knowledge from obstetric textbooks, British midwifery books, journal articles, and nursing publications. Midwifery students and midwives in practice still use these sources, yet perhaps the greatest evolution in the last decade has been the rapid expansion of scientific research and explosion of sources of information. Clinicians today are faced with a bewildering number of studies, guidelines, and ever-changing recommendations from different professional organizations. Thus, the fourth purpose of this book is to review the evidence that supports current guidelines and standard therapeutic interventions.

The voices of authors from practices across the nation allow for a robust recognition of practice variations. The advent of evidence-based care enables midwives to provide care so that shared decision making can be a reality. As society has changed, and midwifery has evolved, so has this text evolved. This edition includes a new chapter that presents an overview of the factors that affect an individual's health beyond the biomedical model. Health is profoundly influenced by social and cultural factors, just as it is influenced by genetics and exposure to infectious organisms. The biomedical model is no longer sufficient as a lens for framing determinants of health. The new chapter introduced in this edition, *Midwifery: Clients, Context, and Care*, is dedicated to the recipients of midwifery care and the midwife pioneers who began modeling holistic care long before the value of such care was recognized by the medical community.

These are the reasons why this text is written and rewritten, in an ongoing cycle. Nevertheless, it is equally important that all readers understand that this text is simply a beginning—a starting point for their exploration of the many facets of midwifery. Today, several complementary midwifery texts are available, and those texts can also be used by individuals in their quest to practice safe, quality midwifery care. Midwives must be lifelong learners, and the Internet is an invaluable resource for ensuring that midwives stay up-to-date with the knowledge needed to be a member of this profession. All readers of this text are encouraged to maintain competency in practice by learning how to stay abreast of current evidence via use of online resources. To that end, clinical chapters list suggested websites to facilitate expansion of knowledge and maintain currency.

Varney's Midwifery is one of the primary texts used in midwifery education in the United States. Forecasting the next 3 decades is an impossible task, but this edition and future editions of this text are intended to help midwives on the journey of knowledge acquisition and evolution of midwifery practice.

—Tekoa L. King,
Mary C. Brucker,
Kathryn Osborne, and
Cecilia M. Jevitt

Acknowledgments

A book is a journey. It has a beginning, an end, and many highs and lows throughout the process. Detours occur. A PubMed search for references can be a black hole of wonders to discover. Fortunately for me, midwifery is also a shared journey and writing this text has been a true collaboration in more ways than I can count.

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—Tekoa L. King

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—Mary C. Brucker

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—Kathryn Osborne

Midwives' work is never their own, but rather an extension of all the midwives ever known. My special thanks and gratitude go to Elizabeth Sharp, Sr. Jeanne Meurer, Terri Gesse, Anne Scupholme, Dorothea Lang, Kitty Ernst, Joyce Thompson, and Helen Varney, the midwives who grew me. My children, Maura, Lorna, and Connor, are recognized here for the unique and different understanding each gave me of pregnancy, birth, and motherhood. And finally, there aren't enough thanks in the world for my husband, Bill Rowe, who kept his own company while I researched and wrote for this edition.

—Cecilia M. Jevitt

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PART I

Midwifery

HOLLY POWELL KENNEDY

There has been no greater time of opportunity for the profession of midwifery than the one we are experiencing right now in the early twenty-first century. However, as the midwifery profession looks ahead, it is essential that we review both our past and our present to inform future goals and pathways. Otherwise, we are likely to repeat past missteps.

Varney's Midwifery is a classic compendium of knowledge about the profession and the practice of midwifery in the United States, within the global context of caring for women and their families. This sixth edition adds to the past versions both in describing the "practice" of midwifery and in unraveling the complex and political environments in which the profession is situated. As I mentioned in my introduction to the fifth edition of *Varney's Midwifery*, the authors of these introductory chapters capture these accomplishments and challenges extraordinarily well, leaving the reader with a sense of awe at the tenacity, resilience, and grit of midwives and the profession of midwifery.

The *History of Midwifery in the United States*, *Professional Midwifery Today*, and *Midwifery: Clients, Context, and Care* chapters provide an overview of what we know about the history of the profession in the United States beginning with the introduction of European immigrants into North America to current times. Midwives in the United States have struggled for centuries to be recognized for their knowledge and contributions to the health care of women. Meeting those challenges head on, midwives have developed increasingly sophisticated guiding documents, policies, and laws that facilitate their practice and increase access to their services for women. An important addition to this edition is the recognition that caring for women is increasingly challenging in an ever-increasing complex world. While women may not be more challenging than they have been in the past, midwives have expanded their lens to embrace the full scope of their complexities.

In the few short years since the fifth edition was published, there have been significant advances, which are well described in this section. Notably, in the United States, the

profession of midwifery has faced some challenges in defining who midwives are, especially in terms of educational preparation. The various U.S. professional organizations gamely took their charge from the International Confederation of Midwives 2011 passage of global standards for education, regulation, and association and adapted them to this country's context. Along the way, they were able to develop a collaborative working group and a consensus document on principles of midwifery legislation and regulation for the United States—a major accomplishment that was inconceivable a few years ago.[1] One of the reasons they were able to do this was their earlier collective efforts to develop an evidence-based statement on normal physiologic birth—something that they could agree on! This evolution demonstrates that collaboration, as is so nicely discussed in the *Professional Midwifery Today* chapter, is built slowly and is founded on trust. We must work collectively within the profession or we will fail in our mission to improve the health of mothers, women, and their families.

Interprofessional collaboration is another challenge that is well described in this section. Over the decades, the profession of midwifery has increasingly garnered the respect and trust of our obstetric colleagues. The American College of Nurse-Midwives (ACNM) and the American College of Obstetricians and Gynecologists (ACOG) have published a significant statement about our joint practice:

> *ACOG and ACNM believe health care is most effective when it occurs in a system that facilitates communication across care settings and among providers . . . Ob-gyns and CNMs/CMs [certified nurse-midwives/certified midwives] are experts in their respective fields of practice and are educated, trained, and licensed, independent providers who may collaborate with each other based on the needs of their patients.*[2]

Considering the looming obstetrician shortage, the acknowledgment of midwives' respective expertise is critical to ensure women continue to have excellent health care.

A truly important addition to this text is the *Midwifery: Clients, Context, and Care* chapter. This chapter establishes the foundational knowledge for understanding health disparities in the United States and the role of midwifery in reversing health disparities. Not only does it provide the evidence on social determinants of health and theoretical perspectives, it provides narratives by midwives of color to help the reader understand the context of these issues in the United States.

The evidence is clear that midwifery care, especially when delivered in a continuity model, can produce exceptional outcomes. A serious challenge for the profession is to consider how we position midwifery to become the standard of care for all women. Even women who develop complications deserve respectful care that incorporates the context of their lives and communities, in collaboration with midwives' medical colleagues. As women are clearly now "finding their voices," we must nurture their strength and allow for the healing of so many who have been hurt in the past. This edition of *Varney's Midwifery* helps us as colleagues who are devoted to that mission. As midwives, we hold the trust of each woman who comes to us for care, and it is our responsibility to assure every woman that our care is stellar. Midwifery is strong and is working day by day with women to strengthen their health and, through them, the

health of the world.

References

1. American College of Nurse-Midwives. Principles for model U.S. midwifery legislation and regulation. Available at: http://www.midwife.org/ACNM/files/ccLibraryFiles/Filename/000000005972/US-MERALegislativeStatement2015.pdf. Accessed December 18, 2017.
2. American College of Nurse-Midwives. Joint statement of practice relations between obstetrician-gynecologists and certified nurse-midwives/certified midwives. Available at: https://www.acog.org/-/media/Statements-of-Policy/Public/sop1102.pdf?dmc=1&ts=20170923T2058080347. Accessed June 3, 2017.

CHAPTER

1

History of Midwifery in the United States

ANNE Z. COCKERHAM

The editors acknowledge Helen Varney Burst, who was the author this chapter in the previous edition.

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Introduction

History matters. Midwives cannot care for women, newborns/infants, and families without exploring their histories: Which challenges and opportunities have these individuals faced? What are the effects of the social, economic, familial, spiritual, physical, and political situations in which they live? Likewise, members of the midwifery profession must understand the profession's history, the contextual aspects of midwifery practice, and the ways in which those experiences have shaped the profession today.

The history of midwifery is extensive. The work of all midwives, in the many settings and periods over the course of history, is worthy of both exploration and celebration—but that is not possible in a chapter designed to present an overview. In the United States today, an individual who seeks a path to professional midwifery can become either a certified nurse-midwife, a certified midwife, or a certified professional midwife. The *Professional Midwifery Today* chapter describes the differences in midwifery education and scope of practice for the various types of midwives. This chapter focuses primarily on nurse-midwifery, and more recently certified midwifery, as the profession has developed and evolved in the United States. Readers interested in the broader history of all types of midwives in the United States can access a wide-ranging body of literature that documents the accomplishments of—and challenges faced by—non-nurse-midwives.[1-6] More detailed histories can be found in sources such as *A History of Midwifery in the United States: The Midwife Said Fear Not* by Helen Varney and Joyce Thompson[7] and *Midwifery and Childbirth in America* by Judith Rooks.[8]

To help readers explore key concepts and themes that inform the midwifery profession, this chapter is organized thematically, rather than chronologically. It addresses the eight themes that have underscored the profession since its inception and continue to bear upon the profession today:

1. *Public image.* During the birth of the nurse-midwifery profession, midwives built on their strong reputation associated with thousands of years of midwifery, yet also struggled to overcome a negative public image stemming from the "midwife problem"—a label attached to the profession by the medical establishment (i.e., physicians). Since that time, nurse-midwives have worked to craft a positive image and gain self-determination by organizing, communicating with one another and the public, and developing and maintaining publicly visible and understandable standards. The public image that midwives are less qualified than physicians to care for low-risk women continues to be addressed today, albeit to a much lesser extent.
2. *Legal authority.* Obtaining the legal authority to practice has been a challenge since the introduction of nurse-midwifery in the United States. Although the earliest nurse-midwives attained legal practice authority in only a few locations, through the efforts of dedicated members of the profession, that list of locations gradually expanded to include every state within the United States. Autonomous legal authority to practice is an ongoing focus of the work of the certified nurse-midwives and certified midwives today.
3. *Scope of practice.* At the profession's inception, nurse-midwives' scope of practice

encompassed maternity care, including prenatal care. Some early nurse-midwives also provided public health care to meet the needs of the communities they served. As the profession evolved, midwives gradually added family planning, gynecologic care, primary care, and specific "advanced skills." These additional elements became part of midwives' scope of practice, thereby enabling these clinicians to better meet their patients' needs. The midwifery scope of practice will continue to evolve as knowledge and healthcare technologies are applied to clinical practice.

4. *Care of underserved populations.* The earliest nurse-midwives initiated and maintained services to meet the needs of the most underserved populations during that era. Midwives have continued caring for members of these populations since then. Despite the increased frequency and severity of medical and other risk factors in these populations, midwifery care has been associated with exemplary outcomes. Midwifery continues to be strongly associated with care for underserved populations today.
5. *Place of birth.* Nurse-midwives initially attended births only in women's homes. As more women began to give birth in hospitals, however, midwives shifted their practice to serve women in that setting. Midwives brought characteristic midwifery practices such as natural childbirth and family-centered care into the hospital setting. During the 1970s, with increased consumer demand for options outside the hospital, midwives responded by expanding services for women in home birth settings and birth centers.
6. *Relationships with other healthcare providers.* Although midwives and physicians share a vision of providing optimal health care, the two groups have faced challenges in negotiating professional boundaries in collaborative care. Despite their periodic conflicts, midwives and obstetricians have achieved consensus and provide collaborative care in many settings. Today interprofessional collaboration is increasingly recognized and valued.
7. *Research.* From the profession's beginnings, nurse-midwives have demonstrated their dedication to documenting the outcomes of midwifery care to demonstrate its quality, safety, and effectiveness. Midwifery scholars and clinicians have created a rich heritage of research and theory, encompassing a wide variety of topics related to midwifery and women's health. Midwifery's professional organizations and journals have played an important role in the development and support of researchers and in disseminating the new knowledge generated through midwife-led research.
8. *Midwifery education.* All nurse-midwives and certified midwives are connected to one another through a long lineage of educational programs, beginning with two "first-generation" programs. Although midwifery educational programs have taken advantage of opportunities and faced challenges in providing sustained service to the profession and students, midwifery educators have consistently sought to align curricula with foundational principles of midwifery practice and, at the same time, incorporate innovative educational elements such as early adoption of distance learning and simulation.

Evolution of Nurse-Midwifery's Professional Image and Organization

The practice of midwifery and the term "midwife" have ancient roots. In many parts of the United States, apprentice-trained midwives served local communities with distinction, but as a whole, the profession had no legal status or national recognition during the 1800s and early 1900s. In large part, this lack of status developed as part of changing societal trends and the increasing interest in allopathic medicine that emerged during the first decades of the twentieth century. As a result, midwifery was often misunderstood and lacked power and cohesion as a profession.

Thus, in the 1920s, when visionary leaders in nursing, public health, and medicine sought to revitalize midwifery in the form of *nurse*-midwifery, they faced an uphill battle to craft an image that combined the best of the timeless tradition of midwifery with the modern standards and approaches adopted primarily by public health nurses. This rebranding of the ancient art of midwifery into the profession of modern nurse-midwifery required that nurse-midwives organize, communicate with one another and the public, and develop and maintain publicly visible and publicly understood standards for the profession.

Midwifery Prior to the Twentieth Century

Although birth customs among Native American cultures in the Americas are not extensively documented, what is available consistently shows women being supported by several other women and use of upright positions for giving birth. Prior to the 1800s, midwives were the leaders in the care of women during childbirth, which was then a woman- and home-centric social event. Midwives enjoyed the respect of community members, who greatly valued midwives' experience. Many midwives were trained through an apprentice model, and their knowledge grew from the needs of the community members. Some midwives had more formal education, including midwives in various immigrant groups who trained in Europe.

Midwives' image began to change during the 1800s, as male physicians gradually extended their practice into obstetrics.[9] Medical training and formal education, which were available only to middle- and upper-class men, started to include knowledge about obstetrics in response to the growing body of knowledge being developed in Europe. Physicians in the United States began to recognize that being present at a woman's birth would likely result in a long and lucrative doctor–patient relationship. A prominent Harvard physician wrote in 1820:

> *Women seldom forget a practitioner who has conducted them tenderly and safely through parturition. . . . the practice of midwifery becomes desirable to physicians [and] ensures to them the permanency and security of all their other business.*[10(pp79-80)]

Use of hospitals for labor and birth, forceps, obstetric anesthesia, and techniques to conquer puerperal fever were widely adopted at the beginning of the twentieth century. These obstetric practices were not accessible to midwives, whose knowledge gradually became viewed by the

public as outdated.[9] At the time, women were not considered to have the mental capacity for higher learning and were excluded from admission to organizations devoted to higher learning. Historian Judy Litoff writes that by 1800, the United States had four medical schools but women were "systematically excluded from attaining a medical education at the precise time when knowledge of the scientific advances in obstetrics would have enabled them to become more competent midwives."[2(p9)] Historian Laurel Thatcher Ulrich further argues that midwives' decline occurred because "Midwives were 'experienced,' whereas physicians were 'learned.' Because the base of the midwives' experience was shared by all women, their authority was communal as well as personal."[11(p134)]

Furthermore, midwives were generally isolated from one another and lacked national or local organizations, journals, or other means of communicating about the profession. European immigrant communities included well-prepared midwives, but these immigrant midwives often did not speak English or have access to the existing healthcare system.[12] African American midwives in the rural South usually could not gain access to formal education. Other midwives serving their communities faced similar challenges, including white "granny" midwives in Appalachia,[2] midwives in the Ozarks of southern Missouri,[13] "parteras" of Spanish descent in California and the Southwest,[14,15] and sanba midwives from Japan in Hawaii and the Pacific Northwest.[16] In summary, as the nineteenth century drew to a close and the twentieth century began, midwives' influence and activities were becoming increasingly more limited across the United States. Lack of access to formal education and scientific developments, no licensure or organization, lack of a means of communication as a profession, and changes in public perception all combined to prevent midwives in many parts of the United States from having access to the official healthcare system during this era.

The "Midwife Problem" During the Early 20th Century

As traditional midwives' sphere of influence continued to shrink, a large and powerful group of physicians, nurses, social reformers, and public health officials put forth the claim that the United States had a "midwife problem."[17] Supporters of this theory argued that uneducated, unregulated midwives were the main cause of maternal and infant morbidity and mortality rates that far exceeded those of most European countries.[2,18] For example, nurse Carolyn Van Blarcom wrote in the *American Journal of Public Health* in 1914 that "it is due in great measure to the ignorance and neglect on the part of midwives that many babies become blind from . . . ophthalmia neonatorum."[19(p197)] Physician Anna Rude's 1923 article in the *Journal of the American Medical Association*, titled "The Midwife Problem in the United States," described the "inadequacy of our laws governing midwives, which contain neither uniform provisions nor required standards."[17(p1)] Some physicians argued for the total abolition of midwives and for legal prosecution of violators. Others, including prominent public health officials, promoted close regulation of midwifery and control over midwives' education, rather than eliminating midwifery practice.[2] Within the context of this "midwife problem," the medical, nursing, and public health communities moved to consolidate power and authority over midwives and childbearing.

In reality, those who argued that midwives were the sole cause or even the most significant cause of poor obstetric outcomes were incorrect. From 1915 until the mid-1930s, physicians' use of obstetric interventions, surgeries, and medications increased and a remarkable rise in maternal and infant mortality followed. A committee of the New York Academy of Medicine reported in 1933 that the maternal death rate for surgeons' practice was 9.9 per 1000 live births, and that for obstetricians' practice was 5.4 per 1000 live births.[2] By comparison, home birth midwives who received public health instruction had the lowest maternal death rate—1.4 per 1000 live births.[2] The true causes of poor obstetric outcomes in the United States were actually quite complex, and included lack of prenatal care that could have identified treatable problems; low standards of education for all providers of maternity care, including physicians; intervention by physicians with unproven and often dangerous obstetric techniques; and high rates of puerperal infections due to patient-to-patient contamination among the growing number of women giving birth in hospitals.[20]

During the 1920s, a handful of nursing, medicine, and public health leaders proposed a solution to "the midwife problem": the training of public health nurses as midwives. This new profession would need to carefully craft its public image as well-educated professionals; America's first nurse-midwives strived to meet that goal from the 1920s and beyond. In 1925, Mary Breckinridge opened the first nurse-midwifery clinical service in Kentucky, called the Frontier Nursing Service (FNS) (**Figure 1-1**). At FNS, British-trained nurse-midwives introduced professional maternity and public health care to women and families in isolated mountain communities, followed strict medical directives that had been written by a medical advisory group, and maintained high-quality records to document the outcomes of their care. On the East Coast, the Maternity Center Association (MCA), a group of nurse-midwives in New York City, provided and supervised professional maternity care to impoverished women whose communities had suffered from high rates of morbidity and mortality, maintained clearly articulated collaborative relationships with physicians, and carefully documented the results of their care.



Figure 1-1 A nurse-midwife of the Frontier Nursing Service on a home visit in Kentucky, circa 1950. Reproduced with permission from Frontier Nursing Service, Hyden, Kentucky.

1955: A Professional Organization for Nurse-Midwives

Building on the work of the FNS, MCA, and others between the 1920s and the 1950s, nurse-midwives recognized that a crucial element for the success of the profession would be the establishment of a professional organization. Several attempts during this period laid important groundwork, including the Frontier Nursing Service–focused American Association of Nurse-Midwives (AANM) and the nurse-midwifery section within the National Organization of Public Health Nursing (NOPHN). For various reasons, including disagreements about the role of physicians, optimal practice settings for nurse-midwifery, and inclusion criteria for the profession and professional organizations, neither the AANM nor the NOPHN were deemed to be the ideal vehicle for a unifying professional organization for nurse-midwives.[21]

Ultimately, the foundational work would come to fruition with the establishment of the American College of Nurse-Midwives (ACNM). Beginning in 1954, Sister M. Theophane Shoemaker, director of the Catholic Maternity Institute (CMI) in Santa Fe, New Mexico, chaired the Committee on Organization. Within a few months of beginning their work, Committee on Organization members articulated objectives and organizational structures, defined nurse-midwifery, set educational standards for nurse-midwifery schools, designed and mailed a questionnaire to prospective members, wrote and mailed two of the eventual six *Organization Bulletins of the Committee on Organization*, and organized upcoming meetings.[22,23] At a May 1955 meeting, the Committee on Organization voted unanimously to proceed with formation of the American College of Nurse-Midwifery. The incorporation took place on November 7, 1955, in New Mexico; members selected that location because it was

one of the few states in which nurse-midwives were practicing and the incorporation process was relatively straightforward.[24] See **Figure 1-2**.



Figure 1-2 Original signators of the *Articles of Incorporation* of the American College of Nurse-Midwifery. From left to right: Sister Theophane Shoemaker CNM, MMS; Pat Simmons CNM; Ann Fox CNM; Sr. Judith Kroska CNM, MMS.

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The new professional organization provided a vehicle through which nurse-midwives could communicate with one another and strategize about how to move their professional interests forward. Rita Kroska designed the organization's seal in 1955 (**Figure 1-3**).[25] The large shield is encircled by a ribboned band containing the inscription, "AMERICAN COLLEGE OF NURSE-MIDWIVES, NEW MEXICO, Nov. 7, 1955. *health and wellbeing of family life, particularly the mother and infant.*"[25] Placing a hyphen between the words "nurse" and "midwife" is a critical piece of punctuation, as it makes "nurse-midwife" a unique, singular profession.



Figure 1-3 The seal of the American College of Nurse-Midwives.

Reproduced with permission from American College of Nurse-Midwives.

Just a few days after ACNM was incorporated, the "First Convention" was held in Kansas City with 17 nurse-midwives in attendance, representing 8 states. The next month, December 1955, the ACNM published the first issue of the *Bulletin of the American College of Nurse-Midwifery*, which included a description of the "prevailing spirit" of the organization:

> *Of all that occurred at the first convention, you would have enjoyed most the fine spirit of unity and enthusiasm which was characteristic of the meetings. The opportunities for frank discussions in the informal, friendly meetings did much to supply the encouragement necessary for launching a new organization full of promise. Our Motto: VIVANT! Let them live!*[24(p4)]

In the second issue of the *Bulletin*, ACNM President Hattie Hemschemeyer reinforced the importance of organizing, communicating, and developing and maintaining professional standards. After reporting that the ACNM had already grown to 124 members, Miss Hemschemeyer wrote:

> *Membership carries with it the need for deliberation and thoughtful action on our part. The college must select carefully the work it undertakes and then do well the work it has undertaken. We need to work with dedication and conviction. . . . We have a pioneer job to do, and if we work as well and as constructively in a group as we have in the past as individuals, we can help to improve professional*

competence, provide better service and educational programs, and make fuller use of resources. The future looks bright.[26(pp5-6)]

A Professional Journal: 1950s to the Present

Beginning with the first issue of the *Bulletin of the American College of Nurse-Midwifery* in 1955, the professional midwifery journal has played a pivotal role in developing nurse-midwifery as a profession. As early as January 1957, a *Bulletin* article informed readers of the importance of participating in the publication of salient articles.[27] The article noted the crucial role that the professional organization and its journal would play in advancing nurse-midwifery as well as remaining true to its ideals:

> *Now that we have a forum, we are in a position to express our spontaneous ideas. . . . The contribution of the nurse-midwife cannot be borrowed from other disciplines. Its uniqueness must be spontaneously defined by nurse-midwives. Then we, as a college, can present these ideas with conviction because they are our own. Responsibility for our growth and influence as a professional group rests squarely on our membership and our leadership.*[27(pp1-2)]

Over time, changes in the name of the national midwifery journal reflected the broader changes in the professional organization and midwifery practice. Between 1955 and 1968, the journal was called the *Bulletin of the American College of Nurse-Midwifery*. When ACNM changed its name in 1969 to the American College of Nurse-Midwives, the *Bulletin* became known as the *Bulletin of the American College of Nurse-Midwives* until 1972. From 1973 to 1999, it was published as the *Journal of Nurse-Midwifery*. In 2000, in response to the expansion of midwifery scope of practice, the journal became *Journal of Midwifery & Women's Health* and remains so today.

Education Program Accreditation

When nurse-midwives founded ACNM in 1955, they recognized the need to establish and maintain high standards for nurse-midwifery education. Objective (d) of Article II of the first Articles of Incorporation was "To plan and develop, with the assistance of allied educational groups, educational programs in nurse-midwifery that will meet the qualifications of the profession."[28(p146),29] By 1960, nurse-midwives were in deep conversation about accreditation for midwifery education programs. Citing the recent growth in midwifery programs, Vera Keane argued that ACNM was the "logical group to establish and support" accreditation. Furthermore, Keane emphasized, accreditation is ideally a "cooperative enterprise" between "accreditors with long range vision and high ideals" and educators who are "willing to share their concerns fully and honestly." According to Keane, benefits of the accreditation process include accreditors' ability to objectively view curricula and assist educators in producing the best nurse-midwives.[30(pp39-41)]

The accreditation of nurse-midwifery education programs was well established by the early

1970s. In 1982, the ACNM Division of Accreditation (DOA) was first recognized by the U.S. Department of Education (USDOE) as a national accrediting body, now named Accreditation Commission for Midwifery Education (ACME). This recognition through accreditation continues today for all midwifery education programs, including those that offer master's and doctoral education, and assures that the curricula pursued by these programs meets educational standards.

Certification of Nurse-Midwives

National certification of individual nurse-midwives was another crucial step forward in establishing publicly recognized standards within the profession. Certification assures consumers that the midwife has appropriate credentials for practice. In 1971, ACNM members voted to approve a bylaws change that included requiring national certification for graduates of accredited nurse-midwifery education programs. In her article about the change, Joyce Cameron detailed the historical background of certification, responded to questions about the purposes of certification, and addressed how national certification affected licensure for nurse-midwifery and the process for national certification.[31] In addition, her article described how certification would be handled for nurse-midwives qualified prior to 1971.

In 1994, ACNM responded to requests from state regulatory agencies to take a leadership role in setting the standards for the credentialing of professional midwives who did not have a nursing background. Using the same criteria specified for nurse-midwifery education programs, the ACNM DOA developed criteria for basic midwifery education programs for non-nurse midwives, and the ACNM Certification Council committed itself to the testing and certification of graduates from ACNM DOA-accredited midwifery programs, who would receive the credential of Certified Midwife (CM).[32] These midwives meet the same endpoint academic and clinical objectives as nurse-midwives. The first education program for non-nurse (direct-entry) midwives preaccredited by the ACNM DOA was established in 1996.

In May 2001, the U.S. Department of Education renewed its recognition of the ACNM Division of Accreditation for preaccreditation and accreditation of nurse-midwifery education programs, and also recognized the expansion of the scope of its activities to include preaccreditation and accreditation of direct-entry midwifery education for the non-nurse.[28] In 2008, the name of the DOA changed to the Accreditation Commission for Midwifery Education (ACME).

Summary

Midwives have both benefited from and struggled with the image associated with the word "midwife." After millennia of experience attending most women's births, midwives' image began to suffer in the late nineteenth century, and the early twentieth century witnessed a well-publicized campaign against the "midwife problem." The new, hybrid profession of nurse-midwifery worked against the negative images, and members of the profession have continued this fight through organization, communication, and developing highly visible professional standards.

Evolution of Legal Practice Authority for Nurse-Midwives

The legal authority to practice nurse-midwifery has been one of the most important determinants in the expansion of midwifery practice in the United States. Initially, nurse-midwifery practice was allowed in only a few jurisdictions. Over time, however, the diligent work of many professionals contributed to the expansion of legal practice and to the involvement of midwives in legislative issues.

Gradual Expansion of Legal Practice Authority: 1920s to 1980s

For the first four decades after the inception of nurse-midwifery in the United States, nurse-midwives had clear legal practice authority in only a limited number of locations, including New York City (Maternity Center Association), Kentucky (Frontier Nursing Service), New Mexico (Catholic Maternity Institute), and a few other areas. Although nurse-midwives were in demand as maternity nursing educators, nursing service staff members, supervisors in hospital obstetrics departments, and consultants in federal and international health organizations, nurse-midwives could legally practice clinical nurse-midwifery in only a few areas for the first several decades of the profession's existence.[33]

The tide began to turn during the 1960s. Nurse-midwifery leaders assessed where nurse-midwives were practicing and demonstrated their commitment to educating midwives about legal practice authority issues. The American College of Nurse-Midwifery carried out a survey in 1961 and 1962 to investigate various aspects of midwifery practice, including the number of nurse-midwives actively practicing clinical midwifery. Results of the survey indicated that 66 of the 213 respondents (approximately 31%) were providing direct midwifery services at that time.[34] A number of articles throughout the 1960s informed *Bulletin of the American College of Nurse-Midwifery* readers about national and state-based legal practice issues.[35,36]

During the 1970s and 1980s, nurse-midwives made major progress, both in articulating official commitment to gaining and maintaining legal practice authority and in expanding the number of jurisdictions in which nurse-midwives could practice. In 1974, the ACNM Legislation Committee participated in the Workshop on the Legal Status of Nurse-Midwifery and subsequently prepared the *Position Statement on Nurse-Midwifery Legislation*. The statement lamented the "barrier[s] to the optimal growth and development of nurse-midwifery due to serious ambiguities in the legal base for practice" and recommended "separate statutory recognition . . . as the basis for nurse-midwifery practice. To the extent possible, this legislation should be uniform throughout the United States and its jurisdictions."[37(p24)]

Keeping Nurse-Midwives Informed About Legislative Issues

In 1976, the *Journal of Nurse-Midwifery* published a theme issue that focused on midwifery-related legislation. The report contained positive news as well as cautionary notes. The authors concluded that nurse-midwifery at that time was "On the whole, a fairly open field.

With few exceptions, laws of states and jurisdictions are not restrictive or clearly prohibitive of the development of nurse-midwifery." However, despite "the trend . . . toward passage of enabling legislation in most states, it is also evident that in many of those states nurse-midwives are still not practicing and that in others, only one or two are employed."[38(p19)]

In 1984, the *Journal of Nurse-Midwifery* again devoted an issue to the legislative status of nurse-midwifery practice in each state, the District of Columbia, Puerto Rico, and the Virgin Islands. In the decade that had passed since its first survey of legal practice authority legislation, much had changed, yet challenges persisted. Nancy Cuddihy described both:

> *The good news is that Certified Nurse-Midwives have established a legal basis for practice in all but two jurisdictions in the United States. The bad news is that this legal basis for practice is a patchwork collection of various nursing and medical practice acts, few of which allow for independence or self-regulation of nurse-midwifery as a profession. This legislative jumble is manifested in the fact that there are five different types of jurisdictional agencies that are currently empowered to regulate nurse-midwives. This lack of uniformity of legislation has been and will continue to be a problem for the profession in attempting to translate nurse-midwifery to legislatures, executive agencies, and other institutions that make public policy.*[39(p55)]

By the early 1990s, further legislative and regulatory changes had occurred and the Political and Economic Affairs Committee of the ACNM assembled a legislative update. In 1992, the *Journal of Nurse-Midwifery* published this report in two parts.[40-42] Organized by ACNM region, the report included information about practice-regulating agencies, statutes and regulations, prescriptive authority, continuing education requirements, insurance reimbursement for midwifery services, and regulation of birth centers.[41,42] The 1992 update included positive news about "significant improvement toward autonomy in CNM practice," including some form of prescriptive authority in 36 states (compared with 18 states in 1984) and mandated third-party reimbursement in 27 states (compared with 14 states in 1984).[40(p159)]

Prescriptive authority for nurse-midwives gradually followed. Today, nurse-midwives have been granted authority to write prescriptions in all 50 states and in the District of Columbia. Aspects of that prescriptive authority were profiled in the *Journal of Midwifery and Women's Health* by Kathryn Osborne in 2011 and 2015.[43,44]

Summary

After the introduction of nurse-midwives in the United States in the 1920s, the legal authority to practice nurse-midwifery was available in only a few areas of the country. Following decades of legislative action, nurse-midwives now have the legal authority to practice and prescriptive authority in all parts of the United States.

Evolution of the Midwifery Scope of Practice

Another factor influencing the image of the profession has been midwifery's response, via shifts in scope of practice, to the changing needs of the women and families whom midwives serve. Although today's midwives provide care to women across the lifespan, including nonmaternity care such as family planning, gynecologic, and primary care services, historically nurse-midwifery practice was more limited. The first nurse-midwives in clinical practice primarily combined maternity care with traditional public health nursing services, but midwifery scope of practice has been evolving ever since.

Maternity Care During the Early Twentieth Century

When nurse-midwifery was introduced, most nurse-midwives in clinical practice cared for women only during pregnancy, labor, birth, and the puerperium.[35] During the first 2 decades of the twentieth century, inadequate maternity care was linked to the high rates of maternal and infant morbidity and mortality. During this period of increased interest in maternal and child health, the federal Children's Bureau was established (in 1912) and researchers began to gather evidence that conclusively linked high-quality prenatal care with reductions in maternal and infant mortality.[45]

From the earliest days of the establishment of prenatal care, the elements of nurse-midwifery–led prenatal care have remained remarkably consistent. Maternity Center Association prenatal care was described as including "instruction in healthful living. . . . The nurse-midwife helped each woman to evaluate her diet . . . on the basis of the recommended allowances of the National Research Council—and suggested ways to supplement deficiencies with foods acceptable to her and her family."[46(p29)]

Similarly, "Mothers' Classes" during the 1940s at the Catholic Maternity Institute in Santa Fe included discussions of "preparation of the body (physical and psychological changes); hygiene of pregnancy: diet, rest, clothing, cleanliness, sex relationships, varicose veins, backache, heartburn, breast enlargement, danger signals, need of medical care; . . . [and] preparation of husband, children . . . and supplies."[47(pp182-183)]

Public Health Services During the Early Twentieth Century

Midwifery has always had a strong link with public health. When nurse-midwifery was introduced in the United States, public health nurses were the logical choice to be prepared as nurse-midwives because public health nurses already included maternal–child health care as part of their services. Additionally, the National Organization for Public Health Nursing (NOPHN) was one of the first nursing organizations to recognize nurse-midwifery.[45] Public health nursing practice constituted a logical extension of some nurse-midwives' work because they were often practicing in areas of great medical need. For example, MCA public health nurses traveled throughout the project area, performing door-to-door case finding, assessing home environments of women who planned a home birth, and encouraging women to seek prenatal care. MCA public health nurses also coordinated with other community services that

pregnant women might seek, such as milk stations, settlement houses, and churches.

Like her MCA colleagues, Mary Breckinridge recognized soon after she founded the Frontier Nursing Service that the FNS would not be successful if it did not also provide comprehensive public health and general primary care services to its rural population.[48,49] An area of particular public health concern for FNS was parasite control and its corollary, sanitation. Worm infestations were rampant in children throughout the area and were a frequent cause of poor health. Slowly, the FNS nurses urged the mountain people to build sanitary toilets and chlorinate infected wells. Likewise, great emphasis was placed on vaccinations and other preventive services.[48] See **Figure 1-4**.



Figure 1-4 A nurse-midwife of the Frontier Nursing Service providing public health care during a home visit.

Courtesy of Frontier Nursing University.

Meeting the Needs of Non-Childbearing Women: 1950s to the Present

By the 1950s, societal factors and scientific advances had converged, and nurse-midwives began to consider expanding their scope of practice. Nurse-midwives' forays into family planning began in 1958, when FNS nurse-midwives served as clinicians in a study of

combined oral contraceptive pills. Under the auspices of researcher John Rock's study, FNS nurses administered pills and incorporated care of study participants into their usual work. After the study was completed and the oral contraceptive pill was approved for use in the United States, the provision and management of birth control pills was integrated into the role of the FNS nurse-midwife. Shortly thereafter, the FNS nurse-midwives were taught to insert intrauterine devices and manage follow-up care.[50]

Although the FNS nurse-midwives were able to expand their role to include family planning services, the addition of family planning care to midwifery practice in other areas was more difficult. Helen Varney Burst recalled as a Yale University nurse-midwifery student in 1962 that:

> *. . . family planning consisted of surreptitiously passing to a woman, literally under the bed covers, a Planned Parenthood pamphlet with the address of the clinic nearest her home circled. I was told that it was "against policy" or "illegal" to disseminate family planning information, much less provide contraceptive methods, in the New York City municipal hospitals.*[51(p527)]

That situation changed in 1965 with the work of Shirley Okrent, a nurse-midwifery student at Kings County Hospital/State University of New York Downstate Medical Center. Okrent was reported to her supervisors for talking with her postpartum patients about family planning in spite of the topic not being part of nurse-midwifery education curriculum or recognized within the nurse-midwifery scope of practice at that time. Fortunately for Okrent, the chair of the Department of Obstetrics and Gynecology had been searching for a nurse-midwife to staff a family planning clinic. During Okrent's remaining education and after her graduation, she learned about intrauterine device placement, diaphragm fitting, and oral contraceptive pill counseling and prescription.[51] Okrent published accounts of her experiences in 1966 and 1970 issues of the *Bulletin of the American College of Nurse-Midwifery*.[52,53] She also published clinical guides on oral contraception, intrauterine devices, and the diaphragm.[54,55] At the time, Okrent's work in expanding midwifery scope of practice met with mixed reviews. Some nurse-midwives resolutely insisted that nurse-midwifery care should end at the 6-week postpartum visit; others opposed any family planning except abstinence; and some worried that family planning work would include abortions.[51]

By the late 1970s, nurse-midwifery scope of practice had expanded further to include other aspects of gynecologic care, initially called interconception care. The 1978 ACNM definition of nurse-midwifery practice added "and/or gynecologically" to its list of management areas. The first edition of Varney's textbook, *Nurse-Midwifery*, in 1980, included one section titled "Management of the Interconceptional Period" and described care during that period as "the primary health care of women who are between menarche and menopause as it relates to the female reproductive system [including] women who not only are not pregnant but who may or may not wish to be pregnant."[51(p528)]

During the 1980s and 1990s, nurse-midwives began screening for gynecologic problems and offering care for women who had sexually transmitted diseases.[49] Midwives also added the care of perimenopausal and menopausal women because, as Mary Barger pointed out, "As the

women whose babies midwives delivered and provided with preventive gynecology care continued their lives, they wanted to keep on seeing midwives."[56(p88)]

During the 1990s, increasing numbers of midwives began providing primary health care to women, particularly in rural and underserved areas. A 1993 ACNM survey found that nurse-midwives were managing acute and chronic conditions such as bronchitis, asthma, colds, ear infections, anemia, mild hypertension, diarrhea, and dermatitis. Nurse-midwives were also addressing physical, sexual, and emotional abuse, as well as drug and alcohol dependence.[57] Mary Ann Shah, the editor of the *Journal of Nurse-Midwifery*, expressed support for the ACNM's 1992 position statement that nurse-midwives are primary care providers. Pointing to the high prevalence of heart disease in women as well as women being "lulled into a false sense of security by the reassurance that their breasts and reproductive organs remain cancer free," Shah argued for nurse-midwives "to make absolutely certain that we are competent to provide the most comprehensive primary care possible."[58(pp185,187)]

Systematic Processes for Defining and Expanding Scope of Practice

Throughout the history of the profession, nurse-midwives have developed systematic processes to define and evaluate their scope of practice and thoughtfully add new elements. Since 1955, the ACNM has served as the central authority charged with defining nurse-midwifery practice. In each update of the ACNM Definition of Nurse-Midwifery Practice, the scope of practice is defined more extensively and includes more elements. For example, the 1961 version stated, "Nurse-midwifery practice is an extension of nursing practice into the areas of management of care of mothers and babies throughout the maternity cycle so long as progress meets criteria accepted as normal." Later versions eliminated the criteria that women be "essentially normal" in recognition that midwifery practice had expanded into care of women with some complications and comorbid conditions. The current definition recognizes certified midwives as well as certified nurse-midwives.

Recognizing that midwifery practice is ever evolving, ACNM developed *Standards for the Evaluation of Nurse-Midwifery Procedural Functions* in 1972.[59] These guidelines formed a structure for nurse-midwives to use in assessing appropriateness of new practice elements. This process was updated in 1992 and published as *Guidelines for Incorporation of New Procedures into Nurse-Midwifery Practice*, which now can be found in the *Standards for the Practice of Midwifery.*

During the 1980s and 1990s, interest in "advanced" skills continued to grow. Surveys of ACNM members found that nurse-midwives were performing procedures that had not been part of their basic education, including placement of fetal scalp electrodes and intrauterine pressure catheters, repair of third- and fourth-degree lacerations, circumcision, use of vacuum extractors and forceps, manual removal of the placenta, paracervical blocks, ultrasonography, colposcopy, endometrial biopsy, external cephalic version, and being first assistant at a cesarean birth.[49] In 1993, a special issue of the *Journal of Nurse-Midwifery* provided, for the first time, a "comprehensive look at nurse-midwifery practice beyond its traditional boundaries."[60(p10S)] The issue included articles and a "home study program" dealing with circumcision, endometrial biopsy, third-trimester ultrasound, amnioinfusion, external cephalic

version, vacuum extraction, subdermal contraceptive implants, and fetal scalp blood sampling.

In spite of some nurse-midwives' enthusiasm for expanding their scope of practice through the addition of advanced skills, midwives have always grappled with defining the boundaries of the profession.[61,62]

Summary

The scope of practice of the earliest nurse-midwives primarily entailed maternity care, including the relatively new prenatal care process. Some early nurse-midwives included public health care in their services to meet the needs of the populations they served. Gradually, to continue to meet women's needs, family planning, gynecologic care, primary care, and "advanced skills" became part of midwives' scope of practice. Throughout this evolution, a systematic process for modifying the scope of practice has guided the profession.

Caring for Underserved Populations

Although the scope of midwifery practice has expanded over the past century, many aspects of midwifery care remain unchanged—particularly the focus on caring for women and families with unique health needs resulting from lack of access to care and other social, geographic, ethnic, or other factors. In fact, nurse-midwifery in the United States is firmly rooted in caring for members of underserved populations. The founders of the first nurse-midwifery services, including the Frontier Nursing Service, the Maternity Center Association, and the Catholic Maternity Institute, established these services as targeted strategies to serve the needs of some of the most underserved people of the day. Studies have consistently shown positive health outcomes of midwifery care, even in populations whose socioeconomic factors are often associated with poorer outcomes.

Nurse-Midwifery Care of Underserved Populations in the Early Twentieth Century

In New York City, the MCA served poor, urban-dwelling women, many of whom lived in cold-water tenement flats and were undernourished, lacked social support, and of high parity. MCA served a predominantly African American and Puerto Rican clientele, groups that struggled with high rates of unemployment, housing discrimination, and workplace exploitation. Many MCA patients were unable to pay the $5 fee the service charged to cover prenatal, labor and birth, and postpartum care.[20]

When Mary Breckinridge decided to establish the Frontier Nursing Service in southeastern Kentucky to provide maternity and general nursing care to thousands of mountain residents, she chose this geographic region because of the area's isolation, high rate of poverty, lack of healthcare options, and poor health outcomes. According to a 1931 American Medical Association publication:

> *The Frontier Nursing Service has set out to provide nursing, public health service and midwifery under medical direction . . . for the remotest sections of the southern mountains. Its work began . . . with two nurse-midwives in a remote Kentucky county in which . . . there was no resident physician for a population of 10,000 people. . . . The country is a veritable frontier—no railroads, no automobile roads, no bridges over its rivers and creeks. . . . Land usable for farming is so scant that the people are very poor.*[63](p633)

Another early nurse-midwifery service established to meet the needs of a underserved population was the Catholic Maternity Institute. In the early 1900s, New Mexico's population was predominantly rural, with fewer than 3 people per square mile in New Mexico compared to nearly 31 people per square mile nationwide.[64] New Mexico was the last state in the nation to establish a state health department, and infant mortality rates in New Mexico were among the highest in the nation when the Catholic Maternity Institute was founded. Medical care was less available to New Mexicans than to residents of other states, with about half the number of

physicians per person.[64]

The Medical Mission Sisters—a Catholic order of nurses, physicians, and other healthcare providers—was organized to provide medical services internationally but was available for service in the United States at that time due to World War II travel restrictions. In response to the great needs in rural New Mexico and at the request of the Archbishop of Santa Fe, the sisters established the Catholic Maternity Institute there in 1944. Sister Theophane Shoemaker, one of the founding members of the Catholic Maternity Institute, described their approach, embodying the ideals of providing what was needed to communities of great need:

> *. . . we became an integral part of the community of Santa Fe. . . . we became acquainted with the families . . . visited every health and social agency and met their officers and staff members. We went on home visits with the public health nurse as a means of learning the importance of tiny alleys and the humble homes to which they lead. We talked with hundreds of mothers . . . and from them we learned that the people are for the most part very poor, uneducated, proud of their culture, and sensitive to protect it. They are willing to accept good care but unwilling to be served by professionals who may not be sympathetic.*[65(p645)]

Today, midwives continue to care for women who represent a wide range of underserved populations. Some of these populations include racial and ethnic minorities; pregnant adolescents; immigrants; impoverished women; residents of Indian reservations; lesbian, gay, bisexual, transgender, and queer (LGBTQ) persons; and low-income and uninsured persons who receive care in Federally Qualified Health Centers (FQHCs). One commonality between these groups is a lesser degree of access to healthcare services compared to the general population. The work of innumerable midwives who have cared for underserved populations is illustrated in the following exemplars.

Race, Ethnicity, and Culture

Midwives have always cared for women who come from diverse cultures, and they continue to play an important role in providing healthcare services to women in racial and ethnic minority groups. A 1994 *Journal of Nurse-Midwifery* article reported the results of more than 30,000 nurse-midwife–attended births between 1981 and 1992 in the in-hospital birth center at the University of Southern California Women's Hospital in Los Angeles. Service staff members cared for a largely Hispanic, low-income population and recorded excellent outcomes, with successes attributed to a cooperative system of interprofessional care that could serve as a model for caring for low-income, minority women and infants.[66] Studies conducted as recently as the late 1990s and early 2000s show that nurse-midwives are still caring for women who belong to many diverse populations and that nurse-midwives were more likely than physicians to care for women from minority populations.[67]

It is important to note that the midwifery workforce has never matched the racial and ethnic composition of midwifery clients. In 1981, Betty Watts Carrington wrote of her concern with "this very low representation of ethnic minorities in American nurse-midwifery," citing, among

other considerations, the *difficulty* "for nonminority healthcare providers to be sensitive to and identify subtle cultural traits and lifestyles that adversely affect minority health."[68(p1)] Carrington voiced frustration about the lack of progress in minority recruitment of nurse-midwives and issued a plea for the profession to "make a greater effort to see that [nurse-midwifery] represents the cultural and ethnic richness of American society."[68(p2)] In spite of work within the profession to develop a more diversified workforce during the 1980s and 1990s, Holly Powell Kennedy and colleagues' "Voices of Diversity in Midwifery Study" in 2006 indicated that midwifery clients were still significantly more racially and ethnically diverse than the midwifery workforce.[69] This is an important focus for the ongoing evolution of the midwifery profession today.

Immigration

Midwives have often served as healthcare providers for immigrant women, and numerous published reports have documented the effectiveness and cultural appropriateness of that care. One exemplary report is that of Kathleen Morrow, who in 1986 described the interplay between midwifery care and birthing customs of Hmong people in California. Morrow concluded:

> *In listening to the Hmong, I became aware of the important role of childbirth traditions in maintaining physical and mental well-being. As nurse-midwives, we can actively assist people by accepting and incorporating their customs whenever possible into their health care. This is only a beginning, and I encourage other CNMs to explore the cultural implications in healing. Technology cannot replace the emotional and spiritual benefit accomplished through maintaining these customs.*[70(p288)]

Native Populations

Native populations and residents of reservations often experience challenging circumstances related to housing, sanitation, employment rates, nutrition, and transportation; midwives have worked to ease native peoples' healthcare difficulties for many years. Nurse-midwifery care with native populations has contributed to improved maternal and neonatal health outcomes and enhanced access to care, including a greater number of prenatal and postpartum visits and nutrition counseling.[71]

Beginning in 1969, Carol Milligan provided midwifery care as an Indian Health Service employee in Bethel, Alaska—a community with the nation's highest infant mortality rate that was so remote that it was accessible only by boat or plane.[8] In the early 1970s, nurse-midwives cared for Navajo women in Fort Defiance, Arizona. They included Hazel Canfield, the first Navajo midwife, and Ursula Knoki-Wilson, the daughter of a traditional midwife, who received the American College of Obstetricians and Gynecologists' 2017 William H.J. Haffner American Indian/Alaska Native Women's Health Award for her work in health care and advancing cultural awareness.

A 2008 ACNM publication described midwives' contributions to the Indian Health Service.[72] At that time, nurse-midwives provided comprehensive care in nine Indian Health Service regions, demonstrating excellent maternal and neonatal statistics. Nurse-midwives' work in this setting is geared toward meeting the unique needs of their communities. For example, the Chinle Comprehensive Health Care Facility provides "midwifery care that is culturally appropriate for the Navajo women and families they serve . . . the Midwifery Service has developed its own educational materials that present information that is grounded in Navajo culture."[72(p2)]

Socioeconomic Risks

A demonstration project in an impoverished agricultural community in California, Madera County, between 1960 and 1963 is an important and often-cited illustration of the effect of midwifery care in populations of women who have socioeconomic risks for adverse health outcomes. At the time, this area suffered from critical shortages of physicians; large numbers of migrant farmworkers in Madera County received late or no prenatal care and gave birth unattended by a physician. Because midwifery was illegal in California at that time, a special law authorizing midwifery practice allowed nurse-midwives to manage women with normal pregnancies, labors, and births. By the end of the project, CNMs were attending 78% of hospital births and access to care had improved dramatically in Madera County. Rates of premature births dropped from 11% to 6.4%, and neonatal mortality declined from 24/1000 live births to 10.3/1000 live births.[73,74] After the program ended and the midwives no longer provided care, a follow-up study showed immediate and significant increases in the rates of premature birth, neonatal mortality, and percentage of women who received no prenatal care. Nearby services did not see such changes, supporting the conclusion that the discontinuation of the nurse-midwifery program was responsible for the worsening of access and outcomes.[73,74] Recognizing the value of this work, state officials in California identified three essential components of the Madera County OB Access Program—nutrition counseling, psychosocial evaluation, and health education—and adopted these three aspects of midwifery practice as required elements in California's Medicaid obstetric service program. They remain required elements today.

The nurse-midwifery care of poor urban women at Grady Memorial Hospital in Atlanta, Georgia, was studied by Elizabeth Sharp and Lizabeth Lewis in 1984. Maternity patients were vulnerable to poor health outcomes due to social and economic circumstances, high parity, ages at the extremes of childbearing years, and race. Nevertheless, Sharp and Lewis concluded that "nurse-midwifery care can be integrated into a large tertiary level obstetric service retaining the philosophic stance of comprehensive care. . . . Practices related to patient options . . . are modifiable in a high technology setting."[75(p364)]

Lesbian, Gay, Bisexual, Transgender, and Queer Persons

LGBTQ persons are at increased risk for poor health outcomes as compared with non-LGBTQ persons. Midwives' commitment to meeting the needs of gender minorities can be found in

published reports dating to the 1980s. In 1984, nurse-midwives Eileen Olesker and Linda Walsh reported the results of their study of lesbians who had become pregnant. The researchers were interested in documenting the needs and perceptions of lesbians in order to improve nurse-midwives' knowledge and sensitivity to the needs of this population. The findings revealed that participants wanted healthcare providers to be knowledgeable about health needs unique to the population, as well as to demonstrate open and supportive attitudes.[76] Another midwifery study of lesbian mothers' experiences with health revealed that women who chose midwifery care reported higher levels of support from and comfort with their provider, as compared to women who chose physician care.[77]

More recently, midwives have demonstrated their commitment to serving the LGBTQ population by revising the ACNM's Core Competencies for Basic Midwifery Practice in 2012 to include a requirement to apply "knowledge, skills, and abilities, including in gynecologic care that include . . . human sexuality, including . . . gender identities and roles, sexual orientation . . . counseling, clinical interventions, and/or referral for sexual and gender concerns."[78] Moreover, the *Journal of Midwifery & Women's Health* has published several articles over the last several years aimed at enhancing midwives' knowledge and skills in caring for persons who identify as LGBTQ.[79]

Women Served by Federally Funded Health Centers

Federally funded health centers provide comprehensive primary care, with special emphasis on underserved populations, including migrant workers, homeless persons, residents of public housing, and others in need of affordable health care.[80] A 2010 ACNM publication provides details about 16 FQHCs that offer midwifery care.[81] Each center demonstrates midwifery principles of caring for underserved populations in culturally appropriate ways.

Summary

Since the profession's inception, midwives have cared for members of underserved populations, beginning with women and families in isolated and mountainous regions of Kentucky, impoverished women in crowded tenements of New York City, and women in rural and medically underserved New Mexico. Throughout the profession's history, midwives have continued to serve medically at-risk populations, and research has shown that midwifery care has provided benefits to those whom the profession serves.

Place of Birth

Having explored midwifery history through the lens of the populations midwives serve, we now consider where midwifery-attended births have taken place. Although most nurse-midwife–attended births occurred in women's homes in the earliest years, larger societal trends have caused the place of birth to shift over the years. Regardless of where births have taken place, midwives have worked to keep the woman at the center of the birth experience.

Births in Women's Homes

Prior to the 1950s, nearly all women gave birth at home, and nurse-midwives attended women at home. As the place of birth then largely changed from home to hospital, nurse-midwives worked diligently over the years to obtain hospital privileges so that they could continue to remain "with woman" during labor and birth.

The earliest nurse-midwifery services in the United States were created as home birth services and continued in this fashion for decades, with the majority of births taking place in women's homes. During the 26 years that the Maternity Center Association/Lobenstine Clinic provided clinical services (1932–1958), clinic staff attended a total of 7099 births; 6116 of these births took place in women's homes. Outcomes of these home births were excellent. The maternal mortality rate of the clinic was 0.9 per 1000 live births, as contrasted with a maternal death rate of 10.4 per 1000 live births for that geographic district as a whole, and 1.2 per 1000 live births for a leading hospital in New York City at that time.[82]

Likewise, in rural southeastern Kentucky, the vast majority of births attended by Frontier Nursing Service nurse-midwives between 1925 and 1950 occurred in their patients' mountain homes. As with the Maternity Center Association, outcomes of these home births were outstanding, with lower maternal and neonatal mortality rates than in other areas of the country, despite the local population's socioeconomic and health-related risk factors.[83]

In the rural Santa Fe, New Mexico, area, the staff of the Catholic Maternity Institute also attended nearly all births in homes between the service's inception in 1944 and 1950. Indeed, home birth was the sisters' ideal, economically and spiritually. During mothers' classes, the staff emphasized: "Home delivery is good and proper thing (besides being cheaper). Not only the Birthday but birthplace is important. Bethlehem's stable honored world over."[84(p156)] Recounting her experiences attending births in her patients' homes, nurse-midwife Sister Catherine Shean said:

> *I think one of the most beautiful [aspects] for me was when you were in the home and the baby was born and . . . the mother had been cleaned up and she was ready to receive the baby . . . many times the other younger children were invited in to meet the baby. . . . We had the tradition in our midwifery service that when we finished with the mother . . . we would gather together the family and the husband and we would pray with them before we left, thanking God for this new life and for all the help that He had given to us.*[84(pp156-157)]

Although the Catholic Maternity Institute was founded as a home birth service, within 2 years of the service's inception, CMI staff began offering women the choice of giving birth in their homes or in a small freestanding building, La Casita, near CMI's main building. La Casita births met a number of practical needs, including close proximity to the hospital in the event of complications, an increased number of clinical experiences for student nurse-midwives, and more efficient use of the midwives' time, given the long and difficult travel requirements to reach patients' homes. See **Figure 1-5**.



Figure 1-5 Sister Pat Patton of Catholic Maternity Institute holding a newborn; circa 1955.

Courtesy of Medical Mission Sisters.

Shift to Hospital Birth, 1930s to 1950s

Major shifts in national birth trends occurred during the 1930s and 1940s. In 1932, more than 60% of births took place at home; by 1950, however, 88% of births took place in hospitals.[45] A confluence of factors contributed to this change, including explosive growth in the number of hospital beds and the increased number of people using health insurance. Another factor was the widespread acceptance of maternity care practices that required hospitalization, including analgesic and amnesic medications to manage labor pain, and the use of forceps and

episiotomies. Importantly, too, health experts in that era credited the improvements in maternal and infant mortality rates to the increased percentage of hospital (physician-attended) births.

At midcentury, nurse-midwives maintained their focus on meeting women's needs, using established elements of the midwifery model of care. They made concerted efforts to bring consumer advocacy and family-centered maternity care to hospitals. Although many women and their caregivers were no longer in the home at the time of birth, nurse-midwives' leadership was key in promoting rooming-in and breastfeeding; studying the effects of natural (prepared) childbirth and family-centered supportive care on a woman's prenatal, intrapartum, and postpartum experience; and including fathers or significant others in hospital labor and delivery rooms.[85]

Although it is unclear who first used the phrase "family-centered maternity care," MCA director Hazel Corbin is generally credited with widely promoting the concept. Corbin highlighted the need to involve a woman's family, so as to counteract the negative effects a hospital birth experience could have on the development of the family unit. Nurse-midwife and MCA graduate Kate Hyder contributed to development of the United States' first rooming-in unit at Grace–New Haven Community Hospital in the mid-1940s.[7] When nurse-midwife and MCA graduate Ernestine Wiedenbach published a nursing textbook, *Family-Centered Maternity Nursing*, in 1958, she reframed the art and science of obstetric nursing and inspired a generation of nurses to seek midwifery education.[86]

1960s: Resurgence of Home Birth

During the late 1950s and the 1960s, an increasing number of women and families voiced dissatisfaction with the hospital birth status quo: Hospital staff often separated families during labor and birth; women underwent routine enemas, vulvar shaving, and episiotomies; and they gave birth with their legs strapped into stirrups. Finding the hospital environment to be too confining and disempowering, some consumers, midwives, and physicians began to reconsider home birth. During the 1970s, fueled by feminism, counterculture ideals, and the women's health movement, consumer demand for home birth grew.

The consumer dissatisfaction with professional health care contributed to increased interest in midwives who were often characterized as "lay midwives." These midwives, with varying educational backgrounds, offered home birth services. Lay midwifery suffered from its own challenges as an unregulated group during this era. The educational preparation of non-nurse-midwives during the 1970s and 1980s was highly variable. Lay midwives and their advocates worked to resolve questions regarding the desirability of formal education, standards, credentialing, and regulation. A number of groups and organizations were founded during the 1970s to attempt to unite supporters of lay midwifery and home birth, including the National Association of Parents and Professionals for Safe Alternatives in Childbirth (NAPSAC), Home Oriented Maternity Experience (HOME), Association of Childbirth at Home International (ACHI), and National Midwives Association (NMA). Existing organizations, such as the International Childbirth Education Association (ICEA) and La Leche League, added their support. The first national meeting of lay midwives took place in 1977 in El Paso, Texas.[7]

In spite of increasing consumer demand for home birth, nurse-midwives' professional consensus on birth at home was slow to develop. Nurse-midwives who chose to provide home birth services in the early 1970s did so without support from ACNM. A statement on home births, adopted in 1973 and published in a 1975 *Journal of Nurse-Midwifery*, was critical of home birth: "ACNM considers the hospital or officially approved maternity home as the site for childbirth because of the distinct advantage to the welfare of mother and child."[87(p15)] Some ACNM members voiced opposition to this statement, citing a lack of supporting research. Additional discussion ensued and, in 1980, the ACNM published a statement on practice settings that acknowledged the home as an acceptable practice location.

During the 1990s, ACNM established a formal structure to address the needs of midwives providing home birth services.[88] Today home birth is strongly supported by ACNM within evidence-based guidelines that address safety, and appropriate candidates for care in this setting as reviewed in more detail in the *Birth in the Home and Birth Center* chapter.

1970s: The Birth Center Movement

While some nurse-midwives, consumers, and non-nurse-midwives viewed home birth as the best alternative to hospital birth for selected low-risk women, others became interested in a different option—the birth center. Building on the foundation established by La Casita, the Catholic Maternity Institute's freestanding birth center in Santa Fe, which operated from 1946 through 1969,[84] the modern birth center movement developed during the 1970s and 1980s. The first freestanding birth center of that era opened in 1975: Maternity Center Association's demonstration model, the Childbearing Center (CbC). MCA staff opened the CbC after they "detected a new determination in some childbearing couples . . . to give birth out of hospital." Birth center pioneer and nurse-midwife Ruth Lubic describes the reason that couples desired out-of-hospital birth:

> . . . *hospitals—or rather the professionals functioning within them—had grown* increasingly insensitive to the need of members of human families for each other's pres*ence in times of crisis and celebration, particularly during and surrounding childbirth. This attitude on the part of medical professionals may have been a side-effect of their almost religious fervor to improve rates of maternal and infant survival.*[89(p225)]

The birth center movement grew quickly, and birth center advocates diligently studied and reported birth center care outcomes. In 1981, MCA funded the first national study of birth center outcomes in 14 centers. Several important publications followed, including two landmark studies: the National Birth Center Study (1985–1987), published in the *New England Journal of Medicine*, and the San Diego Birth Center Study (1994–1996), published in the *American Journal of Public Health*.[90,91] These studies demonstrated that birth center care is safe, effective, satisfying, and cost-effective. The National Birth Center Study II, published in 2013, again demonstrated the safety of the birth center model. This study, led by nurse-midwives Susan Stapleton and Cara Osborne, included more than 15,000 women.[92]

Organizations composed of dedicated professionals helped the birth center movement to succeed. Over the years, the MCA's Cooperative Birth Center Network (CBCN), founded in 1981, evolved to be known as the National Association of Childbearing Centers (NACC) in 1983. In 2005, this organization changed its name to become the American Association of Birth Centers (AABC). AABC's many contributions to the birth center movement include articulating eligibility criteria for birth center care, developing national quality standards, fostering state licensure, developing clinical position statements, securing liability insurance, promoting reimbursement, establishing accreditation mechanisms, and participating in crucial birth center outcomes studies.

Summary

The earliest nurse-midwives attended births almost exclusively in women's homes. As the twentieth century progressed, larger societal influences led more women to choose hospital birth; in turn, nurse-midwives sought opportunities to serve women in that setting. In the 1960s and beyond, women began to demand more control and a less medically focused birth experience. Consequently, home birth experienced a resurgence, and the same factors led to increasing interest in birth center care.

Nurse-Midwife/Physician Collaboration: Opportunities and Challenges

Regardless of whether nurse-midwives have attended births in women's homes, birth centers, or hospitals, collaboration with physician colleagues has been crucial. The earliest nurse-midwives actively worked to avoid conflicts with physicians. Today midwives and physicians optimally work as members of a team, although the midwife–physician relationship has evolved over the years as both professions matured.

Early Models of Successful Collaboration

The new profession of nurse-midwifery emerged from the social and cultural context of the "midwife problem." From the earliest days of this profession, several services demonstrated successful models of physician support and collaborative care utilizing nurse-midwives. One supportive physician was the prominent and influential Ralph Lobenstine, whose work with the Maternity Center Association in New York City was crucial to the MCA's early successes. In a 1939 *American Journal of Nursing* article, Hattie Hemschemeyer described in detail the careful collaboration between MCA nurse-midwives and physicians, and identified the ways in which that collaboration resulted in healthier mothers and babies.[93]

In 1925, when the Frontier Nursing Service was founded in Kentucky, the legal authority for practice stemmed from the clearly articulated support of FNS's physician collaborators. According to the FNS's statement of purpose, midwives were expected to work "under supervision, in compliance with the regulations for midwives of the State Board of Health and the law governing the Registration of Nurses in Kentucky; and in cooperation with the nearest medical service."[49(pp523-524)] Moreover, the FNS hired a physician as medical director, convened a Medical Advisory Committee, and created an extensive medical routine (protocols).[48]

At the Catholic Maternity Institute, the nurse-midwives and their collaborating physician developed and maintained a strong and mutually respectful relationship. Sister Theophane Shoemaker, the CMI director, wrote in 1946 of the support of the medical director:

> *One of the greatest contributors to our early success and to the progress of our work, was Nancy Campbell, M.D., an obstetrician, who from the beginning has been the medical director of our program. She is convinced of* the special contribution nurses trained in midwifery have to offer. . . . Over and over again she told patients about our work and said to them: "Go to the Sisters, because they are trained as nurses and as mid *wives to give good care . . . the nurse-midwives can give better delivery care because they can give more time throughout labor and delivery than I or any other physician can afford to give."*[65(pp645-646)]

Effects of Physician Shortages in the Mid-Twentieth Century

At midcentury, members of the medical community expressed growing interest in nurse-

midwife/physician collaboration, particularly as it affected looming shortages in the maternity care workforce. Concerned about the effects of the post–World War II baby boom, an obstetrician wrote in a 1959 *Bulletin of the American College of Nurse-Midwifery*:

> *. . . the American economy is expanding and with this expansion goes a great increase in population: more babies will be born but there will not be a commensurate increase in physicians and therefore other birth attendants will be needed; and nurse-midwives are the logical people to fill this role.*[94(p9)]

Additionally, nurse-midwives were becoming more visible in some physician-dominated arenas. For example, in the mid-1950s, nurse-midwifery programs opened within university teaching centers at Columbia, Johns Hopkins, and Yale.[95]

Control of Nurse-Midwifery Training and Practice in the 1960s and 1970s

Although some obstetricians supported nurse-midwifery in concept, many physicians believed that obstetricians should control the training and practice of nurse-midwives. Physician John Whitridge wrote of his vision of how best to use the modern nurse-midwife in the *Bulletin of the American College of Nurse-Midwifery* in 1960:

> *Working in cooperation with and under the supervision of physicians, the nurse-midwife can spare the physician many long hours of work for which his special skills are not always required. . . . The nurse-midwife is trained and accustomed to working under medical supervision and would be most unhappy as an independent practitioner of midwifery.*[95(p33)]

In fact, heated debates about this topic occurred within the American College of Obstetricians and Gynecologists (ACOG) throughout the 1960s. In 1959, ACOG convened a Committee on Obstetrical Assistants to study the role of nurse-midwifery in the United States. Debates among committee members ranged from whether ACOG would support nurse-midwifery in general, to committee members' opinions about the name "nurse-midwife." In 1971, the Committee on Obstetrical Assistants became the Committee on Professional Personnel and recommended the adoption of the *Joint Statement on Maternity Care* that the American College of Nurse-Midwives had approved.[96]

This official ACOG recognition of nurse-midwives represented an important step forward in professional recognition but it did not equate to full professional autonomy for nurse-midwives. Indeed, the *Joint Statement* specified that "The cooperative efforts of teams of physicians, nurse-midwives, obstetric registered nurses and other health personnel will be directed by a qualified obstetrician-gynecologist."[96(p22)]

Successful Collaboration Models in the 1960s and 1970s

During the 1960s and 1970s, a number of nurse-midwife/physician interdisciplinary teams demonstrated that nurse-midwife/physician collaboration was a successful model. One

example was the Madera County Demonstration Program in California. Publishing their results in the *American Journal of Obstetrics and Gynecology*, Levy and colleagues argued that nurse-midwives not only relieved a maternity care provider shortage in Madera County, but the collaborative approach of physicians and nurse-midwives also drastically improved maternal and neonatal health indices.[73] Similarly, a maternity care team in Holmes County, Mississippi, demonstrated that infant mortality could be halved by including nurse-midwives and physicians in the same maternity care team.[97] When nurse-midwife Marie Meglen spoke at the 1971 ACOG meeting and described her experiences in the Holmes County service, she implored attendees—most of whom were physicians—to include nurse-midwives in solving maternity care challenges:

> *. . . by using each member of the team to do only those things for which he or she is best prepared, we will be able to provide better care for more patients and, in the long run, change the standard of maternal and infant health care in our area. We have learned a great deal in our first two years in Mississippi, which I would like to pass on to you in hopes that it might expedite your efforts if you should want to make use of nurse-midwives.*[98(p67)]

Similarly, a service in Springfield, Ohio, successfully incorporated nurse-midwives into the Maternal Health Service. Physician John Burnett published information about his experiences in Springfield in *Obstetrics and Gynecology*, increasing awareness among obstetricians about "matching talents with needs" within the obstetrics service and arguing that "the nurse-midwife has demonstrated her ability to join physicians in the practice of total maternity cycle care."[99(p719)]

Increased Consumer Demand for Midwifery: 1970s to the Present

Societal shifts during the 1960s and 1970s, including feminism and the consumer movement, created more middle-class demand for nurse-midwifery. Earlier in the profession's history, nurse-midwives had primarily cared for women from lower-socioeconomic groups and in areas in which few physicians wanted to practice; during the 1970s, however, more middle-class women who could pay for maternity care wanted midwives. In fact, the 1976–1977 survey by the American College of Nurse-Midwives reported that approximately 26% of all nurse-midwives practicing nurse-midwifery worked in some form of private practice arrangement.[100]

By the 1990s, professional autonomy for nurse-midwives had become more established. The ACNM's 1992 revision of the definition of nurse-midwifery practice removed the modifier of "essentially normal" to describe the populations served by nurse-midwives, thereby expanding the scope of "independent management," and removed any reference to medically directed teams. Throughout the 1990s, the *Journal of Nurse-Midwifery* published a number of articles and editorials to provide nurse-midwives with an armamentarium of validation of their experiences, encouragement to continue advocating for themselves, and resources with which to build their knowledge about collaborative practice.[101-103]

Midwife/physician collaboration continues to evolve today. In 2002, ACOG and ACNM published a revised version of the *Joint Statement of Practice Relations Between Obstetrician-Gynecologists and Certified Nurse-Midwives/Certified Midwives* that, for the first time, made no mention of physician supervision or of unequal professional standing between midwives and physicians. The 2011 *Joint Statement* update continued to codify the intention to move forward with mutual respect and collaborative relationships.[104] Unfortunately, there are still some areas where physicians and certified nurse-midwives or certified midwives wish to work collaboratively but are challenged by outdated legislation and problems obtaining reimbursement.

Summary

The complicated relationship between midwives and physicians has been influenced by successful models of collaboration as well as by periods of conflict regarding professional autonomy and competition. In spite of conflicts, midwives and physicians share a common goal of safeguarding the health and safety of women and families. The work toward optimal collaboration to reach that goal continues.

Documenting the Outcomes of Midwifery Care and Midwifery Research

Many midwifery scholars, researchers, and clinicians have used the carefully collected and documented evidence of the excellent outcomes of midwifery care. This research began with the earliest nurse-midwifery service and has continued throughout the profession's history.

Early Nurse-Midwives' Documentation of Outcomes

The Maternity Center Association's work in New York City provides an important example of nurse-midwives' documentation of the outcomes of their care. Data analysis of MCA's comprehensive maternity care system indicates that MCA-provided care resulted in significantly improved rates of maternal, fetal, and neonatal deaths. These and other data, such as method of delivery, total registrations, location of birth and type of birth attendant, patient gravidity, complication rates, and patient and nurse-midwife satisfaction, were reported in meticulous detail in a 1955 MCA publication, *Twenty Years of Nurse-Midwifery, 1933–1953*.[46]

Importantly, the MCA's emphasis on documenting outcomes of safe care during those first 20 years did not overshadow the heart of midwifery care. Near the end of the 20-year report, the authors praised the progress in decreasing mortality rates but lamented that, nationwide, "*too little attention [has been] paid to the social and emotional aspects of childbearing and their influence on family life. The nurse-midwife is helping to restore the emphasis on patient-centered care and total health of mother and child.*"[46(p115)]

From the start of her work in Kentucky, Frontier Nursing Service founder Mary Breckinridge prioritized the collection and use of data to guide leadership decisions. Speaking to the significance of data and its iterative value for improving an organization, Breckinridge wrote, "Research is a continuing thing. As one acts, one gets an insight of what is best for the next action."[48(p159)] Indeed, Breckinridge recognized that the first task was to accurately define the baseline with which her service's work would be compared. Breckinridge described the evolution of the research process one year after the founding of the service:

> *Leslie is a laboratory, our field of research. . . . We ask ourselves questions like these: Will our maternal and infant death-rate in rural sections of Kentucky be lowered by this system of nurse-midwives to figures comparable with those of the Old World? What area and population can be served by each nurse, combining midwifery with generalized public health nurses? . . . We are keeping very exact daily records in order to answer this. What will the cost be? . . . Will the people accept this service? . . . Can the service extend indefinitely with nurses only? . . . Time will tell.*[105(p47)]

And time did tell, showing that the work of Breckinridge and her nurses improved outcomes for mothers, babies, and families in the mountains of Kentucky. After developing and implementing a comprehensive record system, the FNS staff gathered data using a statistical

system set up by the Carnegie Corporation; the results were then analyzed by statisticians from the Metropolitan Life Insurance Company. These findings, reported for each series of 1000 pregnancies of FNS patients, provided some of the first statistical evidence of the safety and effectiveness of nurse-midwifery care in the United States. "The Summary of the Tenth Thousand Confinement Records of the Frontier Nursing Service," written by Metropolitan Life Insurance Company staff, published in a 1958 FNS *Quarterly Bulletin*, and reprinted in a 1960 *Bulletin of the American College of Nurse-Midwifery*, is considered to be a seminal study—one of the most important studies demonstrating exemplary midwifery practice.[83] Although the FNS nurse-midwives faced treacherous mountain terrain, severe weather conditions, nonexistent roads, a lack of electricity and plumbing, and impoverished and poorly nourished patients, their work made a real difference. The maternal death rate of 12 per 10,000 live births for the total period during the first 30 years of the service's existence was dramatically lower than the national maternal mortality rate. In the United States as a whole, the maternal mortality rate was 66.1 per 10,000 live births in 1931. Although the national maternal mortality rate declined to 8.3 per 10,000 live births in 1950, for much of the period between the start of FNS and 1950, the incidence of death secondary to a childbirth-related cause for women cared for by FNS midwives was much lower than that of their peers across the nation.[48,83]

ACNM's Leadership and Support for Research: 1950s to the Present

As nurse-midwifery matured as a profession, and its professional organization mirrored that increasing sophistication, increasing support for research became evident. One of the initial objectives of the American College of Nurse-Midwifery was "to promote research and develop literature in the field of nurse-midwifery."[29] The ACNM's Research and Statistics Committee provided leadership and guidance in documenting midwifery practice and research. As early as 1956, just one year after the ACNM's incorporation, the organization surveyed nurse-midwives about research and board members discussed how to manage data. The results indicated that, not only were ACNM leaders interested in documenting midwifery practice and outcomes, but they were also aware of the need to use the latest statistical analysis and data management methods:

> *Miss Ruth Doran . . . reviewed the information on the questionnaires that have been filled out by members of the College and [was] able to get some expert statistical advice on how data of this kind might be accumulated in the future. A discussion brought out the following points: It seems advisable to have a system whereby information is currently available. It is possible to set up a coding system in mimeograph form that could be transferred to IBM cards at a later date. A new form for gathering information for statistical purposes is needed.*[106(p13)]

The importance of ongoing collection and analysis of data on the outcomes of midwifery care were documented by nurse-midwifery educators at the first nurse-midwifery education workshop. A summary of this meeting noted that educators were preparing nurse-midwives to

"Participate in the systematic gathering and analysis of data for the purpose of evaluating services which affect the health of mothers and babies, and in implementing the findings."[7(p275)]

During the 1970s and 1980s, the *Journal of Nurse-Midwifery* provided advice and encouragement about the role of research. For example, a 1976 article, "Pragmatics of Research," included practical guidance about research topic-generation and prioritization, the role of collaboration in research, and basic legal issues. Additionally, the author devoted a large portion of the article to advice for practicing midwives who were not actively engaging in research activities:

> *Each nurse-midwife has the responsibility to be an intelligent, critical consumer of research . . . to retrieve research findings with ease, evaluate them as to their soundness, strengths, and weaknesses, and apply them in the clinical setting.* [That responsibility] *also involves providing constructive criticism to researchers by reacting to research findings in discussions and conferences, writing letters to journals and sponsoring organizations, and contacting the individual themselves.*[107(p16)]

In the early 1980s, a series of *Journal of Nurse-Midwifery* editorials explored the topic of nurse-midwifery research from a variety of perspectives. Jacqueline Fawcett, research consultant to the *Journal*, described editorial plans to publish original research articles, with special emphasis on application to clinical practice and replication of previous research. The next year, *Journal* associate editor Evelyn Hart reinforced the imperative for "scientific objective accountability for midwifery practice and education through research." She concluded that:

> *Midwifery has arrived at a point where it must assume responsibility for its own research. A scientific attitude and mode of thinking must be valued by midwives as much as skill and acumen in practice. Midwifery must be conveyed to the public, to physicians, and to other health professionals as a professional service just not only by its art, but also by scientific evidence gleaned from research.*[108(pp37-38)]

During this period, nurse-midwives were systematically compiling and summarizing the outcomes of nurse-midwifery care and using those data to change policy at the local, state, and national levels.[109] In addition to influencing the nurse-midwifery practice and regulation, these data were used to persuade healthcare payers to reimburse nurse-midwives for their services.

By 1988, the volume and complexity of midwifery's research endeavors had grown sufficiently that ACNM leaders and members recognized that the Research and Statistics Committee should move to the Division level. Betty Bear, ACNM president at that time, endorsed the change, and other nurse-midwifery leaders, such as Jeanne DeJoseph, Joyce Roberts, and Claire Andrews, contributed to the successful creation of the Division of Research. Lisa Paine served as the first chairperson.[110]

During the late 1980s and 1990s, nurse-midwives benefited from ever-increasing emphasis on research through participation in grant-funded studies; development of data sets; publication

of research-focused articles in the *Journal of Nurse-Midwifery*; and presentation of ACNM annual meeting poster sessions, research forums, and educational sessions and workshops.[110] Indeed, *Journal* readers were challenged to expand their ideas and their practice through articles such as Joyce Fitzpatrick's "The Clinical Nurse-Midwife as Scientist." In that article, Fitzpatrick asked readers: "Why science? Why research? What does knowledge development have to do with the clinical nurse-midwife? Science thrives by asking impertinent questions and getting revolutionary answers. It is time for some revolutionary answers about health and health care delivery."[111(p37)]

Midwifery's Rich Heritage of Theory Development and Clinical Research

Throughout the history of their profession, nurse-midwives have shaped that profession through theory development and exploration of the meaning of the midwifery model of care.[112] The efforts of midwifery theorists have collectively defined the midwifery model of care, including Ernestine Wiedenbach's work on family-centered maternity care [86]; Ela-Joy Lehrman's work on family-centered care, health education, and advocacy for non-intervention[113]; Joyce Thompson's writings about human dignity and self-determination[114]; Holly Powell Kennedy's work on compassion and careful non-intervention[115]; and Jo Anne P. Davis's work defining normalcy.[116]

In addition to theory development, midwives have a rich history of researching many different aspects of care; much of this research is presented in various chapters of this text. Selected examples include the prenatal care model known as CenteringPregnancy[117]; components of prenatal care provided by nurse-midwives compared to physicians[118]; noninvasive methods of assessing uterine size, gestational age, and fetal presentation and position[119]; management of the perineum at birth and the value of delayed cord-clamping[120,121]; position, breathing, and timing of pushing in the second stage of labor[122]; and home birth and birth center outcomes.[90-92,123] Knowledge about perinatal mood disorders and newborn care has also been furthered by midwifery researchers.[124,125]

Summary

Beginning with early nurse-midwives documenting the outcomes of their care in the Frontier Nursing Service and the Maternity Center Association, midwives have diligently contributed to the body of knowledge about midwifery care and women's health. As the result of ACNM's leadership and the work of theorists, researchers, and clinicians, midwifery has a rich research heritage.

Nurse-Midwifery Education

Midwifery could not exist without midwifery education. The history of this education has some notable features: the interconnectedness of educational programs across time and geography; the little-told stories of programs designed specifically to educate black nurse-midwives; the ebb and flow of programs opening and closing; the long-standing commitment to the hallmarks of midwifery care; and the use of educational innovations to enhance learning and skill development and to make midwifery education more widely available, particularly to rural and underserved populations.

Educational Program Interconnectedness: 1930s to the Present

The first nurse-midwifery education program in the United States was New York City's Manhattan Midwifery School, which operated from 1925 to 1931. Little is known about this program, but subsequent nurse-midwifery education programs in the United States can be connected through a clear "genealogy" of programs. The second and third programs to open in the United States constitute what Helen Varney Burst and Joyce Thompson term "first-generation" programs.[126] In 1932, the School of the Association for the Promotion and Standardization of Midwifery opened. Commonly known as the Lobenstine Midwifery School, it became the Maternity Center Association School of Nurse-Midwifery in 1934 (**Figure 1-6** and **Figure 1-7**). The next program, which opened in 1939, was the Frontier Graduate School of Midwifery of the Frontier Nursing Service (FNS) in Hyden, Kentucky, which later became the Frontier School of Midwifery and Women's Health and more recently was renamed Frontier Nursing University.



Figure 1-6 In the late 1930s, a student nurse-midwife at Maternity Center Association's Lobenstine School of Midwifery is taught how to perform blood pressure measurement by Rose McNaught.

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Figure 1-7 A new nurse-midwifery student (Margaret Thomas) in the 1930s being greeted by faculty member Rose McNaught at the Maternity Center Association Lobenstine Clinic and School.

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All subsequent nurse-midwifery education programs are closely linked, with second-generation programs being started by graduates of the MCA and FNS programs. Likewise, third-generation programs were started by graduates of second-generation programs, and so on. Even the newest programs are third- or fourth-generation programs, demonstrating the tight interconnectedness of nurse-midwifery educational programs (**Table 1-1**). In 2003, Helen Varney Burst and Joyce Thompson eloquently described the significance of these connections to nurse-midwives:

Table 1-1 **Timeline and "Generation" of Midwifery Educational Programs**

Educational Program, Affiliation, and Location	Burst/Thompson "Generation" of Program	Year Opened	Year Closed
Manhattan Midwifery School, affiliated with the Manhattan Maternity and Dispensary; program under jurisdiction of the hospital's School of Nursing, New York, NY	n/a	1925	1931
School of the Association for the Promotion and Standardization of Midwifery (commonly known as the Lobenstine Midwifery School); became the Maternity Center Association School of Nurse-Midwifery in 1934. Affiliated with Downstate Medical Center, State University of New York, and Kings County Hospital, Brooklyn, New York, in 1958; also includes an early affiliation of Maternity Center Association (MCA) and Kings County Hospital with Johns Hopkins University during 1958–1960, New York, NY	First	1932	Open today (as State University of New York [SUNY] Downstate)
Frontier Graduate School of Midwifery of the Frontier Nursing Service, Hyden, KY	First	1939	Open today (as Community-based Nurse-Midwifery Education Program [CNEP]/ Frontier Nursing University)
Tuskegee School of Nurse-Midwifery; a joint project of the Macon County Health Department, the Children's Bureau, the Julius Rosenwald Fund, Tuskegee University (although not officially part of Tuskegee University), and the Alabama State Department of Health, AL	Second	1941	1946
Flint-Goodridge School of Nurse-Midwifery; in connection with Flint-Goodridge Hospital and Dillard University; first nurse-midwifery program to be affiliated with a university, New Orleans, LA	Second	1942	1943
Catholic Maternity Institute School of Nurse-Midwifery, Santa Fe, NM	Second	1945	1968
Catholic University of America; affiliated with Catholic Maternity Institute; first nurse-midwifery education program to be part of a master's degree program, Washington, DC	Second	1947	1969
Columbia University Graduate Program in Maternity Nursing, New York, NY	Second	1955	Open today
Johns Hopkins University Nurse-Midwifery Program, Baltimore, MD; closed in 1981 but today is affiliated with Shenandoah University, which provides the midwifery component of graduate education	Second	1956	1981
Yale University Graduate Maternal and Newborn Health Nursing Program, New Haven, CT	Second	1956	Open today

Educational Program, Affiliation, and Location	Burst/Thompson "Generation" of Program	Year Opened	Year Closed
University of Puerto Rico/Caparra Heights District Hospital, San Juan, Puerto Rico	Second	1960	1981
New York Medical College Graduate School of Nursing Nurse-Midwifery Program, New York, NY	Second	1963	1972
University of Utah Graduate Maternal–Infant Nursing Program, Salt Lake City, UT	Third	1965	Open today
Ponce District Hospital, Ponce, Puerto Rico	Third	1966	1975
University of Mississippi Medical Center Nurse-Midwifery Program, Jackson, MS (Refresher)[a]	Second	1971	1972
University of Mississippi Medical Center Nurse-Midwifery Program, Jackson, MS	Third	1969	1985
University of Illinois at Chicago Nurse-Midwifery Program, Chicago, IL	Second	1972	Open today
Maternal Health Service of Community Hospital, Springfield, OH (Refresher)[a]	Third	1971	1975
Loma Linda University Nurse-Midwifery Program, Loma Linda, CA	Third	1971	1975
Beth Israel Hospital, New York, NY (Refresher)	Second	1972	1975
Booth Maternity Refresher Program,[a] Philadelphia, PA	Second	1973	1984
University of Minnesota Nurse-Midwifery Program, Minneapolis, MN	Third	1973	Open today
Medical University of South Carolina Nurse-Midwifery Program, Charleston, SC	Third	1973	2009
Georgetown University Nurse-Midwifery Program, Washington, DC	Third	1973	Open today
St. Louis University Graduate Program in Nurse-Midwifery, St. Louis, MO	Third	1973	1984
Meharry Medical College Nurse-Midwifery Program, Nashville, TN	Second	1973	1985
University of Kentucky Nurse-Midwifery Program, Lexington, KY	Third	1974	1998
University of Medicine and Dentistry of New Jersey Nurse-Midwifery Program, Newark, NJ; now Rutgers Biomedical Health Sciences	Third	1975	Open today
University of California, San Diego Nurse-Midwifery Program, San Diego, CA	Third	1975	1995
U.S. Air Force Nurse-Midwifery Program, Andrews Air Force Base, MD	Second	1975	1997
Grady Memorial Hospital, Atlanta, GA (Refresher)[a]	Third	1972	1975
Emory University Nurse-Midwifery Program, Atlanta, GA	Third	1976	Open today

Educational Program, Affiliation, and Location	Burst/Thompson "Generation" of Program	Year Opened	Year Closed
University of Arizona Nurse-Midwifery Program, Tucson, AZ	Third	1977	1985
University of Miami Nurse-Midwifery Program, Miami, FL	Third	1978	2014
San Francisco General Hospital, San Francisco, CA (Refresher)[a]	Third	1976	1978
San Francisco General Hospital/University of California San Francisco Interdepartmental Nurse-Midwifery Education Program, San Francisco, CA	Third	1978	Open today
University of Pennsylvania, Philadelphia, PA	Second	1980	Open today
University of Colorado, Denver, CO	Third	1980	Open today
Oregon Health Sciences Center, Portland, OR	Third	1981	Open today
University of Southern California, Los Angeles, CA	Second	1981	2003
Rush University/Presbyterian Hospital, Chicago, IL	Third	1982	1988
Stanford University, Stanford, CA	Third	1982	1987
University of Florida, Gainesville, FL	Third	1982	Open today
Case Western Reserve University, Frances Payne Bolton School of Nursing, Cleveland, OH	Fourth	1983	Open today
University of California, San Francisco/University of California, San Diego Intercampus Nurse-Midwifery Program, San Francisco/San Diego, CA	Third	1983	1999
Baylor College of Medicine, Houston, TX	Fourth	1984	2004
Education Program Associates, San Jose, CA	Third	1987	1998
Parkland Memorial Hospital/The University of Texas Southwestern Medical Center, Dallas, TX	Fourth	1989	2006
University of Michigan, Ann Arbor, MI	Third	1990	Open today
Charles R. Drew University, Los Angeles, CA	Third	1990	2006
University of New Mexico, Albuquerque, NM	Second	1991	Open today
University of Alabama, Birmingham, AL	Third	1991	1996
University of Texas–El Paso/Texas Tech University Health Sciences Center, El Paso, TX	Fourth	1991	2004
Boston University School of Public Health, Boston, MA	Fourth	1991	2006
Baystate Medical Center, Springfield, MA	Third	1992	Open today
East Carolina University, Greenville, NC	Fourth	1992	Open today
Jackson Memorial Hospital, Miami, FL (Refresher)[a]	Fourth	1992	1997
University of Rhode Island College of Nursing, Warwick, RI	Second	1993	2007
Marquette University, Milwaukee, WI	Third	1993	Open today
University of Washington School of Nursing, Seattle, WA	Second	1993	Open today

Educational Program, Affiliation, and Location	Burst/Thompson "Generation" of Program	Year Opened	Year Closed
University of Texas Medical Branch, Galveston, TX and University of Texas at Houston Collaborative Education Program; later became solely University of Texas Medical Branch, Galveston, TX	Third	1992	2005
North Central Bronx Hospital, New York, NY	Third	1994	1995
University of Rochester, Rochester, NY	Fourth	1994	2001
University of California Irvine/University of California, Los Angeles, CA	Fourth	1994	2003
New York University, New York, NY	Third	1995	Open today
San Diego State University, University of California, San Diego, CA	Third	1995	Open today
State University of New York–Stony Brook, Stony Brook, NY	Third	1995	Open today
Vanderbilt University School of Nursing, Nashville, TN	Third	1995	Open today
University of Missouri, Columbia, MO	Fourth	1995	2002
Ramsey Clinic, St. Paul, MN (Refresher)[a]	Fourth	1995	1998
Institute of Midwifery, Women and Health, Philadelphia, PA; renamed Institute of Midwifery at Philadelphia University in 2003; renamed Institute of Midwifery at Jefferson in 2017.	Fourth	1996	Open today
The Ohio State University College of Nursing, Columbus, OH	Second	1997	Open today
University of Cincinnati, Cincinnati, OH	Fourth	1997	Open today
Shenandoah University, Winchester, VA	Fourth	1997	Open today
University of Puerto Rico School of Public Health, San Juan, Puerto Rico	Third	1998	2016
University of Kansas Medical Center, Kansas City, KS	Fourth	1999	Open today
University of Maryland, Baltimore, MD	Third	1999	2008
University of Indianapolis, Indianapolis, IN	Second	2003	Open today
Wayne State University, Detroit, MI	Fourth	2004	Open today
California State University, Fullerton, CA	Fourth	2004	Open today
Seattle University, Seattle, WA	Third	2010	Open today
Texas Tech University Health Sciences Center School of Nursing Nurse-Midwifery Program, Lubbock, TX	Fourth	2010	Open today
Baylor University, Louise Herrington School of Nursing, Waco, TX	Third	2011	Open today
Bethel University, St. Paul, MN	Fourth	2014	Open today
University of Pittsburgh, Pittsburgh, PA	Third	2016	Open today

[a] Focused programs for internationally educated midwives to prepare for practice in the United States.

How many of us can actually say we touched . . . or were touched by . . . our founding foremothers? The answer is . . . every single one of us! . . . Every nurse-

midwife in the country can trace their historical roots across four generations of nurse-midwifery education programs . . . whose roots reach back through their program director one, two, or three generations to either Hattie Hemschemeyer at Maternity Center Association (MCA) or Mary Breckinridge at the Frontier Graduate School of Midwifery of the Frontier Nursing Service (FNS). . . . This process of reaching to our roots . . . is vital to understanding where we have come from and what important components of philosophy and the midwifery model of care have stood the test of time in caring for women and childbearing families.[126(p464)]

Of particular note when examining the interconnectedness of nurse-midwives through education programs are two second-generation programs whose histories have received little attention. The Tuskegee School of Nurse-Midwifery and the Flint-Goodridge School of Nurse-Midwifery were programs for black nurse-midwives, whose first directors were graduates of the MCA school.

Tuskegee School of Nurse-Midwifery: 1940s

Lucinda Canty's 1994 master's thesis provides important documentation of the Tuskegee School of Nurse-Midwifery.[127] This school opened in September 1941 to educate black public health nurses as midwives, with the goal of decreasing maternal and infant mortality in the black communities of the South.[128] The school was a joint project of the Macon County Health Department, the Children's Bureau, the Julius Rosenwald Fund, Tuskegee University (although not officially part of the university), and the Alabama State Department of Health. Program directors included two Maternity Center Association graduates, Margaret Thomas and F. Carrington Owens, and a 1944 Tuskegee graduate, Claudia Durham.[129] The program closed in 1946 after 5 years of operation, during which 31 black nurse-midwives graduated. Although information is not available for all of the Tuskegee graduates, Canty's thesis describes the contributions of 10 of the graduates to the nurse-midwifery profession and to the black community. At least 10 of the Tuskegee graduates worked in health care, and most of them trained and worked with "granny" midwives (traditional apprentice-trained community-based midwives who were not nurses). At least four Tuskegee graduates continued their education after graduating from the Tuskegee School, and several served as nursing educators.

One Tuskegee School of Nurse-Midwifery graduate, Maude E. Callen (**Figure 1-8** and **Figure 1-9**, received national attention in 1951 when *Life* magazine published a photographic essay, "Nurse Midwife Maude Callen Eases Pain of Birth, Life, and Death."[130] After graduating in 1943, Callen practiced in rural Berkeley County, South Carolina, an area in which she resided and in which residents experienced high rates of maternal and infant morbidity and mortality, as well as high rates of tuberculosis and other infectious diseases. The *Life* feature presented Callen's work through approximately 30 compelling photographs and accompanying text that described her relationships with community members and other healthcare workers, her demanding schedule in service to her patients, and the healthcare and economic struggles faced by the population in that community. The national notoriety Callen

gained from the *Life* feature contributed to donations that enabled the opening of the Maude Callen Clinic in Pineville, South Carolina.[127]



Figure 1-8 Photograph from *Life Magazine's* 1951 photographic essay about Maude Callen.

© W. Eugene Smith / Contributor/The LIFE Picture Collection/Getty



Figure 1-9 Photograph from *Life Magazine's* 1951 photographic essay about Maude Callen.

© W. Eugene Smith / Contributor/The LIFE Picture Collection/Getty

Linda Janet Holmes, in her report of the history of midwives of color in the ACNM, *Into the Light of Day*, noted that the *Life* article served an important purpose in depicting "a Black woman in a health care professional role in a mainstream publication" and as a "tireless holistic healer who functions as midwife, bondswoman, dietician, and counselor in her backwoods and deeply impoverished South Carolina community." However, Holmes also points out that the *Life* article referred to Callen by her first name—"a common practice at the time to denote lesser status for Black women"—in spite of the women and families in her community calling her "Miss Callen."[131](p11)

Flint-Goodridge School of Nurse-Midwifery: 1940s

Another pioneering education program that has been largely overlooked in the historical record is the Flint-Goodridge School of Nurse-Midwifery in New Orleans, Louisiana, which was open from 1942 to 1943. For a number of years, the superintendent of Flint-Goodridge Hospital had been interested in establishing a nurse-midwifery training program as a way to decrease the high rates of maternal and infant mortality in New Orleans' black population by

training nurse-midwives as replacements for traditional granny midwives.[132] Established using funds from the U.S. Children's Bureau and the Rosenwald Fund, the Flint-Goodridge School of Nurse-Midwifery was affiliated with Dillard University and Flint-Goodridge Hospital. Public health nurse and Lobenstine/Maternity Center Association nurse-midwifery graduate Kate Hyder was selected to lead the Flint-Goodridge program. A Jamaican midwife and MCA graduate, Etta Mae Forte, served as an additional faculty member.[132] On June 15, 1943, nine months after the first two students were admitted, the Flint-Goodridge School of Nurse-Midwifery graduated those two students. It closed shortly thereafter, though the historical record does not include a complete account of the reasons for the school's closure. According to Horch's thesis, a 1943 Flint-Goodridge Hospital report cited the "war emergency" as the reason for closure and stated that the closure was intended to be "temporary."[132(p45)]

One graduate of the Flint-Goodridge School of Nurse-Midwifery was Deola Lange Cyrus; the other graduate's name is missing from the historical record. Cyrus described her work and the role of lay midwives in Louisiana in the 1940s in a *Quarterly Bulletin of the Louisiana State Board of Health* article. Given that she was a formally educated nurse-midwife, Cyrus's favorable assessment of granny midwives is a noteworthy description of the important role of midwives in that place and time:

> *In Louisiana the plantation midwife continues to play an important role in rural community life. She is regarded as a counselor on family problems, a healer of all ills, a leader in civic affairs, a soothsayer, and a liaison between Negro families and the "boss-man." Above all else, she is the one person who can be depended on when the expectant mother's time is nigh. Neither freezing weather, hurricanes, high water, nor torrid heat will prevent the granny woman from making her appearance when she gets the word.*[132(p51)]

Patterns of Education Programs Opening and Closing: 1960s to the Present

In the decades following the inception of nurse-midwifery education in the United States, distinct patterns emerged, including rapid increases in the number of educational programs opening during periods when midwifery was encouraged. The late 1960s and early 1970s were a time of increased acceptance of nurse-midwifery and widespread proliferation of educational programs. Many factors converged to create the need to educate more nurse-midwives, including official recognition by organized obstetrics, increased visibility and involvement of the women's movement and feminism, increased recognition of nurse-midwifery by consumers, nurse-midwives' involvement in federally funded projects, an insufficient supply of obstetricians to care for post–World War II baby boomers, and demonstration of the effectiveness of the obstetric interdisciplinary team concept.

The 1990s witnessed another growth spurt in nurse-midwifery education programs, with an unprecedented 26 programs opening; by the end of the twentieth century, 45 ACME-accredited basic nurse-midwifery education programs existed. This growth was in part a result of states' recognition of the quality and cost-effectiveness of midwifery care and funding of local programs.[126] Growth of programs preparing candidates for the certified midwife credential has

been slower, with two schools providing this master's degree in 2017 and six states licensing certified midwives at this time.

Despite periods of growth in numbers of programs, midwifery educators have always faced challenges in maintaining programs, and many programs have closed after only a few years or decades of operations. Programs have closed for numerous reasons, such as funding challenges or relocation of resources within a department and development of a new focus for the educational institution. Nevertheless, some programs are now more than a half-century old (e.g., Columbia, Yale, Utah). An editorial in a 1959 *Bulletin of the American College of Nurse-Midwifery* provides a useful look then and now at the ebb and flow of nurse-midwifery education programs:

> *In the realm of experimentation, there has been no evidence of fear. Wherever a possibility for educational improvement or a broader scope of influence arose, nurse-midwives have made the necessary adjustments and moved in to try out another facet of service.*[133(p38)]

Foundational Concepts and Innovations in Midwifery Education

The original list of concepts integral to nurse-midwifery practice included a family-centered approach to health care, effective communication, collaboration with other healthcare providers, client education, continuity of care, community resources, and health promotion. In 1985, the following concepts were added: informed client choice, reproductive bioethical considerations, and birth as a normal physiologic process. Facilitation of healthy family relationships and respect for cultural variations were added in 1992. In 1997, these foundational concepts were renamed "Hallmarks of Midwifery," and some additional concepts were included: menses and menopause as normal processes, the therapeutic value of human presence, and care of underserved populations. Educational programs articulate how these hallmarks are threaded through their curricula as part of their accreditation evaluations.[134]

Nurse-midwifery educators have been early adopters of educational innovations in many forms, including modular programs and distance education. Nurse-midwifery faculty at the University of Mississippi Medical Center developed a mastery learning modular curriculum in 1972. Although the modular approach was not a new concept, few programs used the approach to structure the entire curriculum in a student-centered and flexible way. The modular approach has been so durable that the International Confederation of Midwives still promotes its use in midwifery curriculum development.

Distance education has provided another opportunity for midwifery educators to use innovative approaches to meet the needs of midwifery students and, by extension, women and families. This idea was first implemented for nurse-midwifery education in the 1970s, when pilot programs such as Educational Program Associated in California laid the groundwork on which later programs were built. Distance nurse-midwifery education took a quantum leap forward when combined with web-based technologies in the 1990s.[135] A major contributor to the progress of innovative distance learning was the Community-Based Nurse-Midwifery Education Program (CNEP), begun in 1989 and affiliated with the Frontier Nursing Service.[136]

Summary

Since opening the Lobenstine School, nurse-midwives educated in the United Stated have been connected through a lineage of educational programs, including several programs for black nurse-midwives, whose stories are largely absent from the historical record. All nurse-midwifery educational programs have navigated difficult situations to remain viable, and many programs were unable to remain open, resulting in a pattern of educational programs opening and closing. Throughout, midwifery educators have remained committed to disseminating foundational concepts and introducing innovations.

Conclusion

Just as a clinician can provide optimal care for an individual and family by understanding that person's history, so midwives must understand the profession's history. By exploring these themes and topics, today's midwives can enhance their understanding of the profession's roots and the opportunities and challenges midwives have faced for nearly a century. Armed with that knowledge, midwives can optimally guide the profession through the next century and beyond.

References

1. Robinson SA. A historical development of midwifery in the black community: 1600–1940. *J Nurse Midwifery*. 1984;29(4):247-250.
2. Litoff JB. *American Midwives: 1860 to the Present*. Westport, CT: Greenwood Press; 1978.
3. Roush RE. The development of midwifery—male and female, yesterday and today. *J Nurse Midwifery*. 1979;24(3):27-37.
4. Morrison SM, Fee E. Nothing to work with but cleanliness: the training of African American traditional midwives in the South. *Am J Public Health*. 2010;100(2):238-239.
5. Smith MC, Holmes LJ. *Listen to Me Good: The Story of an Alabama Midwife*. Columbus, OH: Ohio State University Press, 1996.
6. Logan OL, Clark K: *Motherwit: An Alabama Midwife's Story*. New York: E.P. Dutton, 1989.
7. Varney H, Thompson JB. *A History of Midwifery in the United States: The Midwife Said Fear Not*. New York: Springer; 2016.
8. Rooks JP. *Midwifery and Childbirth in America*. Philadelphia: Temple University Press; 1997.
9. Dye NS. History of childbirth in America. *Signs*. 1980;6(11):97-108.
10. Fox CG. Toward a sound historical basis for nurse-midwifery. *Bull Am Coll Nurse Midwives*. 1969;14(3):76-82.
11. Ulrich LT. *A Midwife's Tale: The Life of Martha Ballard, Based on Her Diary, 1785-1812*. New York: Knopf; 1990.
12. Dawley K. The campaign to eliminate the midwife. *Am J Nurs*. 2000;100(10):50-92.
13. Perry DS. The early midwives of Missouri. *J Nurse Midwifery*. 1983;28(6):15-22.
14. Manocchio RT. Tending communities, crossing cultures: Midwives in 19th-century California. *J Midwifery Womens Health*. 2008;53(1):75-81.
15. Ortiz FM. History of midwifery in New Mexico: Partnership between curandera-parteras and the New Mexico Department of Health. *J Midwifery Womens Health*. 2005; 50(5):411-417.
16. Smith SL. *Japanese American Midwives: Culture, Community, and Health Politics, 1880-1950*. Chicago, IL: University of Illinois Press; 2005.
17. Rude AE. The midwife problem in the United States. *JAMA*. 1923;81:987-992.
18. Kobrin FE. The American midwife controversy: a crisis of professionalization. *Bull Hist Med*. 1966;40(4):350-363.
19. Van Blarcom CC. Midwives in America. *Am J Public Health*. 1914;4(3):197-207.
20. Ettinger LE. *Nurse-Midwifery: The Birth of a New American Profession*. Columbus, OH: Ohio State University Press; 2006.
21. Dawley K. Doubling back over roads once traveled: creating a national organization for nurse-midwifery. *J Midwifery Womens Health*. 2005;50(2):71-82.
22. Committee on Organization. *Organ Bull*. 1954;1.
23. Committee on Organization. *Organ Bull*. 1955;2.
24. First convention—Kansas City. *Bull Am Coll Nurse Midwifery*. 1955;1(1):1-4.
25. Kroska R. The emblem of the American College of Nurse-Midwives. *J Nurse-Midwifery*. 1973;18(3): 23-24.
26. Hemschemeyer H. Sends message to members. *Bull Am Coll Nurse Midwifery*. 1956;1(2):5-6.
27. Looking ahead. *Bull Am Coll Nurse Midwifery*. 1957;2(1):1-2.
28. Carrington BW, Burst HV. The American College of Nurse-Midwives' dream becomes reality: the Division of Accreditation. *J Midwifery Womens Health*. 2005;50(2):146-153.
29. American College of Nurse-Midwifery. *Articles of Incorporation and By-Laws*. Silver Spring, MD: American College of Nurse-Midwifery; 1955.
30. Keane V. Accreditation and responsibility. *Bull Am Coll Nurse Midwifery*. 1960;5(2):39-41.
31. Cameron J. Why national certification. *Bull Am Coll Nurse Midwives*. 1971;16(4):94-99.
32. Burst HV. Historical perspectives: an update on the credentialing of midwives by the ACNM. *J Nurse Midwifery*. 1995;40(3):290-296.
33. Thomas MW. *The Practice of Nurse-Midwifery in the United States (Children's Bureau Publication No. 436)*. Washington, DC: Department of Health, Education and Welfare; 1965.
34. Woodville L. Descriptive data: nurse midwives—U.S.A. *Bull Am Coll Nurse Midwifery*. 1963;1:30-38.
35. Runnerstrom L. Nurse-midwifery legislation: pro and con. *Bull Am Coll Nurse Midwifery*. 1966;11(2):62-68.
36. Crawford M. Nurse-midwifery: licensure or certification. *Bull Am Coll Nurse Midwifery*. 1968;13(2):18-22.
37. American College of Nurse-Midwives. Legislative scene: position statement on nurse-midwifery legislation. *J Nurse Midwifery*. 1974;19(4):24.
38. Part II: survey findings. *J Nurse Midwifery*. 1976;21(2):5-19.
39. Cuddihy NR. On nurse-midwifery legislation. *J Nurse Midwifery*. 1984;29(2):55-56.
40. Bidgood-Wilson M. The legislative status of nurse-midwifery: trends and future implications. *J Nurse Midwifery*. 1992;37(3):159-160.

41. Bidgood-Wilson M, Barickman C, Ackley S. Nurse-midwifery today: a legislative update, Part I. *J Nurse Midwifery*. 1992;37(2):96-97.
42. Bidgood-Wilson M, Barickman C, Ackley S. Nurse-midwifery today: a legislative update, Part II. *J Nurse Midwifery*. 1992;37(3):175.
43. Osborne K. Regulation of prescriptive authority for certified nurse-midwives and certified midwives: a national overview. *J Midwifery Womens Health*. 2011;56(6):543-556.
44. Osborne K. Regulation of prescriptive authority for certified nurse-midwives and certified midwives: 2015 national overview. *J Midwifery Womens Health*. 2015;60(5):519-533.
45. Corbin H. Historical development of nurse-midwifery in this country and present trends. *Bull Am Coll Nurse Midwifery*. 1959;4(1):13-26.
46. Maternity Center Association. *Twenty Years of Nurse-Midwifery, 1933–1953*. New York, NY: Maternity Center Association; 1955.
47. Cockerham AZ. *A Mission for Mothers: Nurse-Midwifery and the Society of Catholic Medical Missionaries in Santa Fe, New Mexico, 1943–1969*. Unpublished dissertation; 2009.
48. Breckinridge M. *Wide Neighborhoods: A Story of the Frontier Nursing Service*. New York, NY: Harper; 1952.
49. Stone SE. The evolving scope of nurse-midwifery practice in the United States. *J Midwifery Womens Health*. 2000;45(6):522-531.
50. Beasley WBR. The nurse-midwife as a mediator of contraception. *Am J Obstet Gynecol*. 1967;98(2):201-207.
51. Burst HV. Historic perspectives. History of nurse-midwifery in reproductive health care. *J Nurse Midwifery*. 1998;43(6):526-529.
52. Okrent SA. The nurse-midwife in a family planning clinic. *Bull Am Coll Nurse Midwifery*. 1966;11(2):48-54.
53. Okrent SA. Family life education in a family planning clinic. *Bull Am Coll Nurse Midwifery*. 1970;15(3):78-85.
54. Okrent SA. *A Clinical Guide to Oral Contraception*. New York, NY: Shirley Okrent; 1973.
55. Okrent SA. *A Clinical Guide to the Intrauterine Device and the Vaginal Diaphragm*. New York, NY: Shirley Okrent; 1974.
56. Barger MK. Midwifery practice: where have we been and where are we going? *J Midwifery Womens Health*. 2005;50(2):87-90.
57. Fullerton JT. Reflections on nurse-midwifery role and functions. *J Nurse Midwifery*. 1994;39(2):107-109.
58. Shah MA. The nurse-midwife as primary care provider [Editorial]. *J Nurse Midwifery*. 1993;38(4):185-187.
59. American College of Nurse-Midwives. Standards for the evaluation of nurse-midwifery procedural functions. *Bull Am Coll Nurse Midwives*. 1972;18(2):50-51.
60. Avery MD, DelGiudice GT. High-tech skills in low-tech hands: issues of advanced practice and collaborative management. *J Nurse Midwifery*. 1993;38(2 suppl):9S-17S.
61. Thompson JE. Professional maturity or independence? *J Nurse Midwifery*. 1984;29(5):307-310.
62. Schuiling KD, Slager J. Scope of practice: freedom within limits. *J Midwifery Womens Health*. 2000;45(6):465-447.
63. Solenberger ER. Nurses on horseback. *Hygeia*. 1931:633-638.
64. DeMark JB, ed. *Essays in 20th Century New Mexico History*. Albuquerque, NM: University of New Mexico; 1994.
65. Shoemaker MT. Is nurse-midwifery the solution? *Public Health Nurse*. 1946;38:644-648.
66. Greulich B, Paine LL, McClain C, Barger MK, Edwards N, Paul R. Twelve years and more than 30,000 nurse-midwife-attended births: the Los Angeles County + University of Southern California Women's Hospital birth center experience. *J Nurse Midwifery*. 1994;39(4):185-196.
67. Declercq ER, Williams DR, Koontz AM, Paine LL, Streit EL, McCloskey L. Serving women in need: nurse-midwifery practice in the United States. *J Midwifery Womens Health*. 2001;46(1):11-16.
68. Carrington BW. Minority recruitment. *J Nurse Midwifery*. 1981;26(1):1-2.
69. Kennedy HP, Erickson-Owens D, Davis JAP. Voices of diversity in midwifery: a qualitative research study. *J Midwifery Womens Health*. 2006;51(2):85-90.
70. Morrow K. Transcultural midwifery: adapting to Hmong birthing customs in California. *J Nurse Midwifery*. 1986;31(6):285-288.
71. Ross MG. Health impact of a nurse midwife program for the Navajo Indian population, Fort Defiance, Arizona. *Nurs Res*. 1981;30:353-355.
72. American College of Nurse-Midwives. Certified nurse-midwives and the Indian Health Service: a perfect match. Updated 2008. Available at: http://www.midwife.org/acnm/files/cclibraryfiles/filename/000000002226/ihs_packet_june_08.pdf. Accessed April 13, 2017.
73. Levy BS, Wilkinson FS, Marine WM. Reducing neonatal mortality rate with nurse-midwives. *Am J Obstet Gynecol*. 1971;109(1):50-58.
74. Raisler J, Kennedy H. Midwifery care of poor and vulnerable women, 1925–2003. *J Midwifery Womens Health*. 2005;50(2):113-121.

75. Sharp ES, Lewis LE. A decade of nurse-midwifery practice in a tertiary university-affiliated hospital. *J Nurse Midwifery*. 1984;29(6):353-365.
76. Olesker E, Walsh LV. Childbearing among lesbians: are we meeting their needs? *J Nurse Midwifery*. 1984;29(5):322-329.
77. Harvey SM, Carr C, Bernheine S. Lesbian mothers: health care experiences. *J Nurse Midwifery*. 1989;34(3):115-119.
78. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
79. Walker K, Arbour M, Waryold J. Educational strategies to help students provide respectful sexual and reproductive health care for lesbian, gay, bisexual, and transgender persons. *J Midwifery Womens Health*. 2016;61(6):737-743.
80. Carter M. Nurse-midwives in federally funded health centers: understanding federal program requirements and benefits. *J Midwifery Womens Health*. 2012;57(4):365-370.
81. American College of Nurse-Midwives. Certified nurse-midwives (CNMs) and federally qualified health centers (FQHCs): a perfect match. Updated 2010. Available at: http://www.midwife.org/acnm/files/cclibraryfiles/filename/000000004189/fqhc_packet_2010.pdf. Accessed April 13, 2017.
82. Laird MD. Report of the Maternity Center Association Clinic, New York, 1931–1951. *Am J Obstet Gynecol*. 1955;69(1):178-184.
83. Metropolitan Life Insurance Company. Summary of the tenth thousand confinement records of the Frontier Nursing Service. *Frontier Nursing Service Quart Bull*. 1958;33(4). Reprinted in *Bull Am Coll Nurse-Midwives*. 1960;5:1-9.
84. Cockerham AZ, Keeling AW. Finance and faith at the Catholic Maternity Institute, Santa Fe, New Mexico, 1944–1969. *Nurs Hist Rev*. 2010;18:151-166.
85. Nickel S, Gesse T, MacLaren A. Ernestine Wiedenbach: her professional legacy. *J Nurse Midwifery*. 1992;37(3):161-167.
86. Wiedenbach E. *Family-Centered Maternity Care*. New York, NY: G. P. Putnam's Sons; 1958.
87. ACNM statement on home births. *J Nurse Midwifery*. 1975;20(3):15.
88. Jackson ME, Bailes AJ. Home birth with certified nurse-midwife attendants in the United States: an overview. *J Nurse Midwifery*. 1995;40(6):493-473.
89. Lubic RW. Alternative maternity care: resistance and change. In: Romalis S, ed. *Childbirth: Alternative to Medical Control*. Austin, TX: University of Texas Press; 1981:217-249.
90. Rooks JP, Weatherby NL, Ernst EK, Stapleton S, Rosen D, Rosenfield A. Outcomes of care in birth centers. The National Birth Center Study. *N Engl J Med*. 1989;321(26):1804-1811.
91. Jackson DJ, Lang JM, Swartz WH, et al. Outcomes, safety, and resource utilization in a collaborative care birth center program compared with traditional physician-based perinatal care. *Am J Public Health*. 2003;93(6):999-1006.
92. Stapleton SR, Osborne C, Illuzzi J. Outcomes of care in birth centers: demonstration of a durable model. *J Midwifery Womens Health*. 2013;58(1):3-14.
93. Hemschemeyer H. Midwifery in the United States: how shall we care for the million mothers whose babies are born at home? *Am J Nurs*. 1939;39(11):1181-1187.
94. Falson JB. The obstetrician and the nurse-midwife. *Bull Am Coll Nurse Midwifery*. 1959;4(1):8-9.
95. Whitridge J. Nurse-midwife fills a gap in obstetric care. *Bull Am Coll Nurse Midwifery*. 1960;5(2):28-35.
96. American College of Nurse-Midwives. Joint statement on maternity care. *Bull Am Coll Nurse Midwives*. 1971;16(1):22-23.
97. Meglen MC. Nurse-midwife program in the Southeast cuts mortality rates. *Contemporary OB/GYN*. 1976;8:79-97.
98. Meglen MC. Nurse-midwives and the maternity care team. *Bull Am Coll Nurse Midwifery*. 1972;17(3):65-72.
99. Burnett JJ. A physician-sponsored community nurse-midwife program. *Obstet Gynecol*. 1972;40(5):719-723.
100. American College of Nurse-Midwives. *Nurse-Midwifery in the United States: 1976–1977*. Washington, DC: American College of Nurse-Midwives; 1978.
101. Burgin K. CNM/MD relations: cultivating mutual respect. *J Nurse Midwifery*. 1992;37(2):81-83.
102. Vann MK. Professional autonomy for midwives: an essential component of collaborative practice. *J Nurse Midwifery*. 1998;43(1):41-45.
103. Miller S, King T. Professional update. Collaborative practice: a resource guide for midwives. *J Nurse Midwifery*. 1998;43(1):66-73.
104. American College of Obstetricians and Gynecologists, American College of Nurse-Midwives. Joint statement of practice relations between obstetrician-gynecologists and certified nurse-midwives/certified midwives. Updated 2011. Available at: http://www.acog.org/-/media/Statements-of-Policy/Public/sop1102.pdf?dmc=1&ts=20170426T0436213578. Accessed April 13, 2017.
105. Breckinridge M. An adventure in midwifery: the nurse-on-horseback gets a "soon start." *Surv Graph*. 1926;1:25-27,47.
106. American College of Nurse-Midwifery. Summary of minutes of the Executive Board meetings 1956. *Bull Am Coll Nurse Midwifery*. 1957;2(11):11-13.

107. Lanahan CC. Pragmatics of research. *J Nurse Midwifery*. 1976;21(3):14-18.
108. Hart E. Why research in midwifery? [Editorial]. *J Nurse Midwifery*. 1981;26(4):37-38.
109. Diers D, Burst HV. Effectiveness of policy related research: nurse-midwifery a case study. *Image: J Nurs Schol*. 1983;15(3):68-74.
110. Kennedy HP. Reflections on the past and future of midwifery research. *J Midwifery Womens Health*. 2005;50(2):110-112.
111. Fitzpatrick JJ. The clinical nurse-midwife as scientist. *J Nurse Midwifery*. 1988;33(1):37-39.
112. Barger M, Faucher MA, Murphy PA. Part I: Theorists and historical influences. *J Midwifery Womens Health*. 2015;60(1):89-98.
113. Lehrman E. Nurse-midwifery practice: a descriptive study of prenatal care. *J Nurse Midwifery*. 1981;26(3):27-41.
114. Thompson JB. A human rights framework for midwifery care. *J Midwifery Womens Health*. 2004;49(3):175-276.
115. Kennedy HP. A model of exemplary midwifery practice: results of a Delphi study. *J Midwifery Womens Health*. 2000;45(1):4-19.
116. Davis JP. Midwives and normalcy in childbirth: a phenomenologic concept development study. *J Midwifery Womens Health*. 2010; 55(3):206-215.
117. Ickovics JR, Kershaw TS, Westdahl C, et al. Group prenatal care and preterm birth weight: results from a matched cohort study at public clinics. *Obstet Gynecol*. 2003;102(5):1051-1057.
118. Oakley D, Murtland T, Mayes F, et al. Processes of care: comparisons of certified nurse-midwives and obstetricians. *J Nurse Midwifery*. 1995;40(5):399-409.
119. Engstrom JL, McFarlin BL, Sampson MB. Fundal height measurement: accuracy of clinicians' identification of the uterine fundus during pregnancy. Part 4. *J Nurse Midwifery*. 1993;38(6):318-323.
120. Albers LL, Sedler KD, Bedrick EJ, Teaf D, Peralta P. Midwifery care measures in the second stage of labor and reduction of genital tract trauma at birth: a randomized trial. *J Midwifery Womens Health*. 2005;50(5):365-372.
121. Mercer JS, Erickson-Owens DA, Collins J, Barcelos MO, Parker AB, Padbury JF. Effects of delayed cord clamping on residual placental blood volume, hemoglobin and bilirubin levels in term infants: a randomized controlled trial. *J Perinatol*. 2017;37(3):260-264.
122. Yeates DA, Roberts JE. A comparison of two bearing-down techniques during the second stage of labor. *J Nurse Midwifery*. 1984;29(1):3-11.
123. Murphy PA, Fullerton J. Outcomes of intended home births in nurse-midwifery practice: a prospective descriptive study. *Obstet Gynecol*. 1998;92(3):461-470.
124. Beck CT, Gable RK. Comparative analysis of the performance of the Postpartum Depression Screening Scale with two other depression instruments. *Nurs Res*. 2001;50(4):242-250.
125. Seng JS, Sperlich M, Low LK, Ronis DL, Muzik M, Liberzon I. Childhood abuse history, posttraumatic stress disorder, postpartum mental health, and bonding: a prospective cohort study. *J Midwifery Womens Health*. 2013;58(1):57-68.
126. Burst HV, Thompson JE. Genealogic origins of nurse-midwifery education programs in the United States. *J Midwifery Womens Health*. 2003;48(6):464-484.
127. Canty L. *The Graduates of the Tuskegee School of Nurse-Midwifery* [Master's thesis]. New Haven, CT: Yale University School of Nursing; 1994.
128. Kennedy JA. The first graduating class of the Tuskegee School of Midwifery. *J Nat Med Assoc*. 1942;34(3):107-109.
129. Dawley K, Burst HV. The American College of Nurse-Midwives and its antecedents: a historic time line. *J Midwifery Womens Health*. 2005;50(1):16-22.
130. Smith WE. Nurse midwife Maude Callen eases pain of birth, life, and death. *Life*. 1951;31(23):134-145.
131. Holmes LM. *Into the Light of Day: Reflection on the History of Midwives of Color Within the American College of Nurse-Midwives*. Silver Spring, MD: Midwives of Color Committee of the American College of Nurse-Midwives; 2011.
132. Horch J. *The Flint-Goodridge School of Nurse-Midwifery* [Master's thesis]. New Haven, CT: Yale University School of Nursing; 2002.
133. Nurse-midwifery education [Editorial]. *Bull Am Coll Nurse Midwifery*. 1959;4(2):37-38.
134. Avery MD. The history and evolution of the Core Competencies for Basic Midwifery Practice. *J Midwifery Womens Health*. 2005;50(2):102-107.
135. Avery M, Ringdahl D, Juve C, Plumbo P. The transition to web-based education: enhancing access to graduate education for women's health providers. *J Midwifery Womens Health*. 2003;48(6):418-425.
136. Osborne K, Stone S, Ernst E. The development of the Community-Based Nurse-Midwifery Education Program: an innovation in distance learning. *J Midwifery Womens Health*. 2005;50(2):138-145.

CHAPTER

2

Professional Midwifery Today

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Introduction

Midwifery today is a healthcare profession whose members provide care for women throughout the lifespan, with an emphasis on promoting and maintaining health. Modern midwifery is recognized locally as well as globally, with standards for education and practice that make it a profession rather than an occupation or vocation. Safe midwifery practice requires skills, knowledge, and judgment in the provision of health care for women and newborns—but being a safe, legal, independent, interdependent, and successful midwife requires much more than clinical competence. This chapter presents an overview of the profession of midwifery in the United States. Many midwives from the United States work internationally to help train midwives and support women in low-resource nations. A guide to becoming an international consultant is included in **Appendix 2A**.

The Profession of Midwifery

To protect their profession from those who suggest that midwives are undereducated, outdated, or unprofessional, it is important for midwives to be able to answer a critical question: What does it take to be a professional?

According to Ament, "[I]n the United States, the overall objective of protecting the public welfare... is accomplished through three interdependent mechanisms: 1) a prescribed, accredited course of study; 2) national certification; and 3) governmental, usually state or other jurisdiction, licensure."[1] In addition, healthcare professionals are expected to codify their body of knowledge in peer-reviewed journals and textbooks. Midwifery leaders and healthcare policy makers are justified in citing the American College of Nurse-Midwives (ACNM) documents that set standards for education and practice as evidence that midwifery, as practiced by certified nurse-midwives (CNMs) and certified midwives (CMs), is a profession and that these professionals should be licensed in all states and territories.

There is a difference between being a member of a profession and being a "professional." As Kennedy stated, the "midwife's professionalism is a key factor in empowering women during the childbearing process."[2] Kennedy identified three dimensions of midwifery professionalism:

- The *dimension of therapeutics*, which illustrates how and why the midwife chooses and uses specific therapies when providing care
- The *dimension of caring*, which reflects how the midwife demonstrates that she or he cares for, and about, the woman
- The *dimension of the profession*, which examines how midwifery might be enhanced and accepted by "exemplary" practice

Kennedy divided the *dimension of therapeutics* into two qualities that must be held in balance: supporting the normalcy of birth, while simultaneously maintaining vigilance and attention to detail, intervening only when necessary. Kennedy's approach to support the normalcy of birth is often described as the "art of doing 'nothing,'"—that is, appearing calm on the outside while inwardly being actively engaged in data collection and critical thinking.[2]

The *dimension of caring* is demonstrated by "1) respecting the uniqueness of the woman and family; and 2) creation of a setting that is respectful and reflects the woman's needs."[2] Midwives explore and honor each individual woman's personal history and cultural context. These clinicians work in partnership with women for the goal of providing emotional support and strengthening self-confidence of women. Although it is controversial to say that others can empower an individual, such support can increase the self-confidence of a woman and facilitates her own empowerment.

Qualities identified by Kennedy as linked to the *dimension of caring* include "an unwavering integrity and honesty, compassion and understanding, the ability to communicate effectively, and flexibility."[2] Midwives are emotion-workers. For example, exemplary midwives are experts at creating physically and emotionally safe clinical settings; midwives who care for women in labor create a peaceful environment that is conducive to a healthy birth

process, maternal satisfaction, and mother–child bonding in the immediate postpartum period; and midwives caring for women who are seeking a method of contraception use the time to develop a trusting relationship and an environment in which women feel safe enough to disclose honest responses to questions about intimate-partner violence.

The *dimension of the profession* focuses on "the delineation, promotion, and sustenance of midwifery as a professional role."[2] Midwives demonstrate this dimension through evidence-based practice, quality and peer review, continuing education, commitment to and passion for the profession, and nurturing and caring for themselves. The exemplary midwife's focus is not just on the individual woman or birth; in addition, the midwife is driven to foster the profession and advocate for improving women's health care locally and globally.

Professional midwifery in the United States, as represented by certified nurse-midwives, certified midwives, and certified professional midwives, is a dynamic profession. The transition from a focus on individual practice to a focus on the status of a profession within a civil society resembles the evolution of midwifery around the world. In the United States, the scope of midwifery practice has expanded beyond the childbearing years, the core knowledge needed to provide safe care has grown at a rapid pace, and the need to promote interdisciplinary teamwork is well understood. Simultaneously, the civil society has expanded its expectations for all healthcare professionals, and midwives have responded by adopting new standards for education and practice.

Types of Midwives

While an increasing number of individuals are familiar with and use the services of midwives, most midwives have been asked the following questions about their profession: What is the difference between a certified nurse-midwife (CNM), a certified midwife (CM), and a certified professional midwife (CPM)? What is a lay midwife? A direct-entry midwife? An indigenous midwife? While the answers to these questions are evolving, and they can be both confusing and controversial, an exploration of the similarities and differences between midwives is important to the profession and is summarized in **Table 2-1**.[3] The regulatory requirements and scope of practice described here were current as of the time of this text's publication. Laws pertaining to midwifery practice will, like many other statutes, evolve as the political process reflects broader social and cultural changes.

Table 2-1 **Types of Midwives in the United States**

National Midwifery Credentials in the United States	Certified Nurse-Midwife	Certified Midwife	Certified Professional Midwife
Education			
Minimum degree required for certification	Graduate Degree		Certification does not require an academic degree, but is based on demonstrated competency in specified areas of knowledge and skills.
Minimum education requirements for the admission to midwifery education program	Bachelor's Degree or higher from an accredited college or university AND Earn RN license prior to or within midwifery education program	Bachelor's Degree or higher from an accredited college or university AND Successful completion of required science and health courses and related health skills training prior to or within midwifery education program	High school diploma or equivalent Prerequisites for accredited programs vary, but typically include specific courses such as statistics, microbiology, anatomy, and physiology, and experience such as childbirth education and doula certification. There are no specified requirements for entry to the North American Registry of Midwives' (NARM) Portfolio Evaluation Process (PEP) pathway, an apprenticeship process that includes verification of knowledge and skills by qualified preceptors.
Clinical experience requirements	Attainment of knowledge, skills, and professional behaviors as identified by the American College of Nurse-Midwives (ACNM) Core Competencies for Basic Midwifery Education Clinical education must occur under the supervision of an American Midwifery Certification Board (AMCB)-certified CNM/CM or other qualified preceptor who holds a graduate degree, has preparation for clinical teaching, and has clinical expertise and didactic knowledge commensurate with the content taught; > 50% of clinical education must be under CNM/CM supervision		Attainment of knowledge and skills, identified in the periodic job analysis conducted by NARM. NARM requires that the clinical component of the educational process must be at least two years in duration and include a minimum of 55 births in three distinct categories. Clinical education must occur under the supervision of a midwife who must be nationally certified, legally recognized and who has practiced for at least three years and attended 50 out-of-hospital births post-certification. CPMs certified via the PEP may earn a Midwifery Bridge Certificate (MBC) to demonstrate they meet the International Confederation of Midwives (ICM) standards for minimum education.
Education Program Accrediting Organization			
	The Accreditation Commission for Midwifery Education (ACME) is authorized by the U.S. Department of Education to accredit midwifery education programs and institutions. Midwifery education programs must be located within or affiliated with a regionally accredited institution		The Midwifery Education Accreditation Council (MEAC) is authorized by the U.S. Department of Education to accredit midwifery education programs and institutions. The scope of recognition includes certificate and degree granting institutions, programs within accredited institutions, and distance education programs.

Scope of Practice			
Range of care provided	Midwifery as practiced by CNMs and CMs encompasses a full range of primary health care services for women from adolescence beyond menopause. These services include the independent provision of pre-conception care, care during pregnancy, childbirth and the postpartum period, care of the normal newborn during the first 28 days of life, primary care, gynecologic and family planning services and treatment of male partners for sexually transmitted infections CNMs and CMs provide initial and ongoing comprehensive assessment, diagnosis and treatment. They conduct physical examinations; prescribe medications including controlled substances and contraceptive methods; admit, manage and discharge patients; order and interpret laboratory and diagnostic tests and order the use of medical devices Midwifery care also includes health promotion, disease prevention, and individualized wellness education and counseling		Midwifery as practiced by CPMs offer expert care, education, counseling and support to women and their families throughout the caregiving partnership, including pregnancy, birth and the postpartum period. CPMs provide on-going care throughout pregnancy and continuous, hands-on care during labor, birth and the immediate postpartum period, as well as maternal and well-baby care through the 6–8 week postpartum period. CPMs provide initial and ongoing comprehensive assessment, diagnosis and treatment. CPMs are trained to recognize abnormal or dangerous conditions requiring consultation with and/or referral to other health care professionals. they conduct physical examinations, administer medications and use devices as allowed by state law, order and interpret laboratory and diagnostic tests.
Practice settings	All settings - hospitals, homes, birth centers, and offices. The majority of CNMs and CMs attend births in hospitals		Homes, birth centers, and offices. The majority of CPMs attend births in homes and birth centers
Prescriptive authority	All U.S. jurisdictions	New York, Rhode Island, Maine	CPMs do not maintain prescriptive authority; however, they may obtain and administer certain medications in select states
Third party reimbursement	Most private insurance, Medicaid coverage mandated in all states; Medicare, TRICARE	Most private insurance, Medicaid coverage in New York, New Jersey, Rhode Island	Private insurance mandated in 6 states; coverage varies in other states; 13 states include CPMs in state Medicaid plans.
Certification			
Certifying organization	American Midwifery Certification Board (AMCB)		North American Registry of Midwives (NARM)
	AMCB and NARM are accredited by the National Commission for Certifying Agencies		
Requirements prior to taking national certification examination	Graduation from a midwifery education program accredited by the Accreditation Commission for Midwifery Education (ACME); AND Verification by program director of completion of education program		Completion of NARM's Portfolio Evaluation Process (PEP) OR Graduation from a midwifery education program accredited by the Midwifery Education Accreditation Council (MEAC) OR AMCB-Certified CNM/CM with at least ten community based birth experiences

Certification			
	AND Verification of master's degree or higher **CNMs must also submit evidence of an active RN license at time of initial certification*		OR Completion of an equivalent state licensure program include a new sentence below: All applicants must also submit evidence of current adult CPR and neonatal resuscitation certification or course completion.
Recertification requirement	Every 5 years		Every 3 years
Licensure			
Legal status	Licensed in 50 states plus the District of Columbia and US territories as midwives, nurse-midwives, advanced practice registered nurses, or nurse practitioners	Licensed in Delaware, Maine, New Jersey, New York, and Rhode Island	Licensed or otherwise regulated in 31 states (4 states regulate by registration, certificate or voluntary licensure)
Licensure agency	Boards of Midwifery, Medicine, Nursing, Nurse-Midwifery, or Departments of Health	Boards of Midwifery, Medicine, Complementary Health Care Providers or Departments of Health	Boards of Midwifery, Medicine, Nursing, Complementary Health Care Providers or Departments of Health or Departments of Professional Licensure
Professional Association			
	American College of Nurse-Midwives (ACNM)		National Association of Certified Professional Midwives (NACPM)
Other Midwifery Organizations			
	Midwives Alliance of North America (MANA) International Center for Traditional Childbearing (ICTC)		

Note: This table does not address individuals who are not certified and who may practice midwifery with or without legal recognition.

Reproduced with permission from American College of Nurse-Midwives. Comparison of certified nurse-midwives, certified midwives, certified professional midwives clarifying the distinctions among professional midwifery credentials in the U.S. Available at: www.midwife.org/Updated-CNM-CM-CPM-Comparison-Chart-October-2017. Accessed November 15, 2017.[3]

Terms such as "lay midwife" and "direct-entry midwife" lack a common definition. For some, the term "lay midwife" describes an individual who has no formal education as a midwife, while others use this term to refer to a midwife who is not recognized by a government entity. The term "direct-entry midwife" typically refers to a midwife who has entered the profession without first becoming a nurse. In some states, direct-entry and licensed midwife are categories of licensure that co-mingle a variety of midwives and are separate from the licensure of CNMs.

The terms "traditional midwife," "community midwife," and "indigenous midwife" acknowledge the women or men who follow traditional customs as they attend births in their community. These midwives typically work in areas where they have limited access to the

formal education and well-staffed hospitals found in larger cities. Traditional midwives often are or have learned from elders who are influential and trusted because they provide care in concert with local belief systems. Examples include aboriginal midwives from Canada, *comodronas* from Guatemala, and *parteras f*rom Mexico. Traditional apprentice-trained midwives have a large role in provision of maternity care in low-resource nations. Readers interested in learning more about the history and current role of these midwives can refer to the Resources section at the end of this chapter.

As the population of the United States becomes more diverse, one can expect to find traditional midwives serving their newly established communities. It is a mistake to assume that women who receive care from these midwives have poor outcomes or to inject new providers into a community without building a collaborative relationship with traditional midwives. Because hospital-based birthing and fear of complicated birth have become the cultural norm in the United States, criticism of women who chose alternative settings or providers sometimes seems to be common. Nevertheless, women who fear iatrogenic complications, unnecessary cesarean sections, and loss of precious time with their families before and after birth can find evidence to support their concerns about the hospital-based model. Midwives have the opportunity to promote a healthcare system where normal healthy women with uncomplicated pregnancies receive care from well-educated midwives and have access to out-of-hospital childbirth. If complications develop, the healthcare system should be designed so these women have immediate, nonpunitive access to a setting where appropriate care is available. Midwives also have the opportunity to decrease the rate of unnecessary interventions and increase the level of satisfaction for women who give birth in the hospital setting.

Prior to the 1990s, many midwives in the United States who were not CNMs resisted becoming nurses in order to be eligible for midwifery education programs and were opposed to adopting national standards for education and certification. This resistance partly stemmed from concerns that the next steps would be formal education requirements that did not recognize apprenticeship education, followed by required state licensure. Being a “lay midwife” and attending home births is seen by some midwives as the ultimate in independent practice and a source of pride.

The CPM credential, which was first issued in 1994, was originally developed to provide competency-based certification for midwives who were primarily apprentice trained in out-of-hospital birth. The natural consequences of creating the CPM certification examination were the obligation to ensure that those who take the examination meet common standards for education and practice and the creation of a structure within which to discipline those who do not perform in a manner consistent with the standards. Standards for education, certification, and practice for CPMs have been developed, and CPMs continue to seek to expand licensure in states that do not currently have CPM licensure statutes.[4]

At approximately the same time as the development of the CPM credential, in 1991, the board of directors of the American College of Nurse-Midwives (ACNM) endorsed the development of an alternative educational path to midwifery that did not require a nursing degree and that would lead to the CM credential. Over the next 7 years, the requirements to accredit education programs and certify graduates who were not registered nurses were

designed and tested to ensure that, after graduation and certification, one could not distinguish between the knowledge and skills of a CNM and a CM. The first CM credential, which required passing the same certification examination that is offered to nurse-midwives, was issued in 1998.

Although significant variations between CPMs, CNMs, and CMs still exist (**Table 2-1**), the collaborations between the three membership organizations for midwives—the ACNM, the Midwives Alliance of North America (MANA), and the National Association of Certified Professional Midwives (NACPM)—focus more on common values and goals than on differences.[5]

It is increasingly clear that in the United States, where the consumer may find it difficult to distinguish between the various types of midwives who have different credentials, each individual who uses the title “midwife” assumes responsibility for the image of the entire profession. Since the publication of the International Confederation of Midwives (ICM) standards on education and regulation, the professional organizations representing midwives in the United States have engaged in regular discussions about how to meet and support the ICM standards and increase access to high-quality midwifery care in a variety of settings. These discussions have sought to clarify the differences between CNMs, CMs and CPMs. Over time, the differences may decrease.

It is also wise to remember that very few of the midwifery profession’s labor-intensive accomplishments would have moved from internal ideals to cultural norms without consumer support. From the Maternity Center Association (now known as the Childbirth Connection and a program in the National Partnership for Women and Families) to Citizens for Midwifery, consumers have provided inspiration, influence, and financial resources to promote and protect access to midwifery care.[6,7] The list of individuals who created a public demand and stood beside midwifery during some difficult times is long and diverse.

Core Competencies

Core competencies delineate expected fundamental knowledge, skills, and behaviors. These statements serve as the reference point for standardization of the curricula for otherwise diverse education programs and the criteria for accrediting education programs, including those that are not within colleges or schools of nursing. Core competencies inform regulatory agencies, consumers, and employers of what, at a minimum, can be expected from those who graduate from an accredited program.

Using core competencies as a measurement of a student's success enables education programs to recognize that many individuals enter midwifery programs with preexisting skills. It also enables students to focus their studies on new areas, rather than repeating previously learned information. In addition, clear, meaningful competencies reassure the public as well as the midwifery community that all accredited programs graduate well-prepared individuals.

Hallmarks of Midwifery

The *Core Competencies* include six overarching competencies, each of which delineate the skills and knowledge that can be expected of new graduates of ACME accredited midwifery education programs. While each of the competencies describes critical skills and knowledge necessary for midwifery practice, it is the first competency—the *Hallmarks of Midwifery*—that provides the foundation upon which midwifery practice is built. These hallmarks describe the characteristics of midwifery care that are unique to the profession; characteristics that reflect the philosophy of ACNM and highlight components of care that are valued by midwives. A link to the full *Core Competencies* can be found in the Resources section of this chapter, and the *Hallmarks of Midwifery* are presented in **Table 2-2**.[8]

Table 2-2	**Hallmarks of Midwifery**
The art and science of midwifery are characterized by the following hallmarks:	
Recognition of menarche, pregnancy, birth, and menopause as normal physiologic and developmental processes	
Advocacy of non-intervention in normal processes in the absence of complications	
Incorporation of scientific evidence into clinical practice	
Promotion of woman- and family-centered care	
Empowerment of women as partners in health care	
Facilitation of healthy family and interpersonal relationships	
Promotion of continuity of care	
Health promotion, disease prevention, and health education	
Promotion of a public health care perspective	
Care to vulnerable populations	
Advocacy for informed choice, shared decision making, and the right to self-determination	
Integration of cultural humility	
Incorporation of evidence-based complementary and alternative therapies in education and practice	
Skillful communication, guidance, and counseling	
Therapeutic value of human presence	
Collaboration with other members of the interprofessional healthcare team	

Reproduced with permission from American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.[8]

Accreditation

Earning a college degree is a significant measure of success in the United States. A degree represents knowledge obtained in an institution that adheres to national standards that are established to ensure preparation of students who are well educated, by qualified faculty, in their chosen field. Students, employers, and consumers want to know that a degree reflects mastery of a prescribed set of knowledge and skills.

To increase the value of formal education and to protect students from fraud, the federal government and professional education organizations have established standards for institutions of higher education that address the learning environment, content of the curriculum, and qualifications of faculty. In the case of midwifery, the Accreditation Commission for Midwifery Education (ACME) has been recognized by the U.S. Department of Education as a programmatic accrediting agency since 1982 for nurse-midwifery education programs and since 1994 for direct-entry midwifery programs. Maintaining midwifery accreditation standards that are separate from those required for nursing education has allowed the CNM/CM profession to self-regulate, maintain a strong public voice for improving access to midwifery care, and influence public policy that affects the health of women and families.

Certification

While accreditation recognizes educational programs, certification recognizes the individual professional. For CNMs and CMs, certification—that is, passing an examination that measures mastery of fundamental knowledge needed for safe practice and obtained through a recognized program of study—is required to obtain a state license to practice, to obtain hospital staff privileges, and to qualify for reimbursement from public and private health insurance plans. The criteria for taking the examination, the content of the examination, and the requirements for maintaining certification are developed under the auspices of organizations that do not serve as advocates for the profession. While members of the profession can serve as expert advisors, certification organizations work to protect the recipients of care and follow the standards established by the National Commission for Certifying Agencies. CNMs have been certified by examination since 1971, CPMs since 1994, and CMs since 1998.

Licensure and Regulation

The assumption of responsibility for the life and health of another individual—or individuals, in the case of the maternal–fetal dyad—comprises a legal and social contract with multiple contingency clauses. State legislators have responsibility for protecting citizens from unsafe healthcare practitioners and do so by creating state laws that govern practice. State agencies are charged with adopting rules and regulations that further clarify the law. A typical state midwifery practice act will establish (1) qualifications for initial and renewed licensure, (2) scope of practice, (3) relationship with physicians, (4) prescriptive authority with special requirements related to prescribing controlled substances, and (5) definitions of unlawful or unprofessional conduct. Because the laws governing licensure are forged through the legislative process, they are subject to the influence of multiple stakeholders. Additionally, the process of getting a bill passed can be unpredictable. As a result, there is variation in midwifery scopes of practice and requirements for licensure or authorization to practice from state to state.

Many state practice acts are not entirely consistent with the standards of practice endorsed by professional midwifery organizations and taught in accredited midwifery education programs. Despite this discrepancy, the recognized midwife is expected to follow the rules and regulations for whatever method of authorization he or she is granted by the state.

The practice of midwifery is also subject to state and federal laws governing the practice of medicine, Medicare and Medicaid payment, pharmacy, controlled substances, hospital accreditation, and licensing of freestanding birth centers. Going beyond the expectations of regulators, professionals also assume responsibility for setting and meeting their own standards of performance.

Code of Ethics

Ethics is defined as a guiding set of principles that inform actions.[9] Midwives must be well versed in the ethics involved in all healthcare interactions.[10,11] The subject of professional ethics in health care is complex, however, and the brief introduction presented here is not a comprehensive review of this important topic. Additional resources that address health literacy, health numeracy, values clarification, options counseling, the interface between legal and ethical issues, and ways to communicate risk are listed at the end of this chapter. The *ACNM Code of Ethics with Explanatory Statements* provides an in-depth review of common ethical dilemmas faced by midwives and provides guidance for resolving these dilemmas.[12]

An ethical framework for practice, beginning with the concept of accountability, is critical to the continuation of midwifery as an independent and respected profession.[10,11] As defined in the ACNM's *Code of Ethics*, CNMs and CMs have three ethical mandates: to individual recipients of care; for the public good; and to the profession of midwifery. Ethical guidelines encourage self-regulation, foster professional identity, protect midwives and clients, and serve as a measure of professional maturity.[9]

The *ACNM Code of Ethics* was first published in 1990, and the ICM ethical code was introduced in 1993. These documents, as well as MANA's *Statement of Values*, provide guidance for the ethical behavior of midwives in various roles, including caring for women and their families, education, research, public policy, business management, and financial organization of health services.[13,14]

Bioethical Principles

Bioethics is the ethics of working with or caring for human beings. Four broad ethical principles define modern bioethics: respect for autonomy, nonmaleficence (do no harm), beneficence (do good), and justice (**Table 2-3**).[15,16]

Table 2-3 Bioethical Principles

Bioethical Principle	Definition	Midwifery Application
Autonomy	Self-determination	The midwife respects the right of the woman to make decisions regarding her care.
Beneficence	Do good	The midwife acts in a way that promotes the woman's best interests and well-being.
Nonmaleficence	Do no harm	The midwife avoids any actions that cause harm to the woman or her infant.
Justice	Fairness	The midwife accords the woman her due rights and treats all women equally.

Modified with permission from Mighty HE, Fahey J. Clinical ethics in obstetrics and gynecology. In: Barbieri RL, Reece EA. *Obstetrics and Gynecology: The Essentials of Clinical Care.* Stuttgart, Germany: Thieme Publishers; 2010:517-526.[15] https://www.thieme.com. Reprinted by permission.

Respecting an individual's privacy, ensuring confidentiality, and encouraging shared

decision making are all extensions of these bioethical principles. Research has shown that when healthcare providers do not respect these rights, their behavior may be seen as a form of abuse and could lead to psychological trauma for the woman.[16] Healthcare professionals can also experience ethical dilemmas when the application of one ethical principle appears to contradict a second principle.

Privacy and Confidentiality

Protection of a woman's privacy is not just ethical; in most cases it is also mandated by the Health Insurance Portability and Accountability Act of 1996 (HIPAA). When working in collaboration with other healthcare providers, only those aspects of health information that are immediately pertinent to the individual's care should be disclosed, and the woman should be personally notified if the midwife desires to contact a consultant about the woman's health.

Family members, partners, or friends are often present during office visits or in a birthing site. It is important to privately confirm that the woman has given permission to share personal health information when others are present. A midwife must also be diligent about not discussing client information in places where third parties might overhear. Sharing protected health information via emails, faxes, digital records, the Internet, and social media can all lead to inadvertent breaches in confidentiality that can lead to serious negative consequences.

Shared Decision Making

The concept of *informed consent*—a term often used in legal circles—evolved through court decisions and government regulations. It was not until the 1950s and 1960s that U.S. courts began to mandate that consent be obtained before surgery. The 1970s saw an explosion of court rulings that provided legal guidance regarding informed consent.[17,18]

The ethical foundation for informed consent is a woman's understanding of the recommended treatment and her free consent to that treatment.[19] The components that must be shared with the woman to obtain her informed consent are six-fold: (1) diagnosis or assessment, (2) purpose of the proposed treatment or procedure, (3) possible risks of the treatment, (4) possible benefits of the treatment, (5) alternative treatments and the risks and benefits of those alternatives, and (6) possible benefits and risks of not receiving the treatment or procedure. The assumption underlying informed consent is that the individual is capable of understanding the content of the discussion so that self-determination may be protected and supported.

The term "informed decision making" encompasses both informed consent (providing required information) and informed refusal, and reflects a higher ethical standard than that required by law. Foster identified three essential components of informed decision making: (1) *knowing* or understanding, (2) *competency*, and (3) *voluntary* permission.[8] The ethical/moral interpretation of informed decision making centers on autonomous choice: Was the woman able to exercise her right to decide what happens to her body?

Shared decision making with the right to informed refusal is a more recent ethical approach and is consistent with the ACNM philosophy and core values. It is defined as "an approach

where clinicians and patients share the best available evidence when faced with the task of making decisions, and where patients are supported to consider options, to achieve informed preferences."[20-22] This approach has gained favor as healthcare providers rely more on evidence-based practice that includes disclosure of areas where evidence is scarce or contradictory.

Facilitating shared decision making is a process that may take several conversations. Women need time to process information and ask questions. Healthcare consumers may need concrete explanations to understand the information well enough to make a decision. In addition, a woman's health literacy, her cognitive ability, or the presence of physical, intellectual, or developmental disabilities must be taken into consideration. Some women may experience personal circumstances that curtail their ability to make a decision voluntarily—for example, pressure from family members, the midwife, or other care providers; environmental factors such as lack of privacy, lack of funding, or restriction of healthcare access; and/or an abusive relationship. The midwife must assess for these factors and take into account the cultural context when determining whether the woman is able to make a decision on her own volition at any given time.[18,23,24]

Ethical Dilemmas

Professional ethics dictates that a conflict between two or more moral obligations in a particular situation be addressed through deliberate ethical analysis and decision making, including weighing and balancing principles, and preferably involving and achieving consensus among all affected parties. For example, one healthcare provider's attempt to "do good," such as performing a cesarean section for a diagnosis of failure to progress, might be interpreted by the recipient of care as "doing harm"—in this case, performing surgery without adequate time waiting for a vaginal birth.

Equally challenging is the fact that midwifery is a field in which the professional attending a birth has two individuals for whom to provide care, the mother and the fetus, whose interests may not be in equipoise. However, a woman's right to autonomy does not change because she is pregnant. The consensus of modern ethics is that the duty owed to the fetus may be different from that owed to the mother, and the duty to both change depending on the gestational age and maternal condition(s).[24]

Examples of ethical scenarios are presented in **Table 2-4** and **Table 2-5**.

Table 2-4	**Ethical Scenario 1**
A woman with a low-risk pregnancy is "miserable" and requests an induction at 37 weeks' gestation. She is adamant that she will go elsewhere for her care if the midwife will not induce her labor. The midwife validates the woman's feelings and explains the risks of elective induction but supports the position that induction at 37 weeks is not recommended.	
The midwife knows that the benefits to the woman and fetus are maximized (*beneficence*) and harm is minimized (*nonmaleficence*) with spontaneous labor. This professional must weigh this information with the principle of *autonomy*, the woman's right to make an informed decision about her body and fetus.	

Table 2-5	Ethical Scenario 2
A woman presents for her first visit of the pregnancy and tells the midwife that she is uninsured and does not have many financial resources. Normally the midwife counsels women about their genetic testing options in pregnancy at the first visit. It becomes clear to the midwife in the course of their conversation that the woman would not be able to afford any of the costly genetic testing and wonders if counseling should be performed. The midwife decides to counsel this woman in the same manner as any other woman. The midwife's decision to counsel this woman regardless of her ability to afford genetic testing illustrates the principle of *justice*.	

Midwives in the United States

There is no single typical midwife. Nevertheless, demographic data have been collected regularly for more than 50 years for CNMs and provide an insight into both some changes in this profession and some factors that remain the status quo.

Demographic Characteristics

Descriptive data regarding members of ACNM have been collected through membership surveys for more than 5 decades. In the 1963 survey, 229 members were mailed the survey questionnaire and 72% responded.[25] The survey revealed a median age of 41 years for respondents, the existence of six educational programs, and a geographic distribution of practices primarily clustered on the East Coast. Several states or jurisdictions had laws prohibiting midwifery practice. Reports from the 2012–2013 surveys found that the average midwife was a bit older at 50 years; there were more than 40 educational programs; and CNMs practiced in all 50 states, with distribution similar to the general distribution of the U.S. population.[26]

Racial Diversity

One thing that has not changed dramatically over the last half-century is the racial diversity of practicing midwives.[27,28] The population of the United States is increasingly diverse, yet racism, when it is institutionalized, often goes unrecognized by those who are white and racial disparities in maternal and early childhood health outcomes have not significantly improved despite a variety of attempts to increase access to and quality of care.[29] While the midwifery profession has a long history of serving women who are at risk for poor pregnancy outcomes, one solution remains a challenge for the profession—the evidence that race-concordant care can reduce racial disparities in health.[30] While there is recent evidence of an increase in the number of new CNMs/CMs who identify as people of color (14.5% in 2013), the ACNM membership remains disproportionally (more than 90%) white.[26]

The November/December 2016 issue of *Journal of Midwifery and Women's Health* (*JMWH*) provided a comprehensive review of the prolonged struggle to identify and embrace tangible actions that will increase diversity in those who choose the profession of midwifery. The content of this issue of *JMWH* leaves no doubt that changes are needed.[31] Recent actions designed to improve the diversification of the midwifery workforce include the work of the ACNM Diversification and Inclusion Taskforce, which published *Shifting the Frame: A Report on Diversity and Inclusion in the American College of Nurse-Midwives*; provision of antiracist training to the ACNM's board of directors and staff; a theme issue of *JMWH* that explored the problem of racial diversity and the publication of a racial equity toolkit; recent appointment of a member of the Midwives of Color committee to the ACNM's board of directors; and addition of diversity and inclusion as one of the five core commitments for the 2015–2020 strategic plan for the organization.[32,33] The work of the Diversification and Inclusion Taskforce is documented on the ACNM website.[33] Articles remind the reader that

members experience discrimination and exclusion and that overcoming ethnocentrism extends beyond race and ethnicity.

Diversity and Inclusion with Regard to Gender and Identity

Gender is another demographic characteristic that has not changed dramatically over the years. Although "midwife" means "with woman," it does not mandate that the midwife must *be* a woman. Traditionally, though, midwives have been women. Even today, fewer than 2% of CNMs are men, according to recent surveys.[26] Men in midwifery often find themselves as the target of bias from both women and other professionals, including other midwives.

The ACNM Diversification and Inclusion Taskforce has stated that "Differences in identity include but are not limited to race, ethnicity, culture, class, gender and gender identity, sex, sexual orientation, religion, physical and intellectual ability, nationality, citizenship, age, learning style, mental health, professional background, midwifery certification, or academic degree. Diversity also refers to diversity of thought and perspective that come with individual identity."[33] Many of these factors have not been the subject of surveys, but it may be assumed that in some ways midwifery reflects diversity within the general population.

Clearly, midwifery is not a monolithic profession. Some midwives are members of the lesbian, gay, bisexual, transgender, queer, and intersex (LGBTQI) community. They may be religious or not; they may attend temples, churches, or synagogues, or not. Midwives also express a diversity of opinions. For example, in the ACNM there can be civil discussions between members of the caucus focused on reproductive justice and abortion rights and members of the caucus that is pro-life. Midwives do not automatically belong to one political party. Personal scope of practice likewise differs among different midwives.

Midwives have strong opinions, and robust discussion often ensues at their meetings. Nevertheless, respect for others and the goal of caring for women and families should always prevail. Inclusion for all remains an important twenty-first century goal.

Scope of Practice

Midwifery also has a long-standing reputation for preserving a childbirth experience that honors the normal process of birth as well as the transformational power of the childbearing experience.[34] An individual's scope of practice is determined by multiple factors, including legal jurisdiction, institutional policy, location of care, collaborative practice agreements, and individual education and experience.[35] In addition, state laws and facility bylaws may contribute to the definition of the clinical or professional relationship between a midwife and consulting or collaborating physician (**Figure 2-1**). Scope of practice is a complex but dynamic framework for one's clinical practice. For example, a midwife's scope of practice has inflexible boundaries in some aspects (e.g., one cannot perform services that are prohibited by law) and flexible boundaries in other aspects (e.g., advanced clinical skills such as first assisting for cesarean births, may be acquired or needed for a particular setting).[35]

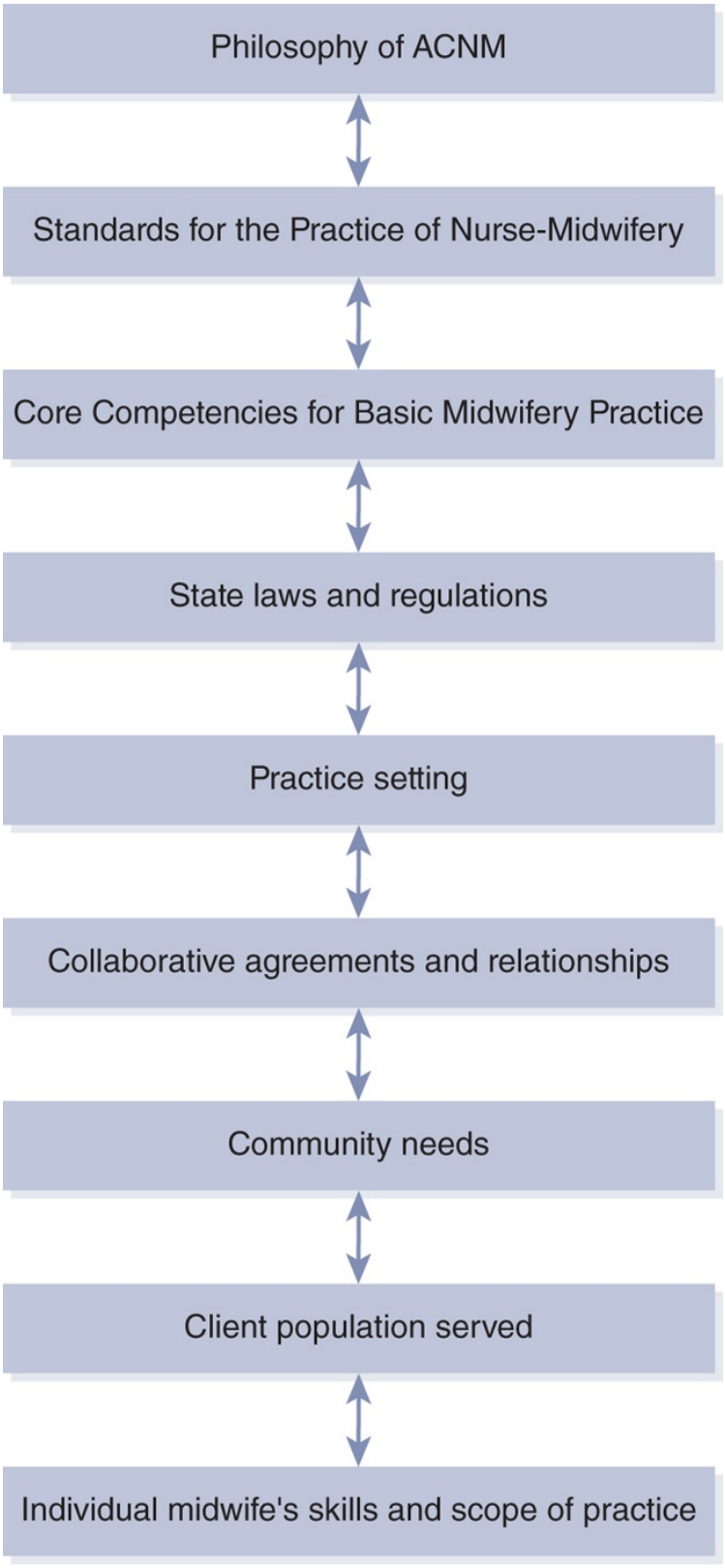


Figure 2-1 Composition of scope of practice.

Modified with permission from Schuiling KD, Slager J. Scope of practice: freedom within limits. *J Midwifery Womens Health*. 2000;45(6):465-471.[35] © 2000, with permission from Wiley.

State laws governing midwifery practice vary. At their best, state laws support independent midwifery practice and collaborative management; at their worst, they require direct physician

supervision of midwives. The rules and regulations governing midwifery practice usually are available on state-sponsored websites. Professional organizations such as ACNM and MANA provide online summaries of all of the states' midwifery laws and work to change laws that do not permit midwives to practice to the full scope of their preparation. A 2015 review by Osborne summarized state regulations regarding prescriptive authority for CNMs and CMs.[36]

Hospital Privileges

Midwives who attend births in a hospital and most birth centers are required to be credentialed and privileged by the healthcare facility prior to caring for women in that setting. Bylaws, as established by the healthcare facility, define the requirements for obtaining privileges, the responsibilities of those who are granted privileges, specific procedures that may be performed by the individual providers, protections offered to those who are privileged, and grounds for removal of privileges. These bylaws may also specify the role and responsibilities of the midwife in relation to the collaborating physician(s) and the responsibilities of the physician in relationship to the midwife. All privileged providers are expected to adhere to institution bylaws, even if the bylaws are more restrictive than the state law.

Practice Patterns

Improving the health of women is a personal, communal, and political responsibility, and midwives work wherever women need them. While many midwives attend births and provide women's health services, they may also work as entrepreneurs, policymakers, and educators. In all of these roles, midwives collaborate with a variety of team members.

In clinical practice, midwives may work for large hospitals or healthcare systems in metropolitan areas, in small private practices in rural communities, and anywhere in between. Midwives may attend births in homes, freestanding birth centers, or hospitals. They may be self-employed in a private business, or they may be employees of physicians or healthcare organizations. They provide care to women from across the socioeconomic spectrum. CNMs and CMs can provide primary health care to women or can limit their practice to women with needs that are age or condition specific, such as family planning, infertility, menopause, incontinence, or pelvic pain. CPMs are typically licensed exclusively to provide maternity care.

Since the 1960s, the majority of CNMs, and now CMs, who attend births have done so in hospitals and freestanding birth centers, whereas the vast majority of CPMs have attended births in homes or freestanding birth centers. Although these trends may continue for a while, the future may present more workplace opportunities for all midwives.

With so many opportunities, the typical midwife searches for a position that is a good match to his or her experience, personality, skill set, and lifestyle. Many midwives become employees of a practice or facility. When considering an employee position, one of the first actions is for the midwife to perform a personal evaluation: Which work and lifestyle factors are important to the individual midwife? Which skills/talents would be important to stress to a prospective employer? What do employers in the area need or want? When evaluating the positives and negatives of any job, it is important to review various aspects of the business that may contribute to success or frustration—for example, availability of and relationship with physicians and other providers (e.g., dieticians, physical therapists), ancillary support (e.g., billing, office flow), reimbursement for professional expenses (e.g., licenses, certification, and continuing education), payment for malpractice premiums, availability of student loan payments, and retirement benefits.

Malpractice Insurance Coverage

Although relatively uncommon, inadequate malpractice coverage can lead to bankruptcy for a midwife. A midwife who is employed by a large healthcare center or university may be covered by the facility's umbrella policy or, alternatively, may be "indemnified" because the company chooses to self-insure so that all employees who are practicing within their job description have what is considered equivalent to malpractice coverage. For most other midwives, two basic kinds of malpractice insurance policies are available: "claims made" and "occurrence."[37,38] Although more difficult to find, the best policy is occurrence, because it covers a midwife as long as the event occurred when the midwife was covered. Claims-made insurance is more common, but covers the midwife only while the policy is in effect or until a

"tail" is purchased. A midwife with a claims-made policy should purchase tail coverage if she or he will no longer be seeing clients under the terms of that policy, but may be able to obtain prior-acts coverage if a new policy is activated.

A midwife needs to know exactly how she or he will be covered for a malpractice claim that might occur during or after employment. Because the statute of limitations for filing a claim for injury related to childbirth may extend for 18 to 20 years, it is critically important that midwives keep copies of their proof-of-insurance forms until they are well past retirement from clinical practice. In addition, when midwives apply for hospital privileges, they are often asked to provide proof of prior malpractice insurance coverage. Members of the American College of Nurse-Midwives have access to a detailed *Professional Liability Resource Packet* and several liability risk reduction statements.[38] These statements identify conditions that pose a higher risk for a professional liability claim and offer risk reduction strategies to enhance health care, prevent claims, or increase the defensibility of a claim. **Table 2-6** summarizes malpractice insurance for midwives.

Table 2-6 Informed Decision Making When Purchasing Malpractice Coverage

Terms	Explanation	What the Midwife Needs to Know
Indemnification	Large healthcare organizations can be self-insured, meaning they indemnify employees who are named in a malpractice lawsuit. Coverage typically does not extend to work settings not owned by the organization.	What are the payout limits of the policy? The midwife is covered only when her or his actions are consistent with the job description. Is the policy an occurrence or claims-made policy? Pros and cons of purchasing a second policy?
Malpractice policy	Purchased from private insurance companies. Individuals can purchase as an individual, and sometimes midwives are covered as an employee of a physician. Need to be clear regarding the circumstances when the policy will end.	Do I need and can I get prior-acts coverage? Do I need to have my own policy or am I named as an employee on a physician policy? Is this an occurrence policy or a claims-made policy? I have an occurrence policy now; can I purchase prior-acts coverage under my new policy? Can I purchase a tail when I no longer need the policy? If yes, how is that priced?
Claims-made coverage	The insured has malpractice coverage only for claims that are made when the policy is in effect.	Can I purchase a tail when I no longer need this policy? If yes, how is that priced?
Occurrence coverage	The insured has malpractice coverage for any claims about care that occurred when the policy was in effect.	If employment ends, coverage for future events ends but coverage is still in place for events that occurred when the policy was in place. Tail coverage should not be needed.
Going bare	The provider decides to not carry malpractice insurance.	While some consider this approach unethical, the rising cost of premiums often brings up this discussion. Two major concerns need to be considered: (1) Your personal or family savings and property may be at risk and (2) if you apply for hospital privileges in the future, a period of clinical practice without malpractice coverage may lead to denial of privileges.
Limits of	Professional liability policies have	Do state law and/or hospital bylaws dictate the minimum

coverage	two sets of limits: a per claim or incident limit and the aggregate limit that the insurer will pay during the policy period (usually one year).Example: $1 million/$3 million	coverage a midwife must carry?

Modified with permission from American College of Nurse-Midwives. ACNM professional liability resource packet. Available at: http://www.midwife.org/index.asp?bid=59&cat=5&button=Search&rec=134. Accessed June 3, 2017.[38]

The Midwife as an Employee

Whether considering one or multiple job opportunities, the midwife should analyze a number of factors that will influence his or her job satisfaction, some of which can be found in **Table 2-7**. This table is designed for a midwife who is interested in joining an existing practice as an employee. The weight of any of the factors will vary based on the individual midwife's desires and needs. The factors are not presented in any specific order, nor is this an exclusive list.

Table 2-7 Factors to Clarify When Considering Joining a Practice

Practice Characteristics	Compensation/Benefits
Location	Salary
Philosophy	Productivity
Clientele (volume, demographics, current outcomes of care)	Requirements/bonuses/overtime
Practice providers (physicians, midwives, nurse practitioners, others, and culture of collaboration)	Payment/nonclinical responsibilities
Support staff (billing, assistants in office, marketing)	Ability to own part of the practice
Clinical hours	Malpractice insurance (type, tail if needed)
Environment (equipment, facilities, financial stability)	Vacation/paid time off
Birth facility	Maternity/paternity leave (if appropriate)
Orientation plan	Other professional benefits (continuing education units, dues, licenses, parking, smartphone)
Time needed for credentialing, insurance networks	Health insurance, retirement
	Student loan repayment

If the prospective employer does not offer a formal contract, asking for confirmation in writing of offered remuneration and job specifics is wise. If the midwife is asked to sign a contract, it is wise to consult with an attorney. Even if a contract is considered non-negotiable, the midwife should thoroughly understand the content prior to signing it. **Table 2-8** provides a list of topics that generally should be negotiated prior to accepting a position and that should appear in a contract.[39,40]

Table 2-8 Topics Usually Found in a Contract

Title of position
Responsibilities, including scope of practice, currency of credentials, expectations of volume and hours

Compensation and benefits, including bonuses, productivity, professional/business expenses, tuition reimbursement, health and malpractice insurance, and paid time off
Duration and requirement for renewal of employment/contract
Reason for termination by employer and/or employee
How to alter or update the contract
Additional Topics That May Be Included
Non-compete clause[a]
Partnership arrangements (buying in or out of a practice)
Termination without cause [a]
Formulae (for bonuses, productivity, quality-based, patient satisfation, and profit-based)
Ownership of records upon dissolution/termination

[a] Not recommended but often included and may be non-negotiable.

Based on Buppert C. *Nurse Practitioner's Business Practice and Legal Guide.* 6th ed. Burlington, MA: Jones & Bartlett Learning; 2018[39]; Slager J. *An Administrative Manual for Midwifery Practices.* 4th ed. Washington, DC: American College of Nurse-Midwives; 2016.[40]

The Midwife as an Entrepreneur

Most midwives consider midwifery to be a vocation. Thus, it can be challenging to think of midwifery as "a business"—yet all midwives need to understand the basic principles of running a successful business. There is a growing need for midwives to become accomplished administrators and business managers.

Many midwives have, either independently or in groups, become business owners. The opportunity to avoid the limitations imposed by the business model or clinical guidelines developed by others, such as physicians, hospitals, and community clinics, can be very tempting, and in some cases, it may be a necessity. While many midwifery-owned businesses have succeeded in spite of inadequate planning or limited resources, the advice offered by successful entrepreneurs is consistent—namely, consult experts, invest in marketing, develop competence in billing, and collect data. Each of these aspects of running an independent midwifery practice is an important factor that can facilitate long-term success.

Business Advice from Experts

It is unwise to open a business without seeking the expertise of, at a minimum, an attorney and an accountant. The legal structure of a midwifery business (e.g., sole proprietorship, partnership, or limited liability company) will have short- and long-term personal and financial consequences. Midwife business owners should be experts on the laws and regulations that govern midwifery practice, but should also know how the laws governing medical practice, the corporate practice of medicine, and pharmacy regulations might affect their plans. Midwives providing care during out-of-hospital births must comply with health department regulations, birth center requirements, building codes, and a variety of business regulations. Midwives who employ others must determine how they will compensate those

employees and follow the relevant employment tax codes and antidiscrimination policies. Beyond malpractice insurance coverage, new business owners are often surprised to learn how many insurance policies need to be purchased and how many business contracts need to be finalized. In all of these areas, good advice can save money, protect investments, and enable midwives to provide high-quality care for women.

Preparing a business plan and seeking guidance from an accountant on the costs of doing business provide clarity for all involved and are requirements when seeking loans to help establish a business. Common elements found in a business plan are listed in **Table 2-9**.[40] The time spent attending to details and establishing a reporting system that provides regular feedback on revenues versus expenses is a good investment, as such a system provides a way to measure success for the entire team and relieves the pressure when the unexpected happens.

Table 2-9	Content for a Typical Business Plan
Cover page with name of business and contact information	
Description of practice	
Services	
Clientele	
Relationships	
Company strategies	
Market research	
Fiscal outlook	
Plans (marketing, operating, and financial)	
Anticipated team and schedule	

Based on Slager J. *An Administrative Manual for Midwifery Practices.* 4th ed. Washington, DC: American College of Nurse-Midwives; 2016.[40]

Given that independent business ownership is valued highly in the United States, many types of support exist for small business owners, including information on how to formulate business plans and where to apply for small business loans. Midwives who are business owners often agree to mentor the next entrepreneur. The Business Section of the ACNM Division of Standards and Practice is committed to sharing information, providing support to midwives interested in the business aspects of midwifery practice, and increasing the number of midwife-owned services. The Business Section partners with ACNM to sponsor the annual conference Midwifery Works, and it has published an *Administrative Manual for Midwifery Practices*.[40] The American Association of Birth Centers also offers workshops on how to open a birth center, where many of these concepts are covered in depth. Other business guides exist in areas outside of midwifery, which may provide additional useful information.[39]

Midwifery services that are not independent businesses still have business and administrative work that must be addressed. No matter the number of employees, a midwifery service has to reach an agreement on scheduling, compensation, records management, monitoring of financial statements, negotiation of collaborative agreements, peer review, and strategies to handle personal and professional adversity. While responsibilities for the success

of the service are shared, there needs to be a designated leader or service director, who serves as the primary contact, assumes responsibility for participating on department or corporate-level committees, is able to describe the success of the service in corporate terms, and knows how to move an agenda forward within the organization. Midwives place a high value on building relationships with women and on positive feedback from the individuals for whom they provide care. Those skills can be extrapolated into the business arena and will serve the midwives well.

Data Collection

Midwives should not forget the lessons learned from Mary Breckenridge, who gathered data about the population she wanted to serve prior to opening the Frontier Nursing Service in 1925. Ms. Breckenridge understood the power of data: baseline descriptive data before opening a service, descriptive and outcome data from the first and every subsequent day of operation, analysis of data to identify relevant findings, and dissemination of the findings. A number of readily accessible mechanisms for collecting and collating practice-specific and national data exist that describe the care provided by midwives. Members of ACNM can join in the ACNM Benchmarking Project,[41] which allows participants to examine their practices and compare them to other like practices across the United States. The MANA Division of Research, with its MANA Stats system, and the American Association of Birth Centers, with its Perinatal Data Registry (PDR), both have developed web-based data collection tools that can be used by individuals and contribute to a national database on the outcomes of midwifery care in all settings.[42,43]

Marketing

Many advisors encourage early attention to a marketing plan. Without a coherent, consumer-friendly message about the services offered and an identified medium for reaching the target population, the business may not have enough clients to sustain itself. Not every practice can cover the cost of a logo and four-color brochures, but all midwives can develop marketing skills. For example, the organized, scientific, lecture approach may intimidate some women, while others may look for messages that midwifery practice is evidence based and provides adequate safeguards in the event of major complications.

Today social media may be the most effective, and least expensive, marketing strategy. Women now expect practices to have an up-to-date website. Regular tweets from midwives can remind women of healthy behaviors. Facebook allows for a robust conversation among women about health issues. To be successful, practices should engage in a variety of social media, while maintaining awareness of all of their clients' privacy.[44,45]

Professional organizations are often a source of marketing advice and materials. Several organizations are involved in national marketing campaigns that can be adapted to local settings, such as the *ACNM Healthy Birth Initiative* (summarized in **Table 2-10**).[46]

Table 2-10	**ACNM Healthy Birth Initiative: Instruments for Use in Practice**[a]

Audience	Component
For midwives and other providers	An interactive online toolkit named *Tools for Optimizing the Outcomes of Labor Safely* (BirthTOOLS), which focuses on promotion of physiologic birth. The toolkit includes the evidence, resources, protocols, and other materials.
For women	Free handout titled *Normal, Healthy Childbirth for Women and Families: What You Need to Know.* Available in Spanish and English. Designed to help consumers understand and promote evidence-based maternity care.
For hospital policymakers, payers, and other organizations	Free handout titled *Birth Matters,* which provides an overview of implementation of strategies to promote physiologic birth from their perspective.

Abbreviation: ACNM, American College of Nurse-Midwives.

[a] Several professionally printed handouts are available for a fee from ACNM.

Based on American College of Nurse-Midwives. Healthy Birth Initiative. Available at: http://www.midwife.org/ACNM-Healthy-Birth-Initiative. Accessed March 28, 2017.[46]

Billing for Services

A midwifery practice cannot continue to exist without financial stability. One of the first financial lessons in the healthcare business is that what is billed for a service is never what the insurance company will pay. Actual reimbursement rates are always lower—a factor that should be taken into consideration when calculating a budget. Whenever the services provided by a midwife are billable, the midwife must clearly document the services provided and complete the steps needed to initiate the billing process. The midwife is responsible for fulfilling the requirements for documentation that support the billing codes. For example, the amount paid for an examination will vary based on the intensity of the examination as measured by the number of systems included in the physical assessment, the types of problems identified, and the amount of time spent providing and coordinating care. If this content is not thoroughly documented in the healthcare record, payment may be reduced or even denied.

However the billing is performed, the owner(s) of the practice is (are) responsible for establishing a system of checks and balances that monitors the accuracy and timeliness of the billing process and limits the opportunity for embezzlement or insurance fraud. The time and money spent establishing a viable healthcare record and billing systems are necessary outlays to ensure the ongoing success of the business. Even midwives who are employees should be informed about the business revenue and business expenses so they will be well informed about the financial stability of the practice.

Other Roles for the Modern Midwife

Modern midwives usually fulfill multiple roles at any one time. Some of these roles not only promote the care of women and newborns, but also support and advance the profession itself. Among these roles are those of being a policymaker, an educator, and a team member. Any or all of these roles may be assumed as a midwife provides direct care to women and newborns.

The Policymaker

The building blocks of the midwifery profession (standards for education, certification, and practice) are key policy decisions with far-reaching influence. Federal, state, and institutional policies determine which healthcare services and birth settings are available to women as well as who will be reimbursed and at what rate. Which education programs receive government funding to support faculty and students is also a matter of policy. Hospitals, clinics, and employers all write policies that influence access to and provision of midwifery care. With so much at stake for the profession and the women cared for by midwives, every midwife needs to be knowledgeable about and engaged in policymaking processes.

Professional organizations remain dependent upon a high volume of work from members to keep policies relevant. Meeting the policy needs of the profession primarily represents a labor of love and a dogged determination to turn a vision into reality.

Many of the midwives who successfully work in policy did not initially see the need for this work, doubted their abilities, or hoped someone else would do it.[47] It is reassuring to know that becoming a policymaker is a learned behavior; thus, the midwifery profession is filled with successful midwife role models, and guidance on how to make this transition is available (**Table 2-11**).

Table 2-11 How to Influence the Policymaking Process: Volunteer, Observe, and Practice

Institutional policies: Write policies for your midwifery practice, hospital, and/or local midwifery organization.
Is there a template to follow?
What must be done to have the policy approved?
Legislative policies: Observe legislative policy in action by attending a hearing that addresses a regulatory issue that affects your practice.
Who seems most effective and why?
What is common etiquette and the standard for appearance?
Was the speaker effective? How did you know?
How did the committee respond?
Identify a mentor.
Come prepared and speak a language the audience can understand.
Create the draft for discussion so others have to respond to your ideas.
Stay on topic.
Know your strengths.
Offer a lived experience.
Be the voice of a midwife or support a woman or family who agrees to speak.
Know your opposition and do not attend alone.

Leave the door open for your return.
- Get help preparing statements.
- Make friends in the room.
- Defer to other experts.

If you can't do policy, support your colleagues who can do this work, including financially.

Modified with permission from Williams DR. We need to say in unison: we are midwives and we do policy! [Editorial]. *J Midwifery Womens Health.* 2008;53(2):101-102.[47] © 2008, with permission from Wiley.

In spite of many past successes, much policy work remains to be done in relation to midwifery. Some physician associations are opposed to laws that recognize advanced practice clinicians and midwives as independent providers; instead, they advocate for physician supervision. Many state laws governing the practice of midwifery need to be changed to permit independent practice. Major decisions are also looming:

- Should CNMs seek more midwifery practice acts that are separate from nursing and include their CM colleagues or stay under the advanced practice registered nurse (APRN) umbrella?
- Can CNMs/CMs and CPMs be licensed under the same practice act?
- Should CPMs be required to earn a college degree?
- Should CNMs/CMs be required to earn a doctoral degree?
- Will the U.S. Congress pass healthcare legislation that moves the profession forward or backward? (Either outcome is always a possibility.)

The Educator

All midwives are educators. Policymakers, potential employers, and consumers need to learn what is unique and valuable about the midwifery approach to care. Many women seek out midwives because they want to learn more about how to care for their own bodies and how to safely prepare for puberty, pregnancy, menopause, and all the points in between. Consumer-oriented materials often are used for this purpose, and many materials are being written by midwives. For example, *JMWH* regularly publishes a health education handout series titled *Share with Women*. These copyright-free handouts, which are targeted to women, review important clinical topics using appropriate language and illustrations for all levels of health literacy. The handouts are also available in Spanish.

Because many midwives are committed to "midwifing" students while these neophytes develop skills in the cognitive, affective, and psychomotor domains, the midwifery profession continues to flourish. The legacy of midwifery also depends on socialization of midwifery students into the role and responsibilities of the midwife. For those who choose an academic career, there are more than 40 midwifery education programs accredited by ACME with numerous midwives on faculty. Midwives who serve as faculty for schools of nursing and medicine have a unique opportunity to prepare the next generation of healthcare providers who respect women's healthcare decisions, understand that midwifery care occurs in a variety of settings, and welcome the opportunity to collaborate with midwives.

The Interprofessional Team Member

All healthcare providers work within a healthcare system that includes professionals who have different scopes of practice, different professional cultures, and different professional roles. Being a team member in the healthcare system does not imply assumption of a subservient role: In some situations the midwife is the team leader, and in others the midwife is a colleague on an interprofessional team. The factors that make interprofessional relationships work well are critically pertinent when a woman develops complications or conditions that extend beyond the scope of midwifery practice. Although it has long been recognized that interprofessional teams provide better care than single-disciplinary groups for individuals with complex healthcare needs,[48,49] interprofessional collaboration and communication have only recently been the focus of research, education, and clinical initiatives.[50,51] Guiding principles of interprofessional team–based care are summarized in **Table 2-12**.

Table 2-12	Guiding Principles of Team-Based Care
The team has a shared vision.	
Role clarity is essential to optimal team building and team functioning.	
All team members are accountable for their own practice and to the team.	
Effective communication is key to the creation of high quality teams.	
Team leadership is situational and dynamic.	

Based on Jennings J, Nielsen P, Buck ML, et al. Executive summary: collaboration in practice: implementing team-based care: report of the American College of Obstetricians and Gynecologists' Task Force on Collaborative Practice. *Obstet Gynecol.* 2016;127(3):612-617.[51]

In the 1999 ground-breaking report from the Institute of Medicine (IOM), *To Err Is Human*, it was estimated that 45,000 to 98,000 individuals die each year in U.S. hospitals due to healthcare errors.[52] Subsequent safety reports have highlighted poor communication and inadequate team coordination as the source of many of these errors. For example, a Joint Commission sentinel event analysis on preventing infant death and injury during birth identified communication problems as the root cause of the healthcare delivery error in 72% of the cases analyzed.[53] In the same analysis, 55% of the organizations studied cited organizational culture, including "hierarchy and intimidation, failure to function as a team, and failure to follow the chain-of-communication," as commonly encountered barriers to effective communication and teamwork.[53]

In the years following these publications, much work has been done to identify ways to foster and support teamwork in healthcare delivery. Successful interprofessional collaboration in care of pregnant women, for example, has been associated with improved outcomes, a high degree of maternal satisfaction, fewer cesarean sections, and lower costs.[54]

According to the International Confederation of Midwives' (ICM) *Essential Competencies for Basic Midwifery Practice*, "The midwife . . . works collaboratively (teamwork) with other health workers to improve the delivery of services to women and families."[55] Moreover, "[t]he midwife has the skill and/or ability to . . . identify deviations from normal during the course of

pregnancy and initiate the referral process for conditions that require higher levels of intervention."[55]

ACNM recognizes that midwives are independent practitioners who function within a complex healthcare system, which includes collaboration with multiple healthcare professionals, to ensure the health and safety of women and their newborns.[56] The levels of collaborative management as defined by ACNM include consultation, collaboration, and referral, and the definitions for each of these levels often serve as guidelines for similar language within state laws and hospital bylaws. While the ACNM definitions address the midwife–physician relationship, the expertise of many other healthcare professionals may be needed to provide the best care possible. It is imperative that all members of the team understand their role with any particular individual (**Table 2-13**).[57]

Table 2-13 The Continuum of Collaborative Management in Midwifery Care

Level of Collaborative Management	Definition	Primary Responsibility for Care	Midwife's Role	Collaborator's Role	Comments
Consultation (with a provider of any specialty)	"The process whereby a CNM or CM seeks the advice or opinion of a physician or another member of the health care team."	Midwife	Primary provider	Advisor/consultant	Prepare for the consultation. Know the woman's health history. Review the basics for management of the diagnosis or problem. Understand the social and psychosocial factors underlying her health. Understand the practice setting and scope of practice. Remember the Midwifery Management Process.
Collaboration	"The process whereby a CNM or CM and physician jointly manage the care of a woman or newborn who has become medically, gynecologically, or obstetrically complicated."	Co-management (depending on the severity of the complication, the midwife may remain the primary care provider)	Normal processes, coordination of care, continuity with the individual	Care for the maternal, gynecologic, or neonatal complications	Use interprofessional communication techniques such as SBAR and closed-loop communication. Clearly delineate roles to ensure all aspects of the POC are considered. Communicate with the woman and her family about the relationship.
Referral[a]	"The process by which the CNM or CM directs the client to a physician or another health care professional for management of a particular problem or aspect of the client's care."	Physician or other referral provider	Coordination of care, timely and full transfer of care, continuity of services	Assumes the primary responsibility for care of the individual	Ensure that referral/transfer is the best POC for the individual. Ensure that the woman understands that she has been transferred to another provider's care and that she has access to appointment and contact information. Consider the potential problem of abandonment of the woman and/or "punting" of difficult-to-care-for women. Use interprofessional communication techniques such as the handoff.

Abbreviations: CM, certified midwife; CNM, certified nurse-midwife; POC, plan of care; SBAR, Situation, Background, Assessment, Recommendation.

[a] Referral in this continuum refers to transfer of care. Referral in the context of insurance is providing an individual with a reference to a specialty provider.

Based on American College of Nurse-Midwives. Position statement: collaborative management in midwifery practice for medical, gynecologic, and obstetric conditions. 2014. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000058/Collaborative-Mgmt-in-Midwifery-Practice-Sept-2014.pdf. Accessed June 3, 2017.[57]

The 2011 ACNM and American College of Obstetricians and Gynecologists (ACOG) *Joint Statement of Practice Relations Between Obstetrician-Gynecologists and Certified Nurse-Midwives/Certified Midwives* declares that "health care is most effective when it occurs in a system that facilitates communication across care settings and among providers."[58] NACPM and MANA have published documents that address the relationship between CPMs and physicians.[4,59] In these documents, midwifery practice is described as autonomous and CPMs are expected to collaborate, refer, and transfer care in critical situations. Essential components

of communication and teamwork are summarized in **Table 2-14**.[60-62]

Table 2-14	Essential Components of Successful Collaboration and Teamwork[a]
Professional competence in each member of the team (common body of knowledge, shared language, similarities in treatment modalities)	
Common orientation to the woman or newborn as the primary unit of attention	
Shared mental model: every member of the team can anticipate and predict the needs of the others	
Recognition and acknowledgment of interdependence among all members of the team	
Interprofessional respect and mutual trust	
Formal system of communication between providers	
Effective communication based on the goal of reaching consensus (an interest in solutions that maximize the contributions of all parties)	
Mutual performance monitoring (identification of mistakes and provision of feedback within team to facilitate self-correction)	
Identified team leader for each situation	
Situation monitoring and adaptability as the situation changes	
Ability to shift work responsibilities as needed to under-utilized team members	

[a] This list is compiled from different analyses of essential characteristics for teams in general and for teams in specific urgent or emergency situations. It is not designed to be complete or the components placed in rank order; rather, the intent is to identify some characteristics that are essential for successful interprofessional team function. Midwives are always members of interprofessional teams.

Based on Interprofessional Education Collaborative Expert Panel. *Core Competencies for Interprofessional Collaborative Practice: Report of an Expert Panel.* Washington, DC: Interprofessional Education Collaborative; 2011[60]; Ivey S. A model for teaching about interdisciplinary practice. *J Allied Health*. 1988;17:189-195[61]; King TL, Laros RK, Parer JT. Interprofessional collaborative practice in obstetrics and midwifery. *Obstet Gynecol North Am*. 2012;39:411-422.[62]

Teamwork and communication are skills that can be learned.[63-65] Although healthcare outcomes following simulation training have not yet fully been determined, it appears that simulation training improves teamwork, team coordination, and interprofessional communication.[66-68]

The U.S. Agency for Healthcare Research and Quality (AHRQ) has developed a series of materials and training curricula, collectively titled TeamSTEPPS, that can be used in healthcare settings to help foster successful teamwork.[63,64,69] The TeamSTEPP curricula emphasize the development of four core competencies: communication, mutual support, situation monitoring, and leadership.

Communication Techniques for Successful Collaboration

Direct and deliberate communication techniques include SBAR, closed-loop communication, and the handoff. SBAR—an acronym for **S**ituation, **B**ackground, **A**ssessment, and **R**ecommendation—is a structured communication tool that has been shown to significantly improve the quality of communication between healthcare providers and to reduce errors.[67] The SBAR approach omits the nonessential elements of a woman's history, distills the most

pertinent information, and clarifies what is needed. The midwife can use the SBAR approach to obtain a consultation from a specialist (**Table 2-15**) or to communicate during an emergency (**Table 2-16**).

Table 2-15	Example of SBAR Used for a Consultation

The midwife at an office is caring for a woman who is at 33 weeks' gestation and was diagnosed earlier in the pregnancy with gestational diabetes. When reading the woman's blood glucose log, the midwife observes that more than 20% of her values are high. She calls the consulting maternal–fetal medicine physician and gives this consult using SBAR.

S: I want to consult with you about a woman with uncontrolled gestational diabetes.

B: Maria Gonzalez is a 24-year-old primigravida at 33 weeks by LMP consistent by 19-week ultrasound. Her 1-hour glucose tolerance test was 150 mg/dL and her 3-hour glucose tolerance test had two elevated values. She was sent to the diabetes education center and received diet and glucose monitoring education. Over the last 2 weeks, 20% of her values are out of range, with five fasting levels between 100 mg/dL and 110 mg/dL and five 2-hour postprandial levels higher than 150 mg/dL, the highest being 180 mg/dL. She had a reactive NST today, the fetus is size equal to dates, and her urinalysis was negative for glucose.

A: My concern is that her diabetes diet is inadequate to control glucose levels, and I believe she needs medication.

R: I would like to schedule her to see you for a consultation within the next few days.

Abbreviations: LMP, last menstrual period; NST, nonstress test; SBAR, Situation, Background, Assessment, Recommendation.

Table 2-16	Example of SBAR Used in an Emergency Situation

The midwife at a small community hospital is caring for a woman who is bleeding heavily immediately after giving birth. She has called for physician assistance from a provider in the next room. When the physician arrives, the midwife says:

S: This woman is having a postpartum hemorrhage.

B: Marta gave birth to her fifth child 15 minutes ago over an intact perineum. Her total EBL is 800 mL. She has received 40 IU of oxytocin (Pitocin), 0.2 mg of ergonovine (Methergine), and 800 mcg of misoprostol per rectum (Cytotec). The placenta appeared intact, and there are no clots in the lower uterine segment.

A: She has severe uterine atony, and I think I feel some placental tissue in the anterior portion of the fundus.

R: I need you to put on gloves and assist me.

Abbreviations: EBL, estimated blood loss; SBAR, Situation, Background, Assessment, Recommendation.

In closed-loop communication, the midwife directs the message to a particular team member, the team member repeats the order or request aloud, and the midwife confirms that the team member heard correctly. This communication approach is particularly important during times of stress, as it allows the entire team to hear the orders and correct any errors before the orders are executed. Closed-loop communication tools such as the *call-out* and the *check-back* can be used to communicate critical information to all members of the team, thereby allowing them to anticipate what will be needed next. Use of such instruments also requires that team members communicate what they intend to do and have done with the information.

When the care of a woman or a neonate needs to be transferred to another provider, especially for a higher level of care, a formal note should be written in the health chart and

shared with the new provider. The goal of this communication is to give the new provider all of the pertinent information needed to safely care for the woman and her family. In several of the chapters in this text, *critical elements* are listed for conditions that typically require a transfer of care. In some situations, a midwife may receive a transfer from another provider. In that case, the midwife who receives the transfer should communicate, either verbally or in writing, with the referring provider to acknowledge that the woman has been seen and provide a summary of her course of care. **Table 2-17** provides an overview of the content of a transfer or handoff note and the critical elements it should contain.

Table 2-17 Sample Critical Elements for a Transfer Note from Out-of-Hospital to Inpatient Care

Record time and destination, method of transportation, and who is accompanying the woman/newborn when care is transferred
Identify the woman/neonate and the transferring provider; as well as the receiving provider
The critical elements are customized to the individual and the situation but generally include:
1. Risk factors for the current situation/disorder requiring transfer
2. Signs and symptoms indicating need for transfer
3. Care provided before transfer: procedures and results; laboratory results or if pending; medications (dosage, route and time of last dose); response to treatment
4. Assessment/diagnosis of the situation
5. Summary of the rationale for the transfer Request for information about the treatment plan for follow-up

Communication skills such as SBAR, closed-loop communication, and the handoff are like any clinical skill: They must be adapted to individual settings and practiced until they become second nature.

The Researcher and Research User

Midwifery has a long tradition that includes learning by watchful waiting; sharing empirical knowledge via oral traditions; defining and protecting the normal, nonmedicalized birth process; and actively challenging "the evidence." These characteristics have served women well, especially when research has validated the midwifery approach. Examples where midwives have had a strong influence in the evolution of best practices include elimination of routine episiotomies,[70] redefinition of the Friedman labor curve,[71] promotion of early and prolonged breastfeeding for neonatal and maternal health,[72] delayed cord-clamping,[73] immediate skin-to-skin contact between mother and newborn,[74] water immersion during labor,[75] an alternative pain assessment tool for laboring women[76] and nonpharmacologic methods of pain control.[77]

A 2017 ACOG Committee Opinion, *Approaches to Limit Intervention During Labor and Birth*, has the potential to decrease what has come to be seen as common disagreements between some physicians and midwives regarding labor management.[78] Acknowledging that

"many common obstetric practices are of limited or uncertain benefit for low-risk women in spontaneous labor," this document makes 11 evidence-based recommendations intended to decrease unnecessary interventions and increase maternal and family satisfaction. These recommendations closely align with ACNM recommendations.[79]

Sackett et al. concisely defined evidence-based practice (EBP) in 2000 as the "integration of the best research evidence with clinical expertise and patient values."[80] Not all midwives need to actively conduct research, but all need to understand relevant research and implement evidence-based care. The call for systematic use of evidence in the field of caring for pregnant women is often credited to the 1989 publication of the two-volume book, *Effective Care in Pregnancy and Childbirth*.[81] In this ground-breaking treatise, the authors carefully evaluated existing research articles and identified those clinical practices supported by research as well as those practices that the evidence did not support.

Several databases that summarize the most recent evidence on a multitude of clinical topics are available to women's healthcare providers. One important evidence-based database is the Cochrane Library. Cochrane Reviews are systematic reviews of primary research in human health care and health policy and are widely recognized for the quality of the reviews.[82] Other sources of research that midwives often use include PubMed, the Up-to-Date Database, and DynaMed.

When assessing research data and results, it is important to remember that "not all evidence is created equal." Once all the research data have been gathered, the findings need to be compared and contrasted. Evidence then is evaluated to determine its strength.[83] Several ratings criteria have been developed that evaluate the strength and quality of research findings. One of the most commonly used rating scales is that used by the U.S. Preventive Task Force, which are listed in the *Health Promotion Across the Lifespan* chapter in this text.

Systematic reviews of the literature have documented that midwifery-led care (care in which the primary provider is the midwife) for essentially healthy women is equivalent to the care provided by physicians. For several outcome measures midwifery care has been found to be associated with better outcomes compared to physician care. A 2008 Cochrane meta-analysis reviewed 11 trials including 12,276 women, and found several statistically significant differences in outcomes for those women who received midwife-led care.[84] All of the studies included in this systematic review were randomized, controlled trials; in addition, the studies were not limited to one country. The findings noted that midwife-led care resulted in fewer prenatal hospitalizations, less use of regional analgesia, fewer episiotomies, and fewer instrument deliveries. In addition, women who received care in a system of midwife-led care were more likely to experience no intrapartum analgesia/anesthesia, a spontaneous vaginal birth, feeling in control during childbirth, attendance at birth by a known midwife, and initiation of breastfeeding. Finally, the newborns of women who had midwife-led care were more likely to have a shorter length of hospital stay. The authors concluded that "most women should be offered midwife-led models of care and women should be encouraged to ask for this option although caution should be exercised in applying this advice to women with substantial medical or obstetric complications."[84]

Similar results were highlighted in a 2011 systematic review that examined outcomes for

APRNs in the United States. For the purposes of this study, the authors defined certified nurse-midwives as a type of APRN, and CNM birth outcomes from 1990 to 2008 were examined separately from those of other groups of providers who were not CNMs.[85] This review summarized the results from all levels of studies, including observational studies, and studies were limited to the United States. A high level of evidence was found that certified nurse-midwives, when compared to physicians, had lower rates of cesarean section birth, episiotomy, operative birth, labor analgesia, and perineal lacerations, and equivalent rates of labor augmentation, low Apgar scores, and low-birth-weight infants. The systematic review also demonstrated a moderate level of evidence that care by nurse-midwives is associated with lower rates of epidural use and induction of labor, comparable or higher rates of vaginal births, comparable or lower rates of newborn intensive care unit admissions, and higher rates of breastfeeding among women who received care from these professionals.[85]

In 2016, the Cochrane Pregnancy and Childbirth Group published a review comparing midwife-led continuity models and other models of care,[86] updating the 2008 systematic review conducted by Hatem et al.[84] Fifteen trials involving 17,674 women were reviewed. In all trials, the pregnant women were randomly assigned to midwife-led continuity models of care or other models of care. In short, women who received the midwife-led continuity model of care were less likely to experience intervention and more likely to be satisfied with their care with at least comparable rates of adverse outcomes compared to women who received other models of care. The major findings from this updated Cochrane Review are summarized in **Table 2-18**.

Table 2-18 Systematic Review of Midwife-Led Continuity of Care, 2016

Outcome or Subgroup Title	Number of Studies	Number of Participants	Relative Risk (95% Confidence Interval)
Primary Outcomes: Significant Risk Reductions Found			
Regional anesthesia	14; high quality	17,674	RR = 0.85 (0.78–0.92)
Instrumental vaginal birth	13; high quality	17,501	RR = 0.90 (0.83–0.97)
Preterm birth: less than 37 weeks' gestation	8; high quality	13,238	RR = 0.76 (0.64–0.91)
Less all fetal loss before and after 24 weeks' gestation plus neonatal death	13; high quality	17,561	RR = 0.84 (0.71–0.99)
More Likely to Experience			
Spontaneous Vaginal Birth	12; high quality	16,687	RR = 1.05 (1.03–1.07)
No difference between groups for caesarean births and intact perineums			
Secondary Outcomes: Significant Risk Reductions Found			
Amniotomy	4	3253	RR = 0.80 (0.66–0.98)
Episiotomy	14	17,674	RR = 0.84 (0.77–0.92)
Fetal loss less than 24 weeks' gestation and neonatal death	11	15,645	RR = 0.81 (0.67–0.98)
More Likely to Experience			
No intrapartum analgesia or anesthesia	7	10,499	RR = 1.21 (1.06–1.37)
Longer length of labor	3	3328	RR = 0.50 (0.27–0.74)
Attended at birth by known midwife	7	6917	RR = 7.04 (4.48–11.08)

Based on Sandall J, Soltani H, Gates S, Shennan A, Devane D. Midwife-led continuity models versus other models of care for childbearing women. *Cochrane Database Syst Rev.* 2016;4:CD004667. doi:10.1002/14651858.CD004667.pub5.[86]

In addition to these large reviews, numerous other published research studies have focused on specific practices of midwives that may account for differences in maternal and neonatal outcomes. In 2012, ACNM updated a PowerPoint slide set titled "The Pearls of Midwifery," which translates the latest evidence in a manner that can be easily communicated to other providers, or to women and their families.[79]

Midwifery as a Worldwide Profession

While midwives maintain their commitment to provide individualized care responsive to the needs of the woman and newborn, they are also recognized as key players in the global community of healthcare professionals who significantly improve the lives of mothers and babies.[87] This expanded allegiance—from the individual, to the profession, to evidence-based practice, to women wherever they need care—is reflected throughout this text.

For many years, midwives in the United States and many countries around the world were undervalued by the policymakers who designed and funded healthcare systems. Most would agree that this low status, and the accompanying low pay, was a direct reflection of the value that policymakers assigned to women and women's health care. In a trajectory that closely follows the emancipation of women, midwives have found their collective voice, established professional standards for education and practice that compare favorably to their physician colleagues, and proved their value to the preservation of a healthy modern society.

While the rate of public acceptance and legal recognition of midwives as professional and autonomous healthcare providers has varied from state to state and from country to country, the global community of midwives is increasingly united around the need to earn and seek this recognition. The International Confederation of Midwives (ICM) is a global federation of midwifery associations that has worked for more than 100 years to support, represent, and strengthen professional associations of midwives throughout the world.

ICM initially provided a much-needed forum for midwives to learn from each other and to expand access to midwifery care. Nevertheless, it was not until 1972 that the confederation agreed to set standards for who should be able to use the "midwife" title. The original focus of this organization—to increase the number of midwives—reflected an understanding that too many women and babies were dying because they were giving birth unattended. Over time, it became clear that a lack of common standards for the preparation and practice of midwives could also put the lives of women at risk.

Many midwives feared that setting standards for midwifery education and practice would isolate traditional midwives and decrease access to care. Others emphasized the value for midwives of setting their own standards and using these standards as the justification to fund education for midwives, especially in countries with limited resources. In 1972, the ICM published its first *Definition of a Midwife*; it was most recently updated in 2011. In 2010, ICM published the *Essential Competencies for Basic Midwifery Practice*; it was most recently updated in 2013.[88]

Between 2008 and 2011, the International Confederation of Midwives accepted the difficult challenge of formally describing the three pillars of midwifery: education, regulation, and essential core competencies for midwifery practice. These core ICM documents have an important impact on midwifery in all nations.[55,88] For example, the Midwifery Education, Regulation, and Association work group,[5] a collaboration of the seven midwifery professional associations in the United States have worked together to adapt the ICM documents for midwifery in the United States. The 2015 publication of *Principles for Model U.S. Midwifery Legislation and Regulation* is one of the outcomes of this collaboration.[5,89] ICM now publishes multiple gap analysis tools and curriculum guidelines designed to assist midwifery

associations and policymakers in actualizing their support for professional midwifery practice. Since its launch in the early 1900s, the ICM has expanded to include, as of 2016, 132 midwifery associations from 113 countries, representing an estimated 500,000 midwives worldwide.[90]

The American College of Nurse-Midwives has been a member of ICM since 1956. The ACNM and many CNMs/CMs have made significant contributions to the ICM mission by strengthening midwifery associations globally, publishing training materials for midwives such as the *Life-Saving Skills Manual for Midwives*[91] and *Home-Based Skills Manual for Midwives*,[92] and advocating for reduction of needless maternal deaths.

Midwives frequently seek ways to share their expertise globally, and they are often sought as team members on international projects. **Appendix 2A** provides information to help prepare midwives for such projects—projects that are meaningful for women in other countries as well as for the midwives themselves.

Conclusion

In the twenty-first century, midwifery is an evolving profession with a strong, inspirational foundation; a mature infrastructure to promote policies that improve access to high-quality midwifery care; highly educated individuals who are defining best practice; and plenty of unfulfilled potential. Midwives have demonstrated their capacity to do the hard work of profession building, to critically evaluate traditional models of care, to challenge policies based on flawed research, and to pursue a more just healthcare delivery system. How the profession changes and grows will reflect who the new midwives are, what brings them to the profession, who educates them, and how much they are willing to give to individual women, to the profession of midwifery, and to the process of creating a world where all women receive the best care possible.

Resources

Articles
Birth Centers
Alliman J, Phillippi JC. Maternal outcomes in birth centers: an integrative review of the literature. *J Midwifery Women*
Stapleton SR, Osborne C, Illuzzi J. Outcomes of care in birth centers: demonstration of a durable model. *J Midwifer*
International Midwifery
Midwifery. *Lancet.* 2014. Available at: http://www.thelancet.com/series/midwifery. Accessed May 21, 2017.
Traditional Midwives and Traditional Birth Attendants
Byrne A, Morgan A. How the integration of traditional birth attendants with formal health systems can increase skilled 2011;115(2):127-134.
Sibley LM, Sipe TA, Barry D. Traditional birth attendant training for improving health behaviours and pregnancy outcor 2012;8:CD005460. doi:10.1002/14651858.CD005460.pub3.
Stanton C. Steps toward achieving skilled attendance at birth. *Bull WHO.* 2008;86(4):241.

Organization	Description	Webpage
American Association of Birth Centers (AABC)	A multidisciplinary membership organization dedicated to the birth center model of care.	http://www.birthcenters.org
American College of Nurse-Midwives (ACNM)	Professional organization for certified nurse-midwives and certified midwives in the United States. The Hallmarks of Midwifery can be found on this site.	http://www.midwife.org http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/00000000
American College of Obstetricians and Gynecologists (ACOG)	Founded in 1951, ACOG is the specialty's professional membership organization dedicated to the improvement of women's health. Interdisciplinary collaboration	https://www.acog.org http://www.acog.org/Resources-And-Publications/Task-Force-and-Work-Group-Repo Based-Care
American Nurses Association (ANA)	ANA advances the nursing profession and advocates on	http://nursingworld.org

	healthcare issues that affect nurses and the public.	
American Public Health Association (APHA)	A champion for the health of all people and all communities, AHA works to strengthen the profession and to speak out for public health issues and policies supported by science.	https://apha.org
Association of Women's Health, Obstetric and Neonatal Nurses (AWHONN)	AWHONN works to improve and promote the health of women and newborns and to strengthen the nursing profession.	http://www.awhonn.org
Childbirth Connection	This organization was founded in 1918 as the Maternity Center Association, is now a program in the National Partnership for Women and Families. It works to improve the quality and value of maternity care through consumer engagement and health system transformation. Childbirth Connection promotes safe, effective, and satisfying evidence-based maternity care	http://www.childbirthconnection.org

	and is a voice for the needs and interests of childbearing families.	
Coalition for Quality Maternal Care (CQMC)	In April 2011, nine national professional, consumer, and human rights organizations announced the formation of this coalition to champion the urgent need for national strategies to improve the quality and value of maternal and newborn health care in the United States.	http://www.midwife.org/Coalition-for-Quality-Maternity-Care
Home Birth Summit	In 2011, 2013, and 2014, the Home Birth Summits convened a multidisciplinary group of leaders, representing all stakeholder perspectives, to address their shared responsibility for care of women who plan home births in the United States.	http://www.homebirthsummit.org
International Confederation of Midwives (ICM)	This global federation of midwifery associations has worked for more than 100 years to support, represent, and strengthen professional associations of midwives	http://internationalmidwives.org

	throughout the world.	
Lamaze International	Established in 1960, and redefined in 2001, Lamaze dramatically changed from being a method for giving birth to a philosophy that provides the foundation and direction for women as they prepare to give birth. This organization's mission is to advance safe and healthy pregnancy, birth, and early parenting through evidence-based education and advocacy.	https://www.lamazeinternational.org
Midwifery Business Network (MBN)	A collaborative of CNMs and CMs who, for more than 30 years, have supported entrepreneur midwives. In 2015, the network was integrated into the Business Section of the American College of Nurse-Midwives' Division of Standards and Practice.	http://www.midwife.org/index.asp
National Association of Nurse Practitioners in Women's Health (NPWH)	NPWH works to ensure the provision of quality primary and specialty health care to women of all ages by women's health	https://www.npwh.org

	and women's health–focused nurse practitioners.	
National Partnership for Women and Families (NPWF)	Founded in 1971 as the Women's Legal Defense Fund, NPWF promotes fairness in the workplace, reproductive health and rights, access to quality, affordable health care, and policies that help women and men meet the dual demands of work and family.	http://www.nationalpartnership.org
National Women's Health Network (NWHN)	Feminist health activists who use policy analysis as a tool. Starting in 1940 with a protest about the risks of estrogen, NWHN has sought to bring the voice of people concerned about women's health to the decision makers who create and implement health policies.	https://nwhn.org https://www.womenshealthnetwork.com
Our Bodies Ourselves (OBOS)	Since its founding in 1969, OBOS has sought to advance health and human rights within a framework of values shaped by women's voices and a	http://www.ourbodiesourselves.org

	commitment to self-determination and equality.	
White Ribbon Alliance	International organization with a mission to catalyze and convene advocates who campaign to uphold the right of all women to be safe and healthy before, during, and after childbirth.	http://whiteribbonalliance.org

References

1. Ament LA. *Professional Issues in Midwifery*. Sudbury, MA: Jones and Bartlett; 2007.
2. Kennedy HP. A model of exemplary midwifery practice: results of a Delphi study. *J Midwifery Womens Health*. 2000;45(1):4-19.
3. American College of Nurse-Midwives. Comparison of certified nurse-midwives, certified midwives, certified professional midwives clarifying the distinctions among professional midwifery credentials in the U.S. Available at: www.midwife.org/Updated-CNM-CM-CPM-Comparison-Chart-October-2017. Accessed November 15, 2017.
4. National Association of Certified Professional Midwives. Who are CPMs? Available at: http://nacpm.org/about-cpms/who-are-cpms/. Accessed June 2, 2017.
5. U.S. Midwifery Education, Regulation, and Association. Available at: http://www.midwife.org/US-MERA. Accessed April 6, 2017.
6. Childbirth Connection. Available at: http://www.childbirthconnection.org. Accessed April 26, 2017.
7. Citizens for Midwifery. Welcome to Citizens for Midwifery! Available at: http://cfmidwifery.org/index.aspx. Accessed April 26, 2017.
8. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
9. Foster IR, Lasser J. *Professional Ethics in Midwifery Practice*. Sudbury, MA: Jones and Bartlett; 2011.
10. Thompson JB. A human rights framework for midwifery care. *J Midwifery Womens Health*. 2004;49(3):175-176.
11. Thompson JB, King TL. Resources for clinicians: a code of ethics for midwives. *J Midwifery Womens Health*. 2004;49(3):263-265.
12. ACNM code of ethics with explanatory statements. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000293/Code-of-Ethics-w-Explanatory-Statements-June-2015.pdf. Accessed June 3, 2017.
13. Midwives Alliance of North America. Statement of values and ethics. Available at: https://mana.org/resources/statement-of-values-and-ethics. Accessed April 16, 2017.
14. Beauchamp TL, Childress, JF. *Principles of Biomedical Ethics*. 5th ed. New York, NY: Oxford University Press; 2001.
15. Mighty HE, Fahey J. Clinical ethics in obstetrics and gynecology. In: Barbieri RL, Reece EA. *Obstetrics and Gynecology: The Essentials of Clinical Care*. Stuttgart, Germany: Thieme Publishers; 2010:517-526.
16. Hodges S. Abuse in hospital-based birth settings. *J Perinatal Educ*. 2009;18(4):8-11.
17. Beauchamp TL. Informed consent: its history, meaning, and present challenges. *Cambridge Q Healthcare Ethics*. 2011;20:515-523.
18. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 390: ethical decision making in obstetrics and gynecology. Available at: http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Ethics/Ethical-Decision-Making-in-Obstetrics-and-Gynecology. Accessed April 26, 2017.
19. Elwyn G, Coulter A, Laitner S, Walker E, Watson P, Thomson R. Implementing shared decision making in the NHS. *BMJ*. 2010;341:c5146.
20. Elwyn G, Frosch D, Thomson R, et al. Shared decision making: a model for clinical practice. *J Gen Intern Med*. 2012;27(10):1361-1367. doi:10.1007/s11606-012-2077-6.
21. American College of Nurse-Midwives. Position statement: shared decision-making in midwifery care. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000305/Shared-Decision-Making-in-Midwifery-Care-Dec-2016.pdf. Accessed April 27, 2017.
22. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 390: ethical decision making in obstetrics and gynecology. *Obstet Gynecol*. 2007;110:1479-1487.
23. Sokol DK. Ethics man: rethinking ward rounds. *BMJ*. 2009;338:571.
24. Yeo GSH, Lim ML. Maternal and fetal best interests in day-to-day obstetrics. *Ann Acad Med Singapore*. 2011;40:43-49.
25. American College of Nurse-Midwives. Descriptive data nurse-midwives—USA. *Bull Am Col Nurse-Mid*. 1963;8(1):30-37.
26. Fullerton J, Sipe TA, Hastings-Tolsma M, et al. The midwifery workforce: ACNM 2012 and AMCB 2013 core data. *J Midwifery Womens Health*. 2015;60(6):751-761.
27. Kennedy HP, Erickson-Owens D, Davis JA. Voices of diversity in midwifery: a qualitative research study. *J Midwifery Womens Health*. 2006;51(2):85-90.
28. Holmes LJ. *Into the Light of Day: Reflections on the History of Midwives of Color within the American College of Nurse-Midwives*. Washington, DC: LJH Consultancies; 2011.
29. Health Resources and Services Administration. The rationale for diversity in the health professions: a review of the evidence. 2006. Available at: http://docplayer.net/255577-The-rationale-for-diversity-in-the-health-professions-a-review-of-the-evidence.html. Accessed June 3, 2017.

30. Serbin, J, Donnelly, E. The impact of racism and midwifery's lack of racial diversity: a literature review. *J Midwifery Womens Health*. 2016;61(6):694-706.
31. Foster J, DeLibertis J, Nixon A. Forging our future as a diverse and inclusive midwifery profession. *J Midwifery Womens Health*. 2015;60(6):662-664.
32. American College of Nurse-Midwives. Shifting the frame: a report on diversity and inclusion in the American College of Nurse-Midwives. 2015. Available at: http://www.midwife.org/acnm/files/ccLibraryFiles/Filename/000000005329/Shifting-the-Frame-June-2015.pdf. Accessed June 3, 2017.
33. American College of Nurse-Midwives. Diversification and Inclusion Task Force. Available at: http://www.midwife.org/Diversification-and-Inclusion-Task-Force. Accessed June 3, 2017.
34. Supporting healthy and normal physiologic childbirth: a consensus statement by ACNM, MANA and NACPM. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000272/Physiological%20Birth%%20FINAL%20May%2018%202012%20FINAL.pdf. Accessed January 19, 2017.
35. Schuiling KD, Slager J. Scope of practice: freedom within limits. *J Midwifery Womens Health*. 2000;45(6):465-471.
36. Osborne K. Regulation of prescriptive authority for certified nurse-midwives and certified midwives: 2015 national overview. *J Midwifery Womens Health*. 2015;60:519-533.
37. American College of Nurse-Midwives. Midwifery strategies for liability risk reduction. Available at: http://www.midwife.org/index.asp?bid=59&cat=9&button=Search. Accessed February 2, 2017.
38. American College of Nurse-Midwives. Professional liability resource packet. Available at: http://www.midwife.org/index.asp?bid=59&cat=5&button=Search&rec=134. Accessed June 3, 2017.
39. Buppert C. *Nurse Practitioner's Business Practice and Legal Guide*. 6th ed. Burlington, MA: Jones & Bartlett Learning; 2018.
40. Slager J. *An Administrative Manual for Midwifery Practices*. 4th ed. Washington, DC: American College of Nurse-Midwives; 2016.
41. American College of Nurse-Midwives. The ACNM benchmarking project. Available at: http://www.midwife.org/Benchmarking. Accessed June 3, 2017.
42. Midwives Alliance of North American. MANA Statistics Project. Available at: https://www.manastats.org/help_public_about. Accessed June 3, 2017.
43. American Association of Birth Centers. AABC Perinatal Data Registry (PDR). Available at: http://www.birthcenters.org/?PDR. Accessed June 3, 2017.
44. Demiris G. Consumer health informatics: past, present, and future of a rapidly evolving domain. *Yearb Med Inform*. May 20, 2016;suppl 1:S42-S47.
45. Arcia A. Facebook advertisements for inexpensive participant recruitment among women in early pregnancy. *Health Educ Behav*. 2014;41(3):237-241.
46. American College of Nurse-Midwives. Healthy Birth Initiative. Available at: http://www.midwife.org/ACNM-Healthy-Birth-Initiative. Accessed March 28, 2017.
47. Williams DR. We need to say in unison: we are midwives and we do policy! [Editorial]. *J Midwifery Womens Health*. 2008;53(2):101-102.
48. Baldwin DC Jr. Some historical notes on interdisciplinary and interprofessional education and practice in health care in the USA. *J Interprof Care*.1996;21(suppl 1):23-37.
49. Avery M, Montgomery O, Sbrandl-Salutz E. Essential components of successful collaborative maternity care models. *Obstet Gynecol North Am*. 2012;39:423-434.
50. Waldman R, Kennedy HP, Kendig S. Collaboration in maternity care: possibilities and challenges. *Obstet Gynecol Clin North Am*. 2012;39:435-444.
51. Jennings J, Nielsen P, Buck ML, et al. Executive summary: collaboration in practice: implementing team-based care: report of the American College of Obstetricians and Gynecologists' Task Force on Collaborative Practice. *Obstet Gynecol*. 2016;127(3):612-617.
52. Kohn LT, Corrigan JM, Donaldson MS. *To Err Is Human: Building a Safer Health System*. Washington, DC: National Academy Press; 2000.
53. The Joint Commission. *Sentinel Event Alert: Preventing Infant Death and Injury During Delivery* (Issue No. 30). Oakbrook Terrace, IL: The Joint Commission; 2004. Available at: http://www.jointcommission.org/assets/1/18/SEA_30.PDF. Accessed April 27, 2017.
54. Jackson DJ, Lang JM, Swartz WH, et al. Outcomes, safety, and resource utilization in a collaborative care birth center program compared with traditional physician-based perinatal care. *Am J Public Health*. 2003;93:999-1006.
55. International Confederation of Midwives. Global standards for midwifery regulation 2010. 2011. Available at: http://internationalmidwives.org/what-we-do/global-standards-competencies-and-tools.html. Accessed June 3, 2017.
56. American College of Nurse-Midwives. Position statement: independent midwifery practice. 2012. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000073/Independent-

Midwifery-Practice-Feb-2012.pdf. Accessed December 21, 2017.
57. American College of Nurse-Midwives. Position statement: collaborative management in midwifery practice for medical, gynecologic, and obstetric conditions. 2014. Available at: http://www.midwife.org/ACNM/files/CNMLibraryData/UPLOADFILENAME/000000000058/Collaborative-Mgmt-in-Midwifery-Practice-Sept-2014.pdf. Accessed December 21, 2017.
58. American College of Nurse-Midwives. Joint statement of practice relations between obstetrician-gynecologists and certified nurse-midwives/certified midwives. Available at: https://www.acog.org/-/media/Statements-of-Policy/Public/sop1102.pdf?dmc=1&ts=20170923T2058080347. Accessed June 3, 2017.
59. Midwives Alliance of North America. Midwives model of care. Available at: https://mana.org/about-midwives/midwifery-model. Accessed June 3, 2017.
60. Interprofessional Education Collaborative Expert Panel. *Core Competencies for Interprofessional Collaborative Practice: Report of an Expert Panel*. Washington, DC: Interprofessional Education Collaborative; 2011.
61. Ivey S. A model for teaching about interdisciplinary practice. *J Allied Health*. 1988;17:189-195.
62. King TL, Laros RK, Parer JT. Interprofessional collaborative practice in obstetrics and midwifery. *Obstet Gynecol North Am*. 2012;39:411-422.
63. King H, Battles J, Baker DP, et al. TeamSTEPPS®2.0: team strategies and tools to enhance performance and patient safety. Agency for Healthcare Research and Quality. Available at: https://www.ahrq.gov/teamstepps/index.html. Accessed June 3, 2017.
64. Merien AER, van der Ven J, Mol BW, Houterman S, Oei SG. Multidisciplinary team training in a simulation setting for acute obstetric emergencies: a systematic review. *Obstet Gynecol*. 2010;115(5):1021-1031.
65. Crofts JF, Ellis D, Draycott TJ, Winter C, Hunt LP, Akande VA. Change in knowledge of midwives and obstetricians following obstetric emergency training: a randomized controlled trial of local hospital, simulation center and teamwork training. *BJOG*. 2007;114:1534-1541.
66. Robertson B, Schumacher L, Gosman G, Kanfer R, Kelley M, DeVita M. Simulation-based crisis team training for multidisciplinary obstetric providers. *Simulation in Healthcare*. 2009;4(2):77-83.
67. Baker DP, Gustafson S, Beaubien J, Salas E, Barach P. *Medical Teamwork and Patient Safety: The Evidence-Based Relation Literature Review*. Rockville, MD: Agency for Healthcare Research and Quality; 2005. Available at: https://archive.ahrq.gov/research/findings/final-reports/medteam/medteamwork.pdf. Accessed April 27, 2017.
68. Miller LA. Patient safety and teamwork in perinatal care: resources for clinicians. *J Perinat Neonat Nurs*. 2005;19(1):46-51.
69. Vardaman JM, Cornell P, Gondo MB, Amis JM, Townsend-Gervis M, Thetford C. Beyond communication: the role of standardized protocols in a changing health care environment. *Health Care Manage Rev*. 2012;37(1):88-97.
70. Jiang H, Qian X, Carroll G, Garner P. Selective versus routine episiotomy for vaginal birth. *Cochrane Database Syst Rev*. **2017.** Available at: http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD000081.pub3/full. Accessed September 23, 2017.
71. Albers LL, Schiff M, Gorwoda JG. The length of active labor in normal pregnancies. *Obstet Gynecol*. 1996;87:355-359.
72. Horta BL, Bahl R, Martines JC, Victoria CG. *Evidence on the Long-Term Effects of Breastfeeding: Systemic Review and Meta-analyses*. Geneva, Switzerland: World Health Organization; 2013. Available at: http://www.who.int/maternal_child_adolescent/documents/breastfeeding_long_term_effects/en/. Accessed May 21, 2017.
73. Mercer JS, Vohr BR, McGrath MM, Padbury JF, Wallach M, Oh W. Delayed cord clamping in very preterm infants reduces the incidence of intraventricular hemorrhage and late-onset sepsis: a randomized, controlled trial. *Pediatrics*. 2006;117(4):1235-1242.
74. Moore ER, Bergman N, Anderson, GC, Medley, N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;11. doi:10.1002/14651858.CD003519.pub4.
75. Cluett ER, Burns E. Immersion in water in labour and birth. *Cochrane Database Syst Rev*. 2009;2:CD000111. doi:10.1002/14651858.CD000111.pub3.
76. Roberts, L, Gulliver B, Fisher J, Clyes KG. The coping with labor algorithm: an alternate pain assessment tool for the laboring woman. *J Midwifery Womens Health*. 2010;55:107-116.
77. Simkin PP, O'Hara M. Nonpharmacologic relief of pain during labor: systematic reviews of five methods. *Am J Obstet Gynecol*. 2002;186(suppl 5):S131-S159.
78. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 687: approaches to limit intervention during labor and birth. Available at: http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/Approaches-to-Limit-Intervention-During-Labor-and-Birth. Accessed June 3, 2017.
79. American College of Nurse-Midwives. Evidence-based practice: pearls of midwifery: a presentation by the American College of Nurse-Midwives, Washington, DC, 2010. Available at: http://www.midwife.org/Pearls. Accessed March 28, 2017.

80. Sackett DL, Straus SE, Richardson WS, Rosenberg WM, Haynes RB. *Evidence-Based Medicine: How to Practice and Teach EBM*. 2nd ed. Edinburgh, UK: Churchill Livingstone; 2000.
81. Chalmer I, Enkin M, Marc JM, Keirse C. *Effective Care in Pregnancy and Childbirth*. Oxford, UK: Oxford University Press; 1989.
82. *Cochrane Database of Systematic Reviews*. Available at: http://www.cochranelibrary.com. Accessed May 21, 2017.
83. U.S. Preventive Services Task Force. Grade definitions. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Name/grade-definitions. Accessed June 5, 2017.
84. Hatem M, Sandall J, Devane D, Soltani H, Gates S. Midwife-led versus other models of care for childbearing women. *Cochrane Database Syst Rev*. 2008;4:CD004667. doi:10.1002/14651858.CD004667.pub2.
85. Newhouse RP, Stanik-Hutt J, White KM, et al. Advanced practice nurse outcomes 1990–2008: a systematic review. *Nurs Econ*. 2011;29(5):230-250.
86. Sandall J, Soltani H, Gates S, Shennan A, Devane D. Midwife-led continuity models versus other models of care for childbearing women. *Cochrane Database Syst Rev*. 2016;4:CD004667. doi:10.1002/14651858.CO004667.pub5.
87. Midwifery. *Lancet*. 2014. Available at: http://www.thelancet.com/series/midwifery. Accessed May 21, 2017.
88. International Confederation of Midwives. Essential competencies for basic midwifery practice. 2010. Updated 2013. Available at: https://internationalmidwives.org/assets/uploads/documents/CoreDocuments/ICM%20Essential%20Competencies%20for% Accessed December 18, 2017.
89. American College of Nurse-Midwives. Principles for Model U.S. Midwifery Legislation and Regulation. Available at: http://www.midwife.org/ACNM/files/ccLibraryFiles/Filename/000000005972/US-MERALegislativeStatement2015.pdf. Accessed December 18, 2017.
90. International Confederation of Midwives. Who we are. Available at: http://internationalmidwives.org/who-we-are/. Accessed June 3, 2017.
91. Marshall MA, Buffington ST, Beck DR, Clark PA. Life-Savings Skills: Manual for Midwives. 4th ed. Silver Spring, MD: American College of Nurse-Midwives; 2008. Available at: http://www.midwife.org/ACNM-Publications. Accessed December 18, 2017.
92. Buffington ST, Sibley LM, Beck R, Armbruster DA. Home Based Life Saving Skills. 2nd ed. Silver Spring, MD: American College of Nurse-Midwives; 2010. Available at: http://www.midwife.org/ACNM-Publications. Accessed December 18, 2017.

APPENDIX

2A

Worksheet for Engaging in Global Midwifery

JOYCE E. THOMPSON

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This appendix is designed for midwifery students as well as midwives who are interested in sharing their expertise in geographic settings outside of their own state/country. The following key steps are intended to help a midwife decide whether international work is personally appropriate and if so, how to actualize that desire. This process takes times and experience. Enjoy the journey!

The first two steps involve personal reflection. The next seven steps involve pragmatics of engaging in global work as a midwife.

Step 1: Reflect on Your Beliefs, Values, and Characteristics

Take time to answer these questions: They are designed to help you learn about yourself and how you view working in cultures other than your own and with other team members. Be honest with yourself. This reflection can help you decide if international work is for you.

A. Values

What are the five most important values that guide my life, my actions, and my decisions?

Why are these values important to me?

Which basic human rights do I believe should be accorded to women and young girls? Are these consistent with my important values?

What do I value about the profession of midwifery and professional midwives?

Upon reflection, are my values congruent with international midwifery work? Do I need to rethink my long-term plans?

B. Working with Others

Which of my personality traits can contribute to positive communication with diverse individuals/cultures or detract from positive communication?

What is my usual response, both verbal and nonverbal, when I do not understand what is being said (perhaps in situations when another language is used or within my own language, such as use of slang words)?

What is my usual response, both verbal and nonverbal, when others do not understand what I am saying?

How well am I able to recognize information shared with me in confidence, and then maintain that confidence? (How trustworthy am I?)

How effective am I in gaining the trust of others in new situations?

Which experience to date have I had in living or working with those who speak a language different from my own?

Upon reflection, which traits or responses above should I strengthen and which should I change or improve?

C. Working as a Team Member

What are my dominant personality traits (e.g., like to be in charge, passive, outgoing)? How do these traits fit with a team? What is the role I usually take in a team situation (e.g., leader, member, quiet one)?

What is my usual response when I feel lacking in knowledge or experience, or am intimidated by others? Is that a useful response? If not, how can I change it?

How do I react when I am faced with a situation requiring a different approach from that I had planned to do?

What is my usual response when I am working with someone who knows more than I do?

Does it make a difference if team members are younger or older, or a different gender than mine?

How well do I work toward compromise? (Examples are important.)

What is my experience in living with decisions that differ from the ones I would have chosen in the situation?

How important is it that others follow my recommen-dations/advice?

How well do I support others in doing “for themselves” rather than doing it for them?

How do I respond to individual team members who do not follow through with agreed activities/responsibilities (do not carry their share of the work)?

Upon reflection, which traits or responses above should I strengthen and which should I change or improve?

D. How Do I Respond to Stress?

What is my usual response to being in a situation of uncertainty (e.g., new geographic place, new people)?

How do I normally deal with stress in my day-to-day living; as a midwife; when I am jet-lagged, tired, or overwhelmed?

Upon reflection, which traits or responses above should I strengthen and which should I change or improve?

E. Can I Live in a World That Is Very Different Than My Home?

Do I have any special dietary needs or healthcare needs, and have I thought about how they could be met in a variety of cultures? Would they limit my ability to be an international consultant? Explain.

Which experiences can I identify that help me understand the variety of "personal space" needs of individuals, including myself?

Have I had any experience living for longer than a few days without running water, indoor plumbing, or electricity? How did I cope?

What is the longest time I have been away from family/friends and have I ever been "homesick" or felt "lonely" when away from home?

What experience have I had in working with those who hold a different worldview than my own?

Upon reflection, which traits or responses above should I strengthen and which should I change or improve?

Step 2: Reflect on What You Have to Offer

Assuming you want to work internationally, which special talents can you provide? You want to be a help with global work, not just an observer. Sometimes you may need to sell yourself to a nongovernmental organization (NGO) or grant funder.

What are my particular areas of expertise in maternal and newborn health, family planning, and midwifery?

In which languages am I fluent?

What can I do (e.g., activities, reading, seeking support from experts) to improve my consultant competencies? (Be specific.)

What time frame will I need to complete the above activities?

Should I be a "consultant" or a "team member" of a larger group?

Step 3: Getting Organized and Ready

Once you have decided you understand and want to do global work, organization is essential. You do not simply show up in another country and plan to work.

Gain midwifery practice expertise, comfort, and confidence before you consider global work.

Learn about global midwifery initiatives, such as the role and work of the International Confederation of Midwives (ICM) and the World Health Organization (WHO).

Obtain licensure as a professional midwife in the area where the work will occur.

Consider joining a team and experiencing global work *before* even thinking you can be a formal "consultant."

Develop other areas of midwifery expertise (e.g., education, regulation, association leadership).

Step 4: Review the Proposed Project or Trip Before Accepting

When you do join a team or become a consultant, know what the position entails. Do not blindly attempt to work globally. A good project has clear, measurable, achievable, and sustainable objectives.

Who/which agency is funding the proposal and what is its track record in midwifery development and/or support?

What is the purpose of the proposal?

What are the expected outcomes and time frame? Who is responsible for each expected outcome?

Learn the language of the project (e.g., expected outcomes often are called terms of reference [TOR]).

Are the outcomes achievable within the time allowed?

Where and when will the project take place, and which language is needed?

What are the financial details, and are they acceptable for the products expected? Do I have the resources needed?

Who is responsible for logistics, including travel arrangements and visas if needed?

What are the plans for sustainability, if appropriate?

Is this a duplicate activity with other NGOs? If so, is it needed? Should I participate?

Step 5: Decide Whether Your Qualifications and Expertise Match What Is Expected

This step provides another time for you to be honest with yourself. Where do you fit within this project? Can you be a useful contributor? If you are a consultant, do you have the skills and knowledge to be successful?

What would be my role: consultant? team member? other?

Do my competencies match the needed expertise for my role?

Do I meet the agency's requirements for being a consultant or team member?

Do I have knowledge of the host country's midwifery situation?

Am I available in the time frame for consultation?

Do I have the language and communication skills expected during the consultation?

If not, how can I plan for the future?

Step 6: Planning for the Consultation Prior to Arriving in

Country

Before you leave the United States, learn about where you are going. The more you know in advance, the faster you can become a global worker or consultant in that country. You do not want to waste time in the country learning information that you already could have known.

Research all available information regarding the country and the situation for midwifery there.
Talk with other midwives who have been in the country.
Review legal regulations relating to the work you will perform.
Identify who is in charge of health care in the country (e.g., health minister).
Learn how midwives' work is integrated with the roles of other healthcare providers.
Prepare a detailed plan of work to accomplish the project objectives.
Communicate with designated contacts in the country and in the agency.
Partner with the host country midwife/association, as appropriate.
Identify and arrange for meetings with key stakeholders in advance.
Prepare materials needed for consultation (e.g., agenda for workshops, handouts, business cards).
Pack needed clothing for the climate and culture; bring a computer with appropriate electrical connections.
Confirm the travel arrangements.

Step 7: Carry Out the Project According to the Terms of Reference

Although you always should expect that some things might occur that are not planned, you also should have a clear idea of how you will proceed to meet the project objectives. This is true whether you are a member of a team or the consultant in charge.

Receive a briefing upon arrival from the country contact person.
Confirm prearranged meetings with key stakeholders and meet with them.
Carry out the detailed plan of work, adjusting it as needed to meet the objectives.
Take notes daily and keep a list of all persons contacted and reasons for any changes.
Prepare a draft report of the consultation.
Report draft findings to designated individuals prior to leaving the country, if expected.

Step 8: Prepare Final Reports

A final report is an essential component of international work. This report is facilitated if the previous steps are followed and notes are taken contemporaneously.

The final report should follow the template of the funding agency, if available. Some funders require that the final report begin with a one-page executive summary.
The report should address each expected outcome and the extent to which it was met.

The report often includes persons and places visited, along with dates, workshop outcomes, or lectures presented.
All reports need to be strategic, with an emphasis on the extent to which the expected outcomes were met, lessons learned, and recommendations for follow-up as needed.
The funder may require a separate "trip report" for travel and per diem reimbursement.
Submit an invoice, usually with the final report to the funder/agency.

Step 9: Reflect on Lessons Learned

Personal reflection after every global experience enhances your future plans. Again, be honest with yourself and learn from the experience.
What did I learn from the experience that will help me in future consultations?
Which mistakes did I make that I prefer not to repeat, and what is needed to overcome these?
Which strengths/special expertise did I demonstrate, and how will these add to future efforts in international midwifery support/consultation?
How do I plan to share my international midwifery experience with others?
If I were a team member, will I be an international consultant next? How do I prepare?

Resources

Organization	Description	Webpage
International Confederation of Midwives	An international organization dedicated to strengthening midwifery globally.	http://internationalmidwives.org
U.S. Central Intelligence Agency, *The World Factbook*	Information on the history, people, government, economy, geography, communications, transportation, military, and transnational issues for 267 world entities.	https://www.cia.gov/library/publications/the-world-factbook/
World Health Organization (WHO)	The WHO's website has webpages on specific countries, diseases, and other information for global work.	http://www.who.int/en/

Note: These are simply a few of the resources available. Additional information may be obtained when specific countries or organizations are identified.

CHAPTER

3

Midwifery: Clients, Context, and Care

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"A person's ability to choose wisely is harmed when their provider does not distinguish between personal beliefs and professional actions."

—Jan M. Kriebs

"If you have come to help me, you are wasting your time. But if you have come because your liberation is bound up with mine, then let us walk together..."

—Lila Watson, Australian Aboriginal woman, in response to mission workers

Introduction

An individual's health is influenced by five factors: genetics (30%), behavioral patterns (40%), social circumstances (15%), health care (10%), and environmental exposures (5%).[1,2] Behavioral patterns and environmental exposures are inexorably linked to social circumstances. It is now well known that social determinants of health (SDOH) have a significant impact on health, and that SDOH have the largest impact on health inequities.[1-5] Yet, worldwide, multiple social, political economic, and environmental inequities continue to contribute to significant health disparities, including preventable morbidity and mortality.[3,4]

Healthcare services can improve the health of individuals only when certain conditions are met. First, the individual must have access to healthcare services. Second, the care offered must be of good quality and based on evidence. Healthcare services are likely to be ineffective if the nonbiologic factors that affect access to and quality of care are not addressed. This chapter introduces key concepts that provide the foundation for understanding health disparities, including root causes and the roles that midwives play in mitigating health disparities. Midwifery is inherently a public health–oriented profession, and midwives have a fiduciary responsibility to seek ongoing education about these topics and application of this body of knowledge in each and every healthcare encounter.

The topics presented in this chapter include some exemplars of well-known disparities in health care. Although this chapter addresses just a few of the SDOH, this content can serve as a platform—a foundation—to help midwives identify which learning is needed to provide quality healthcare services to persons from all cultures and populations.

The first section introduces the concepts of power, privilege, and bias that affect every healthcare interaction. The second section explores the concepts of social structure and power that create SDOH inequities. Subsequent sections provide an overview of healthcare disparities and review several populations in the United States whose health is most impacted by inequity. The final sections describe the techniques used by individual midwives and the midwifery profession as a whole to address health disparities. Finally, population-based health statistics are internationally recognized as indicators of a nation's health and widely used to identify and track trends in the health status of populations. An understanding of reproductive health statistics is essential for all midwives, as these data frequently guide practice. **Appendix 3A** reviews definitions of key reproductive health statistics and explains how they are used and interpreted.

One more important caveat must be discussed before delving into the main chapter content. Language is an evolution and journey of its own, and the language used in this chapter and text is deliberately inconsistent. The term "persons of color" is used in this chapter as an umbrella term that refers to all persons who are not identified by society as white. It can be helpful to consider non-white persons together when looking at the effects of racism. However, important aspects of a person's racial, ethnic, and cultural identity can be muted when many different cultures are grouped together under one umbrella, and this is a cautionary point the reader is asked to bear in mind. Additionally, both the terms "African American" and "black" are intentionally used throughout the text to recognize that labeling persons within this social construct is challenging and imperfect. This chapter also aims to use gender-neutral language.

Due to the great diversity of gender identities, actively evolving norms about how to discuss gender on a population level, and the frequent lack of data that disaggregate the multitude of gender identities, the terms “woman” and “women” are used throughout this textbook without clarifying qualifiers. Language that refers to gender is not perfect, of course, so the reader is asked to consider the text with a critical eye and an awareness that while humans must be referred to with a word or phrase, those labels are never a comprehensive or totally accurate definition for any individual.

Social Structure: Power, Privilege, and Bias

"Society" is one of the most influential contexts within which individuals and communities exist. "Society"—that is, the organized pattern of relationships and interactions within a population—incorporates deeply embedded categories that affect each of us. These socially constructed categories and classifications reinforce hierarchical relationships between identities, such as gender, race, ethnicity, sexual orientation, socioeconomic status, religion, nationality, and immigration status. The existence of a social hierarchy privileges some identities as having implied and/or explicit power in relationship to oppressed identities.

Power is the ability to direct or influence the behavior of others, oneself, or a course of events; *privilege* exists when power unfairly advantages persons in some social categories versus others. Persons who identify with the socially dominant categories in **Table 3-1** have more privileges than do persons in the socially oppressed categories; similarly, persons in socially oppressed categories can be discriminated against or treated differently based solely on their "minority status."

Table 3-1 **Selected Categories That Are Determined by the Social Hierarchy**[a]

Category	Socially Dominant	Socially Oppressed
Gender	Cisgender male	Cisgender female, transgender, nonbinary and gender-nonconforming individuals
Race	White	People of color
Socioeconomic status	Middle and upper class	Poor, working class
Nation	Global North (including the United States)	Global South
Ethnicity	European	All other ethnicities
Sexual orientation	Heterosexual	Lesbian, gay, bisexual, queer, asexual
Religion	Christian	All other religions
Ability/disability	Able-bodies	Persons with disabilities (e.g., physical, mental, learning)
Age	Youth	Elderly people
Immigration status	Citizens	Undocumented persons, individuals with insecure documentation status, refugees, migrant workers, asylum seekers
Body size	Persons with BMI in the range 19–25 mm/kg^2	People with BMI < 19 mm/kg^2 or > 25 mm/kg^2
Language	English	All other languages

Abbreviation: BMI, body mass index.

[a] This table presents some examples of well-known social hierarchical identities that affect individuals. It is not intended to be a definitive guide to all forms of hierarchy.

An individual's social location is composed of multiple social identities and the interactions between them (e.g., race, ethnicity, gender, class, sexual orientation, age, disability/ability, migration status, religion). The individual experience of this multilayered existence reflects the

intersection of privilege and oppression at the social-structural level. Collectively, this framework is known as *intersectionality*.[6,7]

Societal privileges and discriminations grant variable and inequitable access to social, political, and economic resources, such as wages, high-quality education, safe housing and communities, and comprehensive health care. Structural, institutional, and interpersonal discrimination, in turn, contribute to health inequities. Health and health care are affected by power and privilege in society, such that individuals who experience socially dominant identities tend to have better health outcomes.

Healthcare Provider Bias

One of the ways that social hierarchy influences midwifery care is through healthcare provider bias. The social hierarchy shapes our unconscious and conscious beliefs about dominant and oppressed groups. These beliefs are referred to as *bias*, which is favor toward or prejudice against a certain person, group, or thing, typically without merit.

Bias reinforces social hierarchies, and it can result in unequal access to healthcare services and unequal rendering of quality care. This divergence often happens because there is a significant disparity between healthcare providers' *beliefs* and *actions* when interacting with individuals from social identities different than their own. Providers usually value fairness and justice, yet they may exhibit biases to the same extent as the general population and not offer equal care to all people.[8-10] Therefore, the ways in which healthcare providers perpetrate health inequities requires an examination of bias, and specifically implicit bias.

Implicit Bias in Healthcare Providers

Implicit bias is the process by which unconscious attitudes create preferences and assumptions, thereby shaping one's interactions with others and influencing behavior "without the conscious intention of the agent."[8] A provider may have few explicit biases but a high level of implicit biases. This concept explains the contradiction between healthcare providers' beliefs and actions. In essence, implicit bias results in an unintentional contradiction between the conscious desire to provide equal evidence-based care and the actual clinical decision making that occurs.[11] Multiple studies and systematic reviews have found that *implicit racial bias* adversely affects provider–client interactions, treatment decisions, treatment adherence, and healthcare outcomes.[12-14] Groups more likely to receive disparate care secondary to implicit bias are those identified in Table 3-1 as socially oppressed.

Implicit bias is difficult to address because it is unconscious. Factors such as provider stress and fatigue may increase the likelihood of the provider engaging in biased behaviors. A high cognitive load activates implicit biases in an unconscious attempt to categorize data quickly. In settings where midwives are expected to see a high volume of individuals in very short time slots, institutional approaches are needed to reduce cognitive demands, as a first step toward minimizing implicit biases.[8]

Reducing the interpersonal discrimination that results from implicit bias requires both ongoing individual examination and conscious de-biasing.[15] Recognizing the existence of one's

implicit biases is a useful starting point, but ongoing self-education about the nature of bias and triggers that evoke the biases is key to minimizing the adverse effects of implicit bias.[15] Online resources that help midwives identify implicit bias are listed in the Resources section at the end of this chapter.

Social Determinants of Health and Health Disparities

Over the last two decades, public health, medical, and nursing researchers have made significant advances in elucidating the effects of historical, social, political, and socioeconomic factors on individual and population health. As noted earlier, *social determinants of health* is the term widely used to describe these factors. SDOH comprise the wide range of factors that impact health but that have not historically been under the purview of clinical care (**Figure 3-1**).[16,17] The Centers for Disease Control and Prevention (CDC) defines SDOH as "conditions in the environments in which people are born, live, learn, work, play, worship, and age."[4] These factors can either promote good health or contribute to poor health. Research about SDOH has demonstrated that unequal distribution of power, money, and resources underlies the differences that contribute to disparities in health outcomes.[3] Because these forces are social, political, cultural, and economic in nature, clinical care alone cannot address the causes of these disparities. Attention to equity and justice in policy, structure, and culture change are required to address and eliminate these inequities.

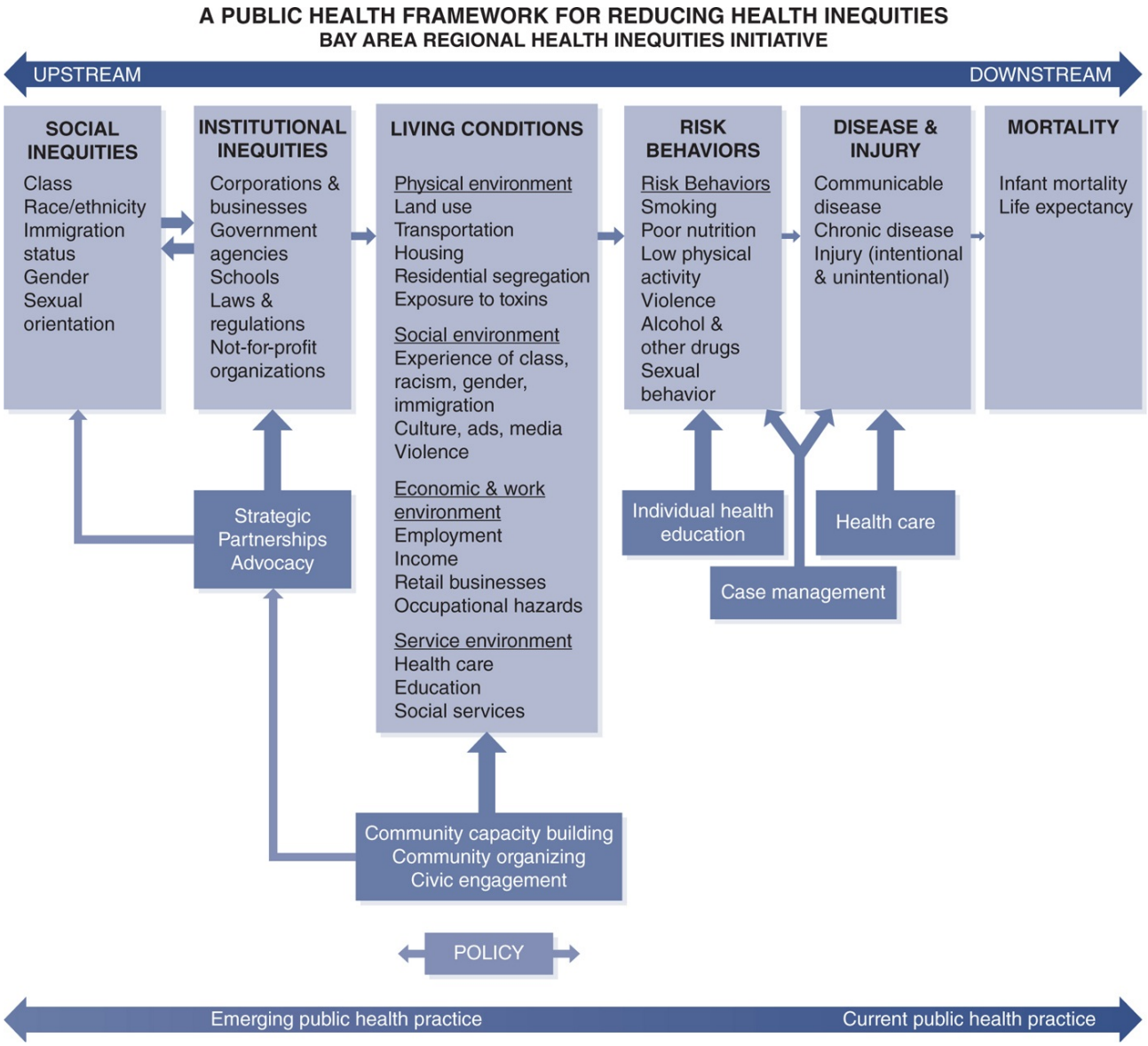


Figure 3-1 Public health framework for reducing health inequities.

Reproduced with permission from Bay Area Regional Health Inequities Initiative. Available at: http://barhii.org/framework/. Accessed January 16, 2018.[16]

Midwives have a long history of recognizing and addressing social determinants of health. Long before the term was coined, midwives worked as general healers within their communities, with their services encompassing a public health and preventive health perspective. For example, grand midwives in the American South, such as Onnie Lee Logan, brought food and social support in addition to clinical care for their clients, who were living in poverty.[17] Frontier midwives, such as Mary Breckinridge, encouraged the building of sanitary toilets and chlorinating infected wells to improve the health of families.[18] Sanba midwives in the American West, such as Toku Tosiko, offered culturally and linguistically competent care to the Japanese immigrant community, serving as cultural brokers as well as healthcare providers.[19]

The commitment to recognize and address social determinants of health continues today, as a

foundational precept of midwifery practice. Contemporary examples include midwives who are involved in maternity care models designed to address race-based health disparities,[20] environmental research and activism addressing high levels of environmental toxins found in breast milk in Mohawk communities,[21] and new models of care such as CenteringPregnancy in which women and families establish social support networks that support optimal health outcomes as they share the journey of pregnancy and parenting.[22,23]

Braveman describes four common mechanisms of action by which SDOH affects health outcomes:[5]

- *Direct, rapid-acting exposures* are specific insults that cause direct, immediate mental and/or physical health consequences for the individual. For example, children raised in substandard housing with chronic exposure to lead in the home or water source can develop long-term cognitive disabilities.
- *Indirect, rapid-acting exposures* are exposures to insults that are immediate but indirectly contribute to adverse mental and/or physical health. For example, the availability of alcohol in a community increases the likelihood of alcohol-related trauma.
- *Indirect impact on health behaviors* comprises exposures to insults that indirectly impact current or future health behaviors. For example, "food deserts"—that is, areas with minimal access to fresh produce and high concentrations of fast-food options—are associated with poor nutrition that, decades later, may result in chronic disease.[24]
- *Complex and long causal pathways* are chronic exposures to social and environmental stressors, including discrimination, that lead to biological "wear and tear." These long-duration causal pathways may take place over a lifetime and/or persist from generation to generation. For example, Geronimus's "*weathering*" theory describes how repeated biological adaptations to chronic stress create physiologic burden (allostatic load) and lead to premature aging and morbidity, with the highest rates found among black women.[25]

Stress, Allostasis, and Epigenetic Mechanisms

The biologic mechanisms by which chronic stress mediates the relationship between SDOH and adverse health outcomes have been the subject of a great deal of research at both the individual and population levels. *Allostasis* refers to the body's dynamic responses to stress that work to maintain homeostasis.[26] When a person experiences chronic stress, the physiologic mediators of allostasis such as cortisol induce adverse effects such as diminished immune response or development of atherosclerosis. This is the mechanism by which chronic stress and high levels of stress adversely affect health through *weathering*.[27] Chronic stress or allostatic load can also induce epigenetic changes. *Epigenetic modifications* refer to DNA methylation, histone modifications, and noncoding RNAs that are the body's response to trauma. Cellular evidence such as shortening of telomere length, which is associated with shortened lifespan, is seen more frequently in persons exposed to chronic stress or discrimination.[28]

Epigenetic changes act as mediators of future adverse health. For example, epigenetic

modifications may explain the biologic manifestations of *historical trauma*, referring to the cumulative emotional and psychological damage that occurs over the lifespan and across generations following massive group experiences.[29] Epigenetics and historical trauma may also be partially responsible for the finding that black women have higher rates of preterm birth and higher rates of infant mortality than do white women with similar demographic characteristics.[30-32] In individuals, epigenetic changes may explain some of the findings that link prenatal or early childhood exposures with adverse health outcomes in adulthood.[27,30-32]

Life-Course Perspective

The response to stressors is highly variable among individuals, so another concept is needed to help midwives understand how these processes affect the health of individuals. The "life-course perspective" has been widely adopted by public health and maternal–child health advocates as a framework for understanding the interplay of SDOH and protective factors on health status.[32] This framework incorporates toxic environmental exposures, nutrition, stress, and health behaviors as environmental influences on biology, including fetal origins of adult disease. Most importantly, the life-course perspective incorporates the protective factors that individuals and communities employ in resistance to environmental risk factors, including resilience, social support, self-esteem, and self-efficacy. The *developmental origins of health and disease* (DOHaD) framework is a rapidly growing body of knowledge that encompasses the theories of allostasis, epigenetics, weathering, and life-course perspective to explore individual and multigenerational effects of prenatal stressors.[26-28,30-33]

Health Disparities

Health disparities are differences in health outcomes and the determinants of those outcomes between groups within a population, as defined by social, demographic, environmental, and geographic attributes.[34] Health disparities occur because unequal access to resources results in worse health outcomes for disenfranchised groups. In essence, health disparities are the downstream effect of social determinants of health. The following sections present an overview of some prominent well-documented health disparities that affect clients of midwifery care. Health disparities are not natural or inevitable, but rather are socially constructed—so they can be eliminated through social change.

Socioeconomic Status

Socioeconomic status (SES) is the term used to describe the social location of an individual, or population, with respect to that person's or population's access to money and resources. Markers frequently used to assess SES include wealth, income, profession, and educational attainment. SES cannot be adequately represented by income level alone, however, because income typically fluctuates significantly over the course of an individual's lifetime. Additionally, SES at specific moments over the life course may have more or less impact; for example, some evidence suggests that childhood SES impacts adult health independent of adult SES.[35]

Socioeconomic status is difficult to quantify because multiple factors influence its effects. For example, abundant evidence shows that the general SES in a neighborhood is correlated with health outcomes independent of individual SES.[36] Low-SES neighborhoods tend to have a cluster of social and politically mediated structural disadvantages that impact both acute and long-term health, such as lack of access to high-quality food sources, increased exposure to environmental toxins, lower-quality education and housing options, and poor access to public transit.[36]

Despite the difficulties inherent in accurately assessing SES, a significant body of evidence indicates that SES is correlated with health, and that this effect is dose-dependent. As a consequence, populations with low SES tend to have poor health outcomes, middle-SES populations tend to have average health outcomes, and high-SES populations tend to have the best health outcomes.[3,36,37]

Racial Disparities

The United States faces a crisis related to the ubiquitous and persistent race-based disparities in health for all persons including maternal–child health. Racial differences in health status and prevalence of illness are not due to genetic differences between racial groups. In fact, there are no specific genetic characteristics that differentiate one "race" from another, and there is just as much genetic diversity within racial groups as there is between them.[38] A person's race does not provide substantive biological information about that individual, nor does it provide an account of the ancestry or culture of the individual. As Dr. Camara Jones, president of the American Public Health Association, has noted, "the variable 'race' is not a biological construct that reflects innate differences, but a social construct that precisely captures the impacts of racism."[39] Nevertheless, racism has resulted in the systematic disenfranchisement of peoples of color within the United States—a factor that is responsible for multiple racial/ethnic disparities. For this reason, race and ethnicity are useful indicators when conducting research about individuals and groups who have borne the brunt of systemic injustice.[40,41]

Racism has traditionally been assumed to operate on three levels: institutionalized, personally mediated, and internalized. In recent years, however, this framework has been expanded to include systemic racism.

- *Institutionalized racism* is the "differential access to the goods, services, and opportunities of society by race."[39] This level of racism is deeply embedded in the structures of our society and "often manifests as inherited disadvantage."[39] It is often invisible and has no identifiable perpetrator. Institutionalized racism shapes an individual's relationships with the social determinants of health, and is the reason that race is associated with socioeconomic status.[39]
- *Personally mediated racism* involves prejudice and discrimination. Prejudice is the "differential assumptions about the abilities, motives, and intentions of others according to their race," whereas discrimination is "differential actions toward others according to their race."[39] This level of racism encompasses conscious discrimination as well as unconscious or implicit bias, and often manifests as disrespect, devaluation, and dehumanization.
- *Internalized racism* is defined as "acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth."[39]
- *Systemic racism* refers to the way racism is embedded in society via policies, structures, and norms (e.g., norms such as white race racial frames) that perpetrate racism.

An overview of race-based disparities in maternal health outcomes, infant health outcomes, and access to and utilization of prenatal care is presented here because these are prominent examples of racial health disparities germane to midwifery practice. However, racial disparities in health outcomes are not limited to maternal–child health.

Racial/Ethnic Disparities in Maternal–Child Health Outcomes

In the United States, black women have a three- to four-fold increased risk of dying of a pregnancy-related cause compared to white women.[42] This racial disparity is even more severe in some specific regions of the country. A study conducted in New York, for example, found black women were 12 times more likely to die from pregnancy-related causes than white women.[43] Although black women do not experience higher rates of preeclampsia, eclampsia, placental abruption, placenta previa, or postpartum hemorrhage compared to white women, they are significantly more likely to die from these complications.[44] Other groups of color are also at increased risk for maternal mortality, with Native Americans/Alaska Natives being 1.5 times more likely to die in childbirth than white women. Maternal mortality is just the tip of the iceberg, though—for every maternal death, there are 100 women who experience severe maternal morbidities.[42]

Similarly, rates of infant mortality demonstrate striking racial disparities. Infants born to Native Americans are 1.5 times more likely to die before their first birthday, and infants born to black parents are 2 times more likely to die in the first year of life. Data suggest that 80% of the black–white disparity in infant mortality is associated with a racial disparity in rates of extremely premature birth.[44] Black and Native Americans are at much greater risk for preterm birth than other racial groups. Racial disparities in preterm birth are especially disturbing because the health impacts for survivors of such births are lifelong effects. Black women are also at increased risk for fetal growth restriction and fetal demise.[44]

Although the etiology of health disparities is complex, multifactorial, and incompletely understood, some of these outcomes can be attributed to social inequities. For example, the higher rates of fetal growth restriction may be due to the fact that black women are at higher risk for having inadequate pregnancy weight gain and experiencing food insecurity in pregnancy, and are more likely to live and work in areas with high levels of environmental pollutants.[44] Most studies have found that Hispanic women are at increased risk for giving birth to infants with neural tube defects. In one study, the spina bifida rate for Hispanic people was 4.18 per 10,000 births, compared to 2.90 and 3.37 per 10,000 births for black and white people, respectively.[44] Hispanics have also been found to have lower rates of folic acid intake —a factor that is associated with an increased risk for neural tube defects.

Native American, Hispanic, and non-Hispanic black women are also at higher risk for starting prenatal care late compared to their white counterparts. In 2014, 11% of Native American women and 10% of black women initiated prenatal care in the third trimester or not at all, compared to only 4% of white women.[45] Insurance status and access to housing, transportation, and child care are all key social factors contributing to the etiology of this disparity.[45] Another likely contributor is perceived discrimination.

Racial and ethnic differences in maternal–child health outcomes are not explained by difference in genetics or “culture,” as studies continue to find that foreign-born individuals have better birth outcomes than U.S.-born persons of the same racial/ethnic groups, despite the fact that foreign-born women tend to start prenatal care later in pregnancy and have lower levels of formal education.[45] Most telling, the significant disparities in infant mortality, low birth weight, and preterm birth are not seen with foreign-born blacks, Asians, Hispanics, and

Filipinos. This phenomenon supports the hypothesis that racial disparities in maternal–child health outcomes are at least partially caused by racism and the inequitable social structures it creates, rather than any innate genetic differences between racial groups.[44-46]

Sexual Orientation and Gender Identity

Formal mechanisms of demographic assessment such as the U.S. Census have been slow to include data on sexual orientation, and still do not collect data on gender identity. Nonetheless, it is clear that a large number of lesbian, gay, bisexual, queer (LGBQ) and transgender and gender-nonconforming (TGNC) individuals live in the United States; a 2016 Gallup poll found approximately 10 million adults in the United States identified as lesbian, gay, bisexual, or transgender, with higher rates observed among people of color.[47] A Williams Institute survey published in 2016 estimated that there are 1.4 million TGNC persons in the United States.[48] LGBQ and TGNC persons are disproportionately affected by poverty.[49] TGNC communities, in particular, report severe poverty, regardless of educational attainment. In general, LGBQ and TGNC people of color are significantly more likely to experience poverty than their white peers.[49] **Table 3-2** defines some common terms used in relation to gender identity.[50-52]

Table 3-2 **Selected Terms Regarding Gender Identity**

Term	Definiton
Agender	A person whose understanding of self is outside of the concept of gender, or does not align with a specific gender identity.
Binary gender construct	A social construct that recognizes only two, mutually exclusive gender identities: male and female. In the United States, gender has historically been understood only through a binary construct.
Cisgender	A person whose gender identity is in alignment with the sex assigned at birth; for example, a person who was assigned female at birth and identifies as a woman.
Gender	A social rather than biological construct, which assigns specific roles, traits, and responsibilities to a person based on the sex assigned at birth. These assigned characteristics are rooted in culture and may vary significantly between cultural groups.
Gender expression	The way in which a person outwardly expresses gender. Gender expression includes mannerisms, style of dress, behavior, and modifiable aspects of physical appearance.
Gender identity	A person's internal understanding of self in regard to gender. Gender identity may or may not be in alignment with one's sex or gender expression.
Gender nonconforming	A person whose gender identity is not limited to solely male or solely female. This umbrella term covers a diverse array of gender identities.
Nonbinary	Similar to "gender nonconforming," this is an umbrella term describing a person whose gender identity is not limited to solely male or solely female.
Sex	A construct that classifies people into separate categories based on their chromosomal makeup and the appearance of their genitals. One's natal sex is the sex that was assigned at the time of birth. Although often thought of as binary, in reality there is a great deal of human variation in regard to sex.
Transgender	A person whose gender identity is not in alignment, in some way, with the sex assigned at birth. Transgender people can have binary or nonbinary gender identities.
Transgender woman	A person who was assigned the male sex at birth and has a female gender identity. Transgender women may or may not pursue medical or surgical interventions to better align their physical appearance with their gender identity.
Transgender man	A person who was assigned the female sex at birth and has a male gender identity. Transgender men may or may not pursue medical or surgical interventions to better align their physical appearance with their gender identity.

Based on Fenway Health. Glossary of gender and transgender terms. 2010 Revision. Available at: http://fenwayhealth.org/documents/the-fenway-institute/handouts/Handout_7-C_Glossary_of_Gender_and_Transgender_Terms__fi.pdf. Accessed December 19, 2017[50]; Selix NW, Rowniak S. Provision of patient-centered transgender care. *J Midwifery Womens Health*. 2016 Nov;61(6):744-751[51]; Centers for Disease Control and Prevention. Lesbian, gay, bisexual and transgender health: transgender persons. Available at: https://www.cdc.gov/lgbthealth/transgender.htm Accessed December 19, 2017.[52]

Today's healthcare infrastructure and systems are ill prepared to meet the needs of LGBQ and TGNC individuals. Non-inclusive data collection, gendered clinic bathrooms, and assumptions of heterosexuality by providers and staff all contribute to a hostile care environment and decrease safety. Electronic health record (EHR) systems are particularly problematic in their inability to accurately capture key health data such as sexual identity, natal sex, and organ systems present; they also create barriers to respectful care by failing to provide clear systems for documenting preferred names and pronouns.[53] Healthcare assessment and decision making are impaired when the provider does not have access to this information.

Research has demonstrated both explicit and implicit biases against LGBQ and TGNC persons among healthcare providers.[54,55] In large national surveys of TGNC individuals, respondents frequently reported delaying or avoiding needed health care or preventive health services due to fear of discrimination.[49]

There are no published studies that describe LGBQ and TGNC content in midwifery curricula. In a national study of obstetrics and gynecology providers, 80% of respondents received no training on TGNC health during residency; only 35.3% reported that they would be comfortable providing care to transgender men, and 29% reported that they would be comfortable providing care to transgender women.[56] These findings are congruent with data regarding the lack of LGBQ and TGNC clinical content in both medical and nursing education programs in the United States. Lim et al.'s 2015 national survey of baccalaureate nursing school faculty demonstrated significant deficits in these areas: 75% of respondents reported that LGBQ- and TGNC-related content was entirely absent or very limited in their school's curriculum, and few reported adequate knowledge on issues pertinent to this population.[55] Lim et al. found a median of 2.12 hours of nursing curriculum time devoted to classroom teaching on LGBQ and TGNC health. Similarly, Obedin-Maliver et al.'s 2011 survey of 176 U.S. medical schools found a median of 2.0 clinical hours related to LGBQ and TGNC health in allopathic training programs, and 0 hours in osteopathic training programs.[55,57]

LGBQ and TGNC Health Disparities

As with many marginalized communities, research on LGBQ and TGNC health is lacking in scope, quantity, and quality, limiting our understanding of the disease burden and health disparities within this population. While limited, the existing data do demonstrate that LGBQ and TGNC individuals experience disparities in both physical and mental health, including those related to depression, anxiety, smoking, alcohol use, and substance abuse. Rates of suicide are higher among LGBQ and TGNC youth as well as among TGNC individuals of all ages compared to their heterosexual peers.[58] Suicidality is highest among those who have experienced social insults such as family rejection, and for TGNC people of color. Available

data also suggest that LGBQ and TGNC persons are at risk of transmission of all sexually transmitted infections (STIs) including human papillomavirus (HPV) and human immunodeficiency virus (HIV), although this risk often goes unrecognized by healthcare providers who treat LBQ women and TGNC individuals.

Metabolic and Cardiovascular Health

Research has consistently demonstrated that LBQ women are more likely to be overweight or obese than their heterosexual peers, with disparities in weight presenting as early as adolescence.[59-61] The clinical significance of this finding, however, is less clear. Systematic reviews of existing data have failed to find evidence of increased rates of obesity-related morbidity such as diabetes, hypertensive disorders, or lipid disorders among LBQ women.[59,62] Despite significant differences in weight status, research has also failed to show a difference in physical activity levels and healthfulness of diet in LBQ versus cisgender heterosexual women.[60] Additionally, limited research has suggested that many overweight or obese LBQ women are metabolically healthy. Similarly, research has revealed greater cardiovascular risk factors in adult LBQ women but has not clearly demonstrated an increase in actual cardiovascular morbidity and mortality.[59,61-63] Data on weight status and metabolic health among TGNC individuals are sparse.

Sexual Health

Misinformation about transmission of STIs and vaginal infections between LBQ women and TGNC individuals is common among both providers and patients. This misunderstanding has contributed to a paucity of research about to STI rates and STI prevention strategies in LBQ women. The data that are available demonstrate that LBQ and TGNC people are at risk of transmission of all STIs including chlamydia, HPV, and HIV.[49,64-69] Bacterial vaginosis (BV) is common in LBQ women,[67] but data are not available on rates of BV in TGNC natal females.

Although the risk of direct female-to-female sexual transmission of HIV is low, HIV infection risk in LBQ women must be assessed in the full context of the individual's life risk factors, which may include sharing of needles during intravenous drug use, sex with natal males, and perceptions of invulnerability. For providers, it is important to recognize that identity and behavior do not always match; actual behaviors must be assessed by taking a comprehensive sexual history.

While data are still scant, more information is available on the incidence of HIV in TGNC persons. Data from the National HIV Surveillance System reveal that a total of 2351 TGNC people were newly diagnosed with HIV between 2009 and 2014; 84% were transgender women, 15.4% were transgender men, and 0.7% were people with nonbinary or other gender identities.[69] The majority of these diagnoses were in African American TGNC individuals.

It is likely that HIV and STI rates in LBQ women and TGNC people are underestimated due to deficiencies in reporting systems, bias in research design, provider discomfort with completing appropriate sexual health histories, and lack of provider knowledge about disease transmission risk.[70] For example, few studies capturing data on sexual health in transgender

men report the natal sex of participants' sexual partners, likely under the assumption that transgender men partner only with natal females.[71] Without this information, it is difficult to assess magnitude of risk; this is particularly concerning given that risky sexual behaviors in transgender men are associated with partnering with natal males.[71]

Violence Against LGBQ and TGNC People

Violence against LGBQ and TGNC persons accounts for a significant proportion of hate-based violence in the United States, and is most common against persons of color.[72] Transgender women of color bear the highest burden of lethal anti-LGBQ and TGNC hate violence in the United States. In November 2017, the Human Rights Campaign and the Trans People of Color Coalition released a report confirming that a total of 25 murders of TGNC people had been reported in the United States that year; another 3 TGNC people were murdered by the end of the year.[73] The number of reported LGBQ and TGNC hate crimes is lower than the true incidence due to both victim hesitance to report these crimes and failure of the law enforcement system to properly investigate and classify these crimes.[74]

Violence against LBQ and TGNC persons, including hate-based violence, often occurs at the hands of school peers, family members, and intimate partners. In a 2014–2015 national survey of 10,528 gay, LBQ, and TGNC youth between the ages of 13 and 21 years, experiences of discrimination and fear were common. In this cohort, 58% reported that they felt unsafe while at school due to their sexual orientation; 27% reported experiencing physical harassment and 13% reported experiencing physical assault due to their sexual orientation.[75] Among students who were subjected to discrimination due to their gender identity, 43% reported feeling unsafe, 20% reported physical harassment, and 9% reported physical assault.[76]

Gender-Based Violence

The term *gender-based violence* is used to describe the violence that occurs as a result of gender role expectations and inequality in the access to power, money, and resources by different genders.[77] This term is broad enough to capture shared root causes in a range of expressions of violence and dominance. In this section, four forms of gender-based violence that have implications for clinical midwifery care are reviewed: (1) violence against LGBQ and TGNC persons, (2) intimate-partner violence (IPV), (3) reproductive coercion, and (4) sexual coercion.

In the United States, 1 in 3 cisgender women and 1 in 6 cisgender men will experience sexual violence in their lifetime. Midwives provide sexual and reproductive health care to individuals of all ages and particularly during times when the psychological effects of gender-based violence may be triggered, such as pregnancy, labor, and birth. The topic of how gender-based violence affects health and the provision of *trauma-informed care* is of particular importance in midwifery. Midwives must be prepared to offer harm-reducing strategies and ready to provide referrals as needed. Harm-reducing strategies include discrete discussions regarding contraceptive options and establishing a safety plan.

Family and intimate-partner violence are also common experiences for LGBQ and TGNC youth and adults. In the 2015 U.S Trans Survey, 10% of respondents reported violence perpetrated by a family member due to their gender identity (at any age), and 26% had been excommunicated by an immediate family member due to their gender identity.[49] Some data suggest that rates of IPV among LGBQ and TGNC adults are approximately the same as those among heterosexual couples;[78] other data suggest higher lifetime rates, including for severe physical IPV.[49,79] Among LBQ women, bisexual women experience the highest rates of IPV and are also more likely than their lesbian and heterosexual peers to experience sexual violence.[79]

Intimate-Partner Violence

Intimate-partner violence is a pattern of assaultive and coercive behaviors that includes inflicted physical injury, psychological abuse, sexual assault, progressive social isolation, stalking, deprivation, intimidation, and threats. IPV is perpetrated by someone who is, was, or wishes to be involved in an intimate or dating relationship, and these behaviors are aimed at establishing control over one partner by the other.[80] IPV can affect any person, regardless of gender, sexual orientation, race, physical ability, or SES. Although cisgender men and women both experience IPV, significantly more cisgender women than men report needing medical care and experiencing longer-term consequences such as ongoing fear and post-traumatic stress disorder symptoms.

Intimate-partner violence may occur for the first time or worsen during pregnancy.[82,83] Persons who are pregnant or in the first year postpartum are at increased risk for homicide, with the majority of these crimes occurring secondary to IPV.[80,82,83] Beyond physical outcomes of IPV, victims are at increased risk for a range of other negative health-related sequelae, including STIs, pelvic inflammatory disease, and unintended pregnancy. Persons who have experienced IPV also have higher rates of physiological disorders and mental health conditions

such as chronic pain, neurologic disorders, gastrointestinal disorders, migraine headaches, depression, post-traumatic stress disorder, anxiety disorders, substance abuse, and suicidal behavior. Among women who are pregnant, IPV is associated with an increased risk for preterm birth and low birth weight.[84] Pregnant women who are hospitalized following assault have higher rates of multiple adverse outcomes, including placental abruption, uterine rupture, fetal demise, and preterm birth.[85]

Screening and support for women who have experienced IPV is reviewed in several chapters in this text. Many individuals may not choose to disclose current or past gender-based violence. However, routine screening can provide an opportunity to offer information about healthy relationships, safety plans, and nonjudgmental support.

For clients who disclose current or past experience with gender-based violence, further assessment of risks and information about safety plans and resources can be offered. It is also important to assess their current safety. All clients, including those who do not disclose current or past IPV, should be provided with safety information about violence. Evidence shows that receipt of safety information reduces violence and increases safety for recipients, even for those who did not disclose an unsafe relationship during the healthcare provider's assessment. Resources for IPV screening and safety information are listed at the end of this chapter.

Reproductive and Sexual Coercion

Reproductive coercion describes "behaviors aimed to maintain power and control in a relationship related to reproductive health by someone who is, was, or wishes to be involved in an intimate or dating relationship with an adult or adolescent."[86] Reproductive coercion includes tampering with a person's birth control in an explicit attempt to make the person become pregnant. It also includes coercive, threatening, or violent behavior intended to make a person end a pregnancy the individual does not want to end or continue a pregnancy the individual does not want to continue. People who are the victims of IPV are at higher risk for reproductive coercion.[86,87]

Sexual coercion "happens within the context of a relationship" and includes a range of behaviors that are used to coerce a person to have sex but do not rely on physical force. Examples of sexual coercion include forcing or repeatedly pressuring a partner to have sex, refusing to use a condom and nonconsensual removal of a condom during sex, intentionally exposing a partner to an STI, or threatening or harming a partner when notified about a positive STI test.

Trauma-Informed Care

Healthcare examinations can trigger fear, trauma, or post-traumatic stress symptoms in persons who have a history of sexual abuse, prior experience with insensitive providers, or prior experience of painful care. Furthermore, the United States has a history of obstetric violence and reproductive coercion that disproportionately affects women of color, a fact that contributes to historical trauma.[88] Approximately one-third of cisgender females have experienced sexual abuse. Because pelvic examinations are commonly recommended in

midwifery practice, trauma-informed care is another technique of import that all midwives can incorporate into their clinical practice.[89] Trauma-informed care includes multiple strategies that allow the client to maintain control, including careful attention to respectful language and obtaining permission, shared decision making, self-collection of vaginal samples, offering to let the client insert a vaginal speculum, and avoiding use of stirrups.

Techniques and Tools to Address Health Disparities in Midwifery Practice

The combined effects of social determinants of health, healthcare provider bias, and discrimination form a potent force that creates and perpetrates health disparities. The population served by the midwifery profession is more racially and ethnically diverse than is the population as a whole,[90] which makes health disparities a topic of prime importance in midwifery practice. This section presents an introductory overview of ways midwives can provide care that incorporates knowledge about social determinants of health, healthcare provider bias, and the effects of discrimination.

Cultural Competency, Cultural Humility, and Structural Competency

Culture is an integrated pattern of human behavior that includes the sum of actions, behaviors, language, thoughts, communications, customs, beliefs, values, and institutions that differentiate one group of people from another group. Culture is dynamic, not static. Moreover, culture is necessarily made up of both visible and invisible components. For example, actions and behaviors are visible components of culture, while the ideas, values, and attitudes that support action and behavior are not visible. Failure to recognize the core aspects of culture that are not visible is a major impediment in any effort to meet an individual's needs.

As the U.S. population has become more diverse, the need to provide *culturally and linguistically appropriate services (CLAS)* has become increasingly necessary in healthcare delivery. CLAS standards for healthcare services were initially developed in 2000 by the U.S. Department of Health and Human Services Office of Minority Health[91]; these standards provide a blueprint for implementing culturally and linguistically appropriate services. In 2003, the publication of *Unequal Treatment*: *Confronting Racial and Ethnic Disparities in Health Care* by the Institute of Medicine further documented the need for CLAS in healthcare services.[14] Subsequently, healthcare organizations have established requirements for cultural competency training.

The *cultural competency* framework requires providers and healthcare systems to "understand the importance of social and cultural influences on patients' health beliefs and behaviors and consider how these factors interact at multiple levels of the health care delivery system." Key to achieving cultural competence is the expansion of cultural knowledge and subsequent adaptation to cultural differences.[92]

Despite being a popular framework that has been widely promoted, cultural competence training has had only modest success. Notably, these programs improve knowledge and attitudes among providers, but have only a limited effect on patient outcomes.[93] In fact, the cultural competency framework may reinforce stereotypes rather than expand awareness and understanding. For example, "culture" is often conflated with racial or ethnic identity, thereby reinforcing racial stereotypes. Perhaps one of the most important critiques of the cultural competency framework is that it maintains the normative perspective of the dominant social categories, which suggests that the patient is responsible for culturally assimilating so as to

improve health.[94]

Nonetheless, there is obvious inherent value in understanding the cultural beliefs and norms that influence a person's perceptions and behaviors. This understanding can be better achieved when approached from a *cultural humility* perspective: "a commitment and active engagement in a lifelong learning process that individuals enter into on an ongoing basis with patients, communities, colleagues and themselves."[95] In addition to promoting ongoing attentiveness, respect, and openness toward others' perspectives and experiences, cultural humility focuses on ongoing critical self-reflection of one's individual culture(s), acknowledging personal beliefs, values, and experiences. This includes critical self-reflection on the culture of the midwifery profession and the healthcare system. Cultural humility encourages consideration of the power dynamics in the patient–provider relationship so that it can be reframed to a partnership and opportunity for advocacy.

Each healthcare interaction occurs in the context of three cultures: (1) the midwife's lived experience, (2) the experiences of the person seeking care, and (3) the culture of the healthcare system itself. Cultural norms and values shape a midwife's approach to care, and cultural forces shape the healthcare system as a whole. Thus, helping a person attain optimal health requires an awareness and assessment of the effects of all three cultures.

In describing any culture or cultural practice, it is important to note that within-group differences are likely to be as great as across-group differences. Wide variations in attitudes, beliefs, and behaviors exist among individuals. To assume that people who share a common culture and language are alike in all ways is to make a dangerous mistake. In addition, just as individuals may have multiple social identities, they may identify with multiple cultures simultaneously and over the course of their lifetimes.

Structural Competency

Structural competency extends the concept of cultural humility; it is the trained ability to understand how symptoms, attitudes, or diseases represent downstream implications of a wide variety of upstream structural systems. Structural competency is defined by the development of five core skills: (1) development of an extra-clinical language of structure, (2) rearticulation of "cultural" presentations in structural terms, (3) imagining of structural interventions, (4) development of structural humility, and (5) recognition of the structures that shape clinical interactions.[96]

Developing an extra-clinical language of structure requires an awareness of how social structures affect the health of communities. Rearticulating "cultural" presentations in structural terms invites the midwife to recognize and describe the structural causes of "cultural," "behavioral," or "lifestyle" risk factors—for example, recognizing and exploring the impact of food deserts and lack of access to safe recreational spaces on the food- and exercise-related habits of clients from low-SES neighborhoods. Imagining structural interventions encourages the midwife to conceive of and participate in developing interventions that address barriers to optimal health. Developing *structural humility* ensures that the midwife recognizes the need to continue learning. This is a lifelong commitment to learning and growing. Taken together, these four skills sets help the midwife recognize the structures that shape clinical interactions,

including the various economic, social, and political forces that affect the person, as well as the contributions made by the power and privilege dynamic between the patient and the clinician in the clinical encounter.

Interventions That Address Social Determinants of Health

Interventions that improve health disparities often involve multidisciplinary partnerships between healthcare providers and community organizations.[97-100] A thorough review of all interventions that may potentially decrease health disparities is beyond the scope of this text, but the reader is referred to the Resources section at the end of this chapter and recent reviews for more information.[97-100] Among the interventions that show promise as a means of improving health disparities in maternity care are screening for social determinants of health at initiation of care, bundling of prenatal care with other services that address social determinants known to affect the population, CenteringPregnancy group prenatal care, home visiting programs using community health workers and nurses, and accountable care organizations.[45] One intervention that has not yet been given significant attention is increasing diversity among the healthcare workforce.

Racial and Ethnic Disparities Effects on the Midwifery Workforce

A growing body of literature demonstrates that racial diversity in the healthcare workforce increases access to care, boosts adherence relative to treatments, and improves the quality of patient–provider interactions for people of color.[101,102] Midwives of color serving clients of similar backgrounds are uniquely positioned to understand the social, economic, and political forces that affect their patients. Cultural and *values concordance* facilitates communication and the provision of high-quality care.[101,102] Although racially diversifying the healthcare workforce is certainly an important intervention that can reduce racial disparities in health, currently 98% of nurse-midwifery practitioners in the United States identify as white.[103] Racism also limits access to midwifery education. Studies of midwives of color have found that institutional and interpersonal racism is pervasive in midwifery education programs, clinical settings, and professional organizations, and this racism poses a barrier to persons of color joining the midwifery profession.[104]

How did midwifery evolve into a predominantly white profession? Also, how does the profession's lack of racial diversity impact a midwife's ability to care for persons from diverse populations? A brief history of midwifery in three different communities of color offers a glimpse into the work of midwives in these communities and highlights the care they have provided. These stories exemplify the value of social/cultural concordant care and show how midwives who are part of the communities that they serve can bridge the gap between cultures to better meet women's needs. These stories also document how racism has contributed to the elimination of midwives of color, thereby producing the lack of workforce diversity now apparent.

New Mexico's Curandera-Parteras

—Felina M. Ortiz

Historically, traditional midwives, especially in communities of color, provided much of the maternity care. Most were elders in their community and were apprenticed for many years by their ancestors.[105] The midwives shared the communities' language and culture. As allopathic medicine was increasingly adopted and traditional midwifery became less valued in many parts of the United States, New Mexico was one of the few states that worked with the traditional Hispanic midwives, known as *curandera-parteras*, instead of against them. Officials of the New Mexico Department of Health recognized the challenge of providing obstetric care in the rural communities and the need for the *curandera-parteras*. In 1936, New Mexico's public health service bridged traditional health and modern medical care with a formal midwifery consultant program.[105] This program offered educational support and supplies to the *curandera-parteras*, while they in turn provided much needed obstetric care in hard-to-reach communities and completed the paperwork that improved New Mexico's vital statistics database. This partnership was one of the many elements that contributed to New Mexico being the state with the highest percentage of midwife-attended births in the nation to this day, almost a century after the program began.

In 1979, New Mexico introduced regulations that required all practicing *curandera-parteras* to obtain formal education and pass the licensing examination in English. These regulations worked toward the elimination of these practitioners, as many of them were not English literate.[105] This system of oppression and the loss of these practitioners disrupted an established system of maternal–child health care and is a potent illustration of institutional and systemic racism.

Grand Midwives of African Ancestry

—Patricia O. Loftman, Betty Jane Watts Carrington, Heather Clarke, Carolyn Curtis, and Karline Wilson-Mitchell

The origin of the traditional grand midwife began in Africa and was transplanted to the Americas through the trans-Atlantic slave trade in the 1600s. The grand midwives brought their knowledge, skills, and cultural traditions of childbirth that had been transmitted throughout generations via observation, apprentice training, personal experience, and oral tradition. During the period of enslavement, these midwives attended births, provided many other healthcare services, and were the foundation of the healthcare system for enslaved people on plantations. The midwife was respected and revered not just as an elder, but as a central and integral figure in the home and community. Grand midwives lived in the community, knew families intimately, and acted as an advocate and a source of comfort and support as they carried on the tradition of serving families through generations.

Onnie Lee Logan (1910–1995), who delivered hundreds of babies between 1931 and 1984, seemed to know every secret, herb, folk remedy, and "God-given motherwit" required to give birth in a poor community. She performed a role that many midwives filled, meeting needs holistically, whether catching a baby or providing food to a poor family.[17] Margaret Charles Smith (1906–2004) worked as a midwife in the days of Jim Crow laws that enforced racial segregation in the Southern United States. Ms. Smith described how she had to enter through the side or back door to attend white mothers. She also described how home births provided

the opportunity for the midwife to "stay with the woman as long as she needed," in contrast to the lonely hospital environment. She viewed her role as providing support, knowledge, and advocacy in her community.[106]

Discriminatory regulations developed by campaigning physicians and public health departments successfully eliminated grand midwives from practice in the 1930s and 1940s using methods that would today be described as structural and systemic racism. Physician representatives created financial incentives associated with providing maternity care for their peers and promoted propaganda that described African American midwives as "illiterate, uneducated, dirty, and dangerous." High rates of maternal and infant mortality were blamed on the practices of the grand midwives rather than on unequal access to public resources. Grand midwives were subsequently required to register with local health departments to obtain a practice permit and were required to attend classes at state training institutes.[107] The campaign was effective—but as more births took place in the hospital with physician attendants, maternal and infant mortality sharply increased, with many deaths due to infectious diseases and complications from unnecessary medical interventions.

The first formal educational program for African American midwives, Tuskegee School of Nurse-Midwifery, opened in 1941 but was in existence for only 6 years. Tuskegee graduated 31 African American nurse-midwives who provided general health care, became liaisons and resources between the community and local health departments, contributed to reductions in maternal and infant morbidity and mortality rates, and educated future generations of African American midwives. Two of the most notable Tuskegee graduates were Maude Callen (1898–1990) and Constance Manning Derrell. Mrs. Callen served her community in South Carolina for more than 50 years, increasing the visibility of nurse-midwives. Traditional grand midwives laid the foundation and their legacy provides inspiration to contemporary midwives of color. Their work can be seen as a blueprint for the provision of high-quality, client-centered care

Indigenous Midwives and the Indian Health Service

—Susan Stemmler and Nicolle L. Gonzales

The history of Indigenous midwifery in the United States is a tapestry of anthologies, held in the hearts of birth knowledge keepers across Turtle Island (North America). Much of that knowledge was passed down through stories from grandmothers to granddaughters, and from medicine women to their apprentices. During the early twentieth century, while traditional grand midwives were serving African American and poor women in the South and *curandera-parteras* were attending the births of Latina women in New Mexico, indigenous traditional midwives were attending the births of American Indian and Alaska Native women in their homes.

Historically, indigenous traditional midwives were responsible for the births of American Indian and Alaska Native people. Over the centuries, American Indian and Alaska Native communities were transformed by U.S. governmental policies and predatory assimilation practices that forever changed the lives of American Indian people.

The relationship between tribal governments and the federal government is founded in the

U.S. Constitution, which recognizes that federally recognized Indian tribes are sovereign nations with certain inherent rights. This distinguishes American Indians and American Natives from all other ethnic groups in the United States. Many of the treaties recognize government-supplied health care as payment for the U.S. government taking Indian lands. Today the Indian Health Services (IHS), which is housed in the U.S. Department of Health and Human Services, is the principal provider of health care and health advocacy for the 567 federally recognized indigenous tribes in the United States.

Traditional midwives were not included or utilized as part of the IHS mission; however, traditional knowledge keepers and birth attendants continued to share ceremonial teachings with their communities outside the formal profession of nurse-midwifery. Today nurse-midwives working for IHS attend more than 75% of all births, although nearly all midwives employed by IHS are not of American Indian or American Native descent. Nonetheless, Native American nurse-midwives have worked to bridge indigenous customs with hospital care.[108] For example, Ursula Knoki-Wilson, who began practice at the Chinle Comprehensive Health Center hospital in 1977, received the 2017 W. J. Haffner American Indian/American Native Women's Health Award for her years of service to advancing women's health care and cultural awareness for American Indian and Alaska Native women.

There is currently a movement to reclaim indigenous birth knowledge within tribal communities. The definition and scope of indigenous midwifery are evolving, and many Indigenous women are seeking this knowledge through their community healers and midwives. Many indigenous midwives choose to work outside the healthcare systems because the dominant culture in these sites does not support the ceremonial wellness that is important to members of these communities. The history of indigenous midwifery is still being written and the future of indigenous midwifery is still being molded, while interweaving evidence-based practice and traditional indigenous practice.

Conclusion

This chapter presents a broad overview but only a brief introduction to a few of the critical factors that affect an individual's health. Space limitations precluded the inclusion of important topics such as health literacy and health numeracy, which are essential for understanding healthcare information. In addition, several critical health disparities were not addressed in this chapter, including those secondary to lack of access to healthcare services, a serious problem for many who live in rural settings. Similarly, information about the needs of populations such as religious minorities, immigrants, persons with disabilities, and persons exposed to trauma or adverse childhood experiences was not included. The content presented here can be used as a framework for a way to approach clients and what needs to be understood to care for clients who come from many different backgrounds, histories, and cultures—midwifery is a lifelong learning process.

Other chapters in this text address the clinical knowledge needed to offer specific healthcare services. Nevertheless, clinical expertise can be effective only when the topics presented in this chapter are addressed. Each healthcare encounter between a midwife and a client occurs within a social and cultural milieu that can either perpetrate health disparities or mitigate them. Addressing social determinants of health, avoiding healthcare provider bias, and providing care that meets the needs of the individual is essential every time, with every person.

Resources

Organization	Description	Webpage
Culturally and Linguistically Appropriate Services		
U.S. Department of Health and Human Services (DHHS)	Culturally and linguistically appropriate services (CLAS) standards.	https://www.thinkculturalhealth.hhs.gov/clas
	Resources for CLAS.	https://www.thinkculturalhealth.hhs.gov/resources/library
Cultural Responsiveness Training for Healthcare Providers		
U.S. Department of Health and Human Resources (DHHS)	Think Cultural Health is a website that has collected resources, online courses, and links to other resources for culturally and linguistically appropriate services.	https://www.thinkculturalhealth.hhs.gov/about https://www.hhs.gov/ash/oah/resources-and-training/tpp-and-paf-resources/cultural-c
	Office of Minority Health, national CLAS standards.	https://www.thinkculturalhealth.hhs.gov/pdfs/EnhancedNationalCLASStandards.pdf?utm_source=H2RMinute+PCMH+May+8%2C+2013&utm_campaign=H2R+Minutes+I
Gender-Based Violence		
American College of Obstetricians and Gynecologists (ACOG) and Futures Without Violence	"Addressing Intimate Partner Violence, Reproductive and Sexual Coercion: A Guide for Obstetric, Gynecologic and Reproductive Health Care Settings," 3rd ed.	https://www.futureswithoutviolence.org/userfiles/file/HealthCare/Reproductive%20Hea
Futures Without Violence	A four-panel double-sided tool folds up to the size of a business card.	https://www.futureswithoutviolence.org/is-your-relationship-affecting-your-health-safet

	The card helps women recognize how their relationship affects their health and provides information on safety planning. The back of the card provides information about referral to the National Domestic Violence Hotline.	
National Domestic Violence Hotline	This site has safety planning, information about legal issues, and additional resources.	http://www.thehotline.org/help/path-to-safety/#leaving
Health Literacy		
Agency for Health Care Research and Quality (AHRQ)	Health Literacy Universal Precautions Toolkit, 2nd ed.	https://www.ahrq.gov/professionals/quality-patient-safety/quality-resources/tools/litera
Health Provider Implicit Bias		
Harvard University	The Implicit Association Test (IAT) measures attitudes and beliefs that may be unconscious, identifying the strength of associations. It is validated and widely used.	https://implicit.harvard.edu/implicit/takeatest.html
LGBQ and TGNC Resources		
Center for Excellence in Transgender Health	Multiple resources for persons who are transgender and gender	http://transhealth.ucsf.edu

	nonbinary people, including guidelines for primary care, online education programs, and referrals.	
Fenway Institute	The National LGBT Health Education Center provides resources and education to support optimizing the health of LGBT persons.	https://www.lgbthealtheducation.org
Gay and Lesbian Medical Association (GLMA)	Guidelines for care of LGBT patients.	http://glma.org/_data/n_0001/resources/live/GLMA%20guidelines%202006%20FINAL.
World Professional Association for Transgender Health (WPATH)	Standards of care for the health of transsexual, transgender, and gender nonconforming people, version 7.	http://www.wpath.org/site_page.cfm?pk_association_webpage_menu=1351&pk_asso
Racial Health Disparities		
Agency for Health Care Research and Quality (AHRQ)	AHRQ activities to reduce racial and ethnic disparities in health care.	https://www.ahrq.gov/research/findings/factsheets/minority/disparities/index.html
Department of Health Policy, School of Public Health and Health Services, George Washington University	“Racial and Ethnic Disparities in Health Care: A Chartbook.”	http://www.commonwealthfund.org/usr_doc/Mead_racialethnicdisparities_chartbook_
Social Determinants of Health		
Centers for Disease Control and	Social determinants of health:	https://www.cdc.gov/nchhstp/socialdeterminants/resources.html

Prevention (CDC)	Definition, resources, statistics, and links to agencies that explore social determinants of health.	
Federal Office of Rural Health Policy - Rural Health Information Hub	Provides information and resources on rural health and health care, including approaches to improving rural health disparities.	https://www.ruralhealthinfo.org/about
Healthy People 2020	This website has multiple resources that explore causes and strategies to decrease adverse health outcomes secondary to social determinants of health.	https://www.healthypeople.gov/2020/topics-objectives/topic/social-determinants-healt
Trauma-Informed Care		
Reproductive Health Access Project	Information on techniques for trauma-informed pelvic examinations and additional resources.	https://www.reproductiveaccess.org/resource/trauma-informed-pelvic-exams/
Public Health Agency of Canada	"Handbook on Sensitive Practice for Health Care Providers: Lessons from Adult Survivors of Childhood Sexual Abuse."	https://www.integration.samhsa.gov/clinical-practice/handbook-sensitivve-practices4h

References

1. McGinnis JM, Williams-Russo P, Knickman JR. The case for more active policy attention to health promotion. *Health Affairs*. 2003;21(2):78-93.
2. Schroeder SA. Shattuck Lecture. We can do better-improving the health of the American People. *N Engl J Med*. 2007;357(12):1221-1228.
3. *Commission on Social Determinants of Health. Closing the Gap in a Generation: Health Equity Through Action on the Social Determinants of Health. Final Report of the Commission on Social Determinants of Health*. Geneva, Switzerland: World Health Organization; 2008. Available at: http://www.who.int/social_determinants/thecommission/finalreport/en/. Accessed January 16, 2018.
4. Centers for Disease Control and Prevention. Health disparities and inequities report—United States, 2013. MMWR Suppl. 2013;62(3):1-187. Available at: https://www.cdc.gov/mmwr/preview/ind2013_su.html#HealthDisparities2013. Accessed January 16, 2018.
5. Braveman P. The social determinants of health: it is time to consider the causes of the causes. *Public Health Rep*. 2014;129(suppl 2):19-31.
6. Bowleg L. The problem with the phrase women and minorities: intersectionality—an important theoretical framework for public health. *Am J Public Health*. 2012;102(7):1267-1273.
7. Hankivsky O, Grace D, Hunting G, et al. An intersectionality-based policy analysis framework: critical reflections on a methodology for advancing equity. *Int J Equity Health*. 2014;13:119.
8. Byrne A, Tanesini A. Instilling new habits: addressing implicit bias in healthcare professionals. *Adv Health Sci Educ*. 2015;20(5):1255-1262.
9. Hall WJ, Chapman MV, Lee KM, et al. Implicit racial/ethnic bias among health care professionals and its influence on health care outcomes: a systematic review. *Am J Public Health*. 2015;105(12):e60-e76.
10. Hatzenbuehler ML, Phelan JC, Link BG. Stigma as a fundamental cause of population health inequalities. *Am J Public Health*. 2013;103(5):813-821.
11. Teal CR, Gill AG, Green AR, Crandall S. Helping medical learners recognize and manage unconscious bias toward certain patient groups. *Med Educ*. 2012;46:80-88.
12. FitzGerald C, Hurst S. Implicit bias in healthcare professionals: a systematic review. *BMC Med Ethics*. 2017;18(1):19.
13. Stone J, Moskowitz GB. Non-conscious bias in medical decision making: what can be done to reduce it? *Med Educ*. 2011;45:768-776.
14. Institute of Medicine. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Washington, DC: National Academies Press; 2003. Available at: https://www.nap.edu/read/12875/chapter/1. Accessed January 20, 2018.
15. Byrne A, Tanesini A. Instilling new habits: addressing implicit bias in health care professionals. *Adv Health Sci Educ*. 2015;20(5):1255-1262.
16. Bay Area Regional Health Inequities Initiative. Available at: http://barhii.org/framework/. Accessed January 16, 2018.
17. Logan OL, Clark K. *Motherwit: An Alabama Midwife's Story*. San Francisco, CA: Untreed Reads Publishing; 1989, updated 2013.
18. Breckinridge M. *Wide Neighborhoods: A Story of the Frontier Nursing Service*. New York, NY: Harper; 1952.
19. Smith SL. *Japanese American Midwives: Culture, Community, and Health Politics, 1880–1950*. Champaign, IL: University of Illinois Press; 2010.
20. The JJ Way: A patient-centered model of care. *Commonsense Childbirth*. Available at: http://www.commonsensechildbirth.org/jjway/. Accessed January 16, 2018.
21. LaDuke W. *All Our Relations: Native Struggles for Land and Life*. Chicago, IL: Haymarket Books; 1999.
22. Robinson K, Garnier-Villarreal M, Hanson L. Effectiveness of CenteringPregnancy on breastfeeding initiation among African Americans: A systematic review and meta-analysis. *J Perinat Neonatal Nurs*. 2018. [Epub ahead of print]. doi:10.1097/JPN.0000000000000307.
23. Mazzoni SE, Carter EB. Group prenatal care. *Am J Obstet Gynecol*. 2017;216(6):552-556.
24. Richadson AS, Ghosh-Dastidar M, Beckman R, et al. Can the introduction of a full-service supermarket in a food desert improve residents' economic status and health? *Ann Epidemiol*. 2017;27(12):771-776.
25. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health*. 2006;96(5):826-833.
26. Shannon M, King TL, Kennedy HP. Allostasis: a theoretical framework for understanding and evaluating perinatal health outcomes. *J Obstet Gynecol Neonat Nurs*. 2007;36:125-134.
27. Patchen L, Rebok G, Astone NM. Differences in obesity rates among minority and white women: the latent role of maternal stress. *J Midwifery Womens Health*. 2016;61:489-496.
28. Shalev I, Entringer S, Wadhwa PD, et al. Stress and telomere biology: a lifespan perspective. *Psychoneuroendocrinol*. 2013;38(9):1835-1842.

29. Brave Heart M. The historical trauma response among Natives and its relationship with substance abuse: a Lakota illustration. *J Psychoactive Drugs*. 2003;35:7-13.
30. Latendresse G. The interaction between chronic stress and pregnancy: preterm birth from a biobehavioral perspective. *J Midwifery Womens Health*. 2009;54(1):8-17.
31. Willis E, McManus P, Magallanes N, Johnson S, Majnik A. Conquering racial disparities in perinatal outcomes. *Clin Perinatol*. 2014;41(4):847-875.
32. Lu M, Halfon N. Racial and ethnic disparities in birth outcomes: a life course perspective. *Mat Child Health J*. 2003:7(1):13-30.
33. Wadhwa PD, Buss C, Entringer S, Swanson JM. Developmental origins of health and disease: brief history of the approach and current focus on epigenetic mechanisms. *Semin Reprod Med*. 2009;27(5):358-368.
34. Cater PO, Baquet C. What is a health disparity? *Public Health Rep*. 2002;117:426-434.
35. Braveman PA, Cubbin C, Egerter S, et al. Socioeconomic status in health research: one size does not fit all. *JAMA*. 2005;294(22):2879-2888.
36. Iton A. Tackling the root causes of health disparities through community capacity building. In: Hofrichter R, Bahtia R, eds. *Tackling Health Inequities Through Public Health Practice: Theory to Action*. Oxford, UK: Oxford University Press; 2010:370-417.
37. National Center for Health Statistics. 2012. *Health, United States, 2011: With Special Feature on Socioeconomic Status and Health*. Hyattsville, MD: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/data/hus/hus11.pdf. Accessed January 20, 2018.
38. Williams DR, Sternthal M. Understanding racial/ethnic disparities in health: sociological contributions. *J Health Soc Behav*. 2010;51(suppl):S15-S27.
39. Jones CP. Levels of racism: a theoretic framework and a gardener's tale. *Am J Public Health*. 2000;90(8):1212-1215.
40. Bailey ZD, Kreger N, Agfenor M, Graves J, Linos N, Bassett MT. Structural racism and health inequities in the USA: evidence and interventions. *Lancet*. 2017;389(10077):1453-1463.
41. Braveman PA, Heck K, Egerter S, et al. The role of socioeconomic factors in Black–White disparities in preterm birth. *Am J Public Health*. 2015;105(4):694-702.
42. Creanga AA, Berg CJ, Ko JY, et al. Maternal mortality and morbidity in the United States: where are we now? *J Womens Health*. 2014;23(1):3-9.
43. New York City Department of Health and Mental Hygiene, Bureau of Maternal and Child Health. Pregnancy-associated mortality, New York City, 2006–2010. New York, NY: New York City Department of Health and Mental Hygiene, Bureau of Maternal and Child Health; 2015.
44. Bryant AS, Worjoloh A, Caughey AB, Washington AE. Racial/ethnic disparities in obstetrical outcomes and care: prevalence and determinants. *Am J Obstet Gynecol*. 2010;202(4):335-343.
45. Gadson A, Akpovi E, Mehta PK. Exploring the social determinants of racial/ethnic disparities in prenatal care utilization and maternal outcome. *Semin Perinatol*. 2017;41(5):308-317.
46. Alhusen JL, Bower KM, Epstein E, Sharps P. Racial discrimination and adverse birth outcomes: an integrative review. *J Midwifery Womens Health*. 2016;61:707-720.
47. Gates GJ. LGBT data collection amid social and demographic shifts of the US LGBT community. *Am J Public Health*. 2017;107(8):1220-1222.
48. Flores AR, Herman JL, Gates GJ, Brown TNT. *How Many Adults Identify as Transgender in the United States?* Los Angeles, CA: Williams Institute; 2016.
49. James SE, Herman JL, Rankin S, Keisling M, Mottel L, Anafit M. *The Report of the 2015 U.S. Transgender Survey*. Washington, DC: National Center for Transgender Equality; 2016. Available at: https://www.transequality.org/sites/default/files/docs/USTS-Full-Report-FINAL.PDF. Accessed December 19, 2017.
50. Fenway Health. Glossary of gender and transgender terms. 2010 revision. Available at: http://fenwayhealth.org/documents/the-fenway-institute/handouts/Handout_7-C_Glossary_of_Gender_and_Transgender_Terms__fi.pdf. Accessed December 19, 2017.
51. Selix NW, Rowniak S. Provision of patient-centered transgender care. *J Midwifery Womens Health*. 2016;61(6):744-751.
52. Centers for Disease Control and Prevention. Lesbian, gay, bisexual and transgender health: transgender persons. Available at: https://www.cdc.gov/lgbthealth/transgender.htm. Accessed December 19, 2017.
53. Callahan EJ, Hazarian S, Yarborough M, Sánchez JP. Eliminating LGBTIQQ health disparities: the associated roles of electronic health records and institutional culture. *LGBT Bioethics: Visibility, Disparities, and Dialogue, Special Report: Hastings Center Report*. 2014;44(5),48-52.
54. Sabin JA, Riskind RG, Nosek BA. Health care providers' implicit and explicit attitudes toward lesbian women and gay men. *Am J Public Health*. 2015;105(9):1831-1841.
55. Lim F, Johnson M, Eliason M. A national survey of faculty knowledge, experience, and readiness for teaching lesbian,

gay, bisexual, and transgender health in baccalaureate nursing programs. *Nurs Educ Perspect*. 2015;36:144-152.
56. Unger CA. Care of the transgender patient: a survey of gynecologists' current knowledge and practice. *J Womens Health*. 2015;24(2):114-119.
57. Obedin-Maliver J, Goldsmith ES, Stewart L, et al. Lesbian, gay, bisexual, and transgender-related content in undergraduate medical education. *JAMA*. 2011;306(9):971-977.
58. Duncan DT, Hatzenbuehler ML. Lesbian, gay, bisexual, and transgender hate crimes and suicidality among a population-based sample of sexual-minority adolescents in Boston. *Am J Public Health*. 2014;104(2):272-278.
59. Eliason MJ, Ingraham N, Fogel SC, et al. A systematic review of the literature on weight in sexual minority women. *Womens Health Issues*. 2015;25(2):162-175.
60. Jun HJ, Corliss HL, Nichols LP, Pazaris MJ, Spiegelman D, Austin SB. Adult body mass index trajectories and sexual orientation. *Am J Prevent Med*. 2012;42(4):348-354.
61. Ward BW, Dahlhamer JM, Galinsky AM, Joestl SS. Sexual orientation and health among U.S. adults: National Health Interview Survey, 2013. *Natl Health Stat Rep*. 2014;77:1-10.
62. Simoni JM, Smith L, Oost KM, Lehavot K, Fredriksen-Goldsen K. Disparities in physical health conditions among lesbian and bisexual women: a systematic review of population-based studies. *J Homosex*. 2017;64(1):32-44.
63. Clark CJ, Borowsky IW, Salisbury J, et al. Disparities in long-term cardiovascular disease risk by sexual identity: the national longitudinal study of adolescent to adult health. *Prevent Med*. 2015;76:26-30.
64. Marrazzo, JM, Koutsky LA, Kiviat NB, Kuypers JM, Stine, K. Papanicolaou test screening and prevalence of genital human papillomavirus among women who have sex with women. *Am J Public Health*. 2001;91(6):947-952.
65. Wood SM, Salas-Humara C, Dowshen NL. Human immunodeficiency virus, other sexually transmitted infections, and sexual and reproductive health in lesbian, gay, bisexual, transgender youth. *Pediatr Clin North Am*. 2016;63(6):1027-1055.
66. Muzny CA, Austin EL, Harbison, HS, Hook EW. Sexual partnership characteristics of African American women who have sex with women: impact on sexually transmitted infection risk. *Sex Transm Dis*. 2014;41(10):611-617.
67. Gorgos LM, Marrazzo JM. Sexually transmitted infections among women who have sex with women. *Clin Infect Dis*. 2011;53(suppl 3):S84-S91.
68. Singh D, Fine DN, Marrazzo JM. *Chlamydia trachomatis* infection among women reporting sexual activity with women screened in family planning clinics in the Pacific Northwest, 1997 to 2005. *Am J Public Health*. 2011;101(7):1284-1290.
69. Clark H, Babu AS, Wiewel EW, Opoku J, Crepaz N. Diagnosed HIV infection in transgender adults and adolescents: results from the National HIV Surveillance System, 2009–2014. *AIDS Behavior*. 2017;21(9):2774-2783.
70. Hayes V, Blondeau W, Bing-You RG. Assessment of medical student and resident/fellow knowledge, comfort, and training with sexual history taking in LGBTQ patients. *Fam Med*. 2015;47(5):383-388.
71. Reisner SL, White JM, Mayer KH, Mimiaga MJ. Sexual risk behaviors and psychosocial health concerns of female-to-male transgender men screening for STDs at an urban community health center. *AIDS Care*. 2014;26(7):857-864.
72. Ahmed O, Jindasurat C. *Lesbian, Gay, Bisexual, Transgender, Queer and HIV-Affected Hate Violence 2013*. New York, NY: National Coalition of Anti-Violence Programs; 2014.
73. Human Rights Campaign, Trans People of Color Coalition. *A Time to Act: Fatal Violence Against Transgender People in America 2017*. Washington, DC: Human Rights Campaign & Trans People of Color Coalition; 2017.
74. Nolan JJ, Haas SM, Turley E, Stump J, LaValle CR. Assessing the "statistical accuracy" of the national incident-based reporting system hate crime data. *Am Behav Sci*. 2015;59(12):1562-1587.
75. Human Rights Campaign. *Violence Against the Transgender Community in 2016*. Washington, DC: Human Rights Campaign; 2016.
76. Kosciw JG, Greytak EA, Gig NM, Villenas C, Danischewski DJ. *The 2015 National School Climate Survey: The Experience of Lesbian, Gay, Bisexual, Transgender, and Queer Youth in Our Nation's Schools*. New York, NY: GLSEN; 2016.
77. United Nations. Glossary on sexual exploitation and abuse. October 5, 2016. Available at: https://hr.un.org/sites/hr.un.org/files/UN%20Glossary%20on%20SEA.pdf. Accessed January 16, 2018.
78. National Center for Victims of Crime, National Coalition of Anti-Violence Programs. *Why It Matters: Rethinking Victim Assistance for Lesbian, Gay, Bisexual, Transgender, and Queer Victims of Hate Violence and Intimate Partner Violence*. Washington, DC: National Center for Victims of Crime & National Coalition of Anti-Violence Programs; 2010.
79. Walters ML, Chen J, Breiding MJ. *The National Intimate Partner and Sexual Violence Survey (NISVS): 2010 Findings on Victimization by Sexual Orientation*. Atlanta, GA: National Center for Injury Prevention and Control & Centers for Disease Control and Prevention; 2013. Available at: http://doi.org/10.1037/e541522013-001. Accessed January 8, 2017.
80. American College of Obstetricians and Gynecologists. Committee Opinion No. 518: intimate partner violence. *Obstet Gynecol*. 2012;119:412-417.
81. Centers for Disease Control and Prevention. The National Intimate Partner and Sexual Violence Survey. September 25, 2017. Available at: https://www.cdc.gov/violenceprevention/nisvs/index.html. Accessed January 18, 2018.

82. Brownridge DA, Taillieu TL, Tyler KA, Tiwari A, Chan KL, Santos SC. Pregnancy and intimate partner violence: risk factors, severity, and health effects. *Violence Against Women*. 2011;17(7):858-881.
83. Cheng D, Horon IL. Intimate-partner homicide among pregnant and postpartum women. *Obstet Gynecol*. 2010;115:1181-1186.
84. Donovan BM, Spracklen CDN, Schweizer ML, Ryckman KK, Saftlas AF. Intimate partner violence during pregnancy and the risk for adverse infant outcomes: a systematic review and meta-analysis. *BJOG*. 2016;123(8):1289-1299.
85. El Kady DE, Gilbert WM, Xing G, Smith LH. Maternal and neonatal outcomes of assaults during pregnancy. *Obstet Gynecol*. 2005;105:357-363.
86. American College of Obstetricians and Gynecologists. Committee Opinion No. 554: reproductive and sexual coercion. *Obstet Gynecol*. 2013;121(2 pt 1):411-415.
87. Clark LE, Allen RH, Goyal V, Raker C, Gottlieb AS. Reproductive coercion and co-occurring intimate partner violence in obstetrics and gynecology patients. *Am J Obstet Gynecol*. 2014;210(1):42-e1.
88. Reilly PR. Eugenics and involuntary sterilization: 1907–2015. *Annu Rev Genomics Hum Genet*. 2015;16:351-368.
89. Sperlich M, Seng JS, Li Y, Taylor J, Bradbury-Jones C. Integrating trauma-informed care into maternity care practice: conceptual and practical issues. *J Midwifery Womens Health*. 2017;62(6):661-672.
90. Declercq E. Midwife attended births in the United States 1990–2012. *J Midwifery Womens Health*. 2015;60(1):10-15.
91. U.S. Department of Health and Human Services. National CLAS standards. Available at: https://www.thinkculturalhealth.hhs.gov/clas. Accessed January 20, 2018.
92. Betancourt JR, Green AR, Carrillo JE, Ananeh-Firempong O. Defining cultural competence: a practical framework for addressing racial/ethnic disparities in health and health care. *Public Health Rep*. 2003;118(4):293-302.
93. Horvat L, Horey D, Romios P, Kis-Rigo J. Cultural competence education for health professionals. *Cochrane Database Syst Rev*. 2014;5:CD009405. doi:10.1002/14651858.CD009405.pub2.
94. Malat J. The appeal and problems of a cultural competence approach to reducing racial disparities. *J Gen Intern Med*. 2013;28(5):605-607.
95. Tervalon M, Murray-García J. Cultural humility versus cultural competence: a critical distinction in defining physician training outcomes in multicultural education. *J Health Care Poor Underserved*. 1998;9(2):117-125.
96. Hansen H, Metzl J. Structural competency in the U.S. healthcare crisis: putting social and policy interventions into clinical practice. *J Bioethical Inquiry*. 2016;13(2):179-183.
97. Centers for Disease Control and Prevention. Strategies for reducing health disparities: selected CDC sponsored interventions—United States 2014. *MMWR Suppl*. 2014;63(1):1-47.
98. McGlade MS, Saha S, Dahlstrom ME. The Latina paradox: an opportunity for restructuring prenatal care delivery. *Am J Pub Health*. 2004;94:2062-2065.
99. Chin MH, Clarke AR, Nocon RS, et al. A roadmap and best practices for organizations to reduce racial and ethnic disparities in health care. *J Gen Intern Med*. 2012;27(8):992-100
100. Council on Patient Safety in Women's Health Care Alliance for Innovation on Maternal Health. Patient safety bundle: reduction of peripartum racial/ethnic disparities. Available at: http://safehealthcareforeverywoman.org/wp-content/uploads/2017/11/Reduction-of-Peripartum-Disparities-Bundle.pdf. Accessed January 16, 2018.
101. U.S. Department of Health and Human Services. The rationale for diversity in the health professions: a review of the evidence. 2006. Available at: http://anyflip.com/vhdl/blku/basic. Accessed January 18, 2018.
102. Butler AS, Bristow LR, eds. *In the Nation's Compelling Interest: Ensuring Diversity in the Health Care Workforce*. Washington, DC: National Academies Press; 2004.
103. Fullerton J, Sipe TA, Hastings-Tolsma M, et al. The midwifery workforce: ACNM 2012 and AMCB 2013 core data. *J Midwifery Womens Health*. 2015;60(6):751-761.
104. Wren Serbin J, Donnelly E. The impact of racism and midwifery's lack of racial diversity: a literature review. *J Midwifery Womens Health*. 2016;61(6):694-706.
105. Ortiz FM. History of midwifery in New Mexico: partnership between *curandera-parteras* and the New Mexico Department of Health. *J Midwifery Womens Health*. 2005;50:411-417.
106. Smith MC, Holmes LJ. *Listen to Me Good: The Story of an Alabama Midwife*. Columbus, OH: Ohio State University Press; 1996.
107. Morrison SM, Fee E. Nothing to work with but cleanliness: the training of African American traditional midwives in the South. *Am J Public Health*. 2010;100:238-239.
108. Ogburn JA, Espey E, Pierce-Bulger M, et al. Midwives and obstetricians-gynecologists collaborating for Native American women's health. *Obstet Gynecol Clin North Am*. 2012;39:359-366.

APPENDIX

3A

Reproductive Health Statistics

MARY C. BRUCKER AND TEKOA L. KING

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Maternal and infant mortality statistics are used extensively to document trends in health status and the results of therapeutic interventions at the population level. These statistics are also used extensively in healthcare research and by governments and institutions worldwide when deciding where to dedicate resources. In the United States, data on births, deaths, and fetal deaths are collected by each state or jurisdiction and reported as vital statistics to the National Center for Health Statistics. The National Vital Statistics System is a partnership between the federal government, 50 states, 2 cities (Washington, DC, and New York City), and 5 territories (Puerto Rico, the Virgin Islands, Guam, American Samoa, and the Commonwealth of the Northern Mariana Islands). The states and territories have the legal responsibility to register vital events. Although most jurisdictions use the standard U.S. forms for birth and death data, some variations are apparent between states and territories with regard to which data are collected. Thus, the data collected are not completely uniform across all jurisdictions.

Health statistics are an accepted measure of the effectiveness of a healthcare system and widely used as indicators of a nation's health. Mortality statistics also illustrate how pregnant women and newborns fare both internationally and within the United States. There are striking differences in mortality and morbidity statistics between the United States, other developed nations, and low-resource nations. Within the United States, there are notable racial and ethnic disparities in maternal and infant health outcomes. This appendix briefly reviews definitions of health-related statistics and how they are used. Definitions of key reproductive statistics are presented in **Table 3A-1**.[1-3] The most recent infant mortality, fetal death, and pregnancy-related mortality ratios are presented in **Table 3A-2**[3-5] and **Figure 3A-1**.[6]

Table 3A-1	Selected Maternal/Child Health Indices

Term	Definition and Explanations
Fetal death rate (see Stillbirth)	Spontaneous death of the fetus occurs prior to birth, irrespective of the duration of the pregnancy. Fetal death is indicated by no signs of life after birth (e.g., heartbeats, umbilical cord pulsations, breathing, or voluntary muscle movement). The fetal death rate is the number of fetal deaths ≥ 20 weeks' gestation that occur during a year divided by the sum of live births plus fetal deaths during the same year, and expressed per 1000 live births plus fetal deaths.
Infant mortality rate (IMR)	Number of infants dying before reaching 1 year of life per 1000 live births in a given year.
Lifetime risk of maternal death or pregnancy-related death	Risk of an individual woman dying from pregnancy or childbirth during her life; calculated by multiplying the maternal mortality rate by 30, or the number of years of exposure to pregnancy between ages 15 and 44. Calculations are based on maternal mortality and fertility rates in the country. This method recognizes that women of high fertility or women lacking in universal access to effective family planning have an extremely high risk of dying as a result of pregnancy or childbirth, as they are repeatedly exposed to the risk of pregnancy.
Live birth	Complete expulsion or extraction of a product of human conception from a woman, irrespective of the duration of pregnancy. The newborn shows any evidence of life (ie, heartbeats, umbilical cord pulsations, breathing, or voluntary muscle movement), regardless of whether the umbilical cord has been cut or the placenta is attached. Heartbeats are distinguished from transient cardiac contractions and breathing is distinguished from fleeting respiratory efforts or gasps.
Maternal mortality rate	An estimate of the number of deaths during pregnancy or within 42 days after completion of the pregnancy, per 100,000 women of reproductive age in a given year. This rate is difficult to obtain and often not used in low-resource countries, since births are more easily measured than the number of women of reproductive age.
Maternal mortality ratio	An estimate of the number of deaths during pregnancy within 42 days after completion of the pregnancy, for every 100,000 live births. This ratio is the method most commonly used to express trends within a country and to make international comparisons. Note: The maternal mortality *ratio* differs from the maternal mortality *rate* in that the denominator is births and not women of reproductive age. The rate is more difficult to measure, especially in countries with less sophisticated data gathering.
Neonatal morality rate	The number of deaths of newborns in the first 28 days of life per 1000 live births.
Neonatal death rate (early versus late)	Early neonatal deaths are the number of neonatal deaths that occur before the first 7 days from birth. Late neonatal deaths are the number of neonatal deaths that occur between 7 and 27 days of age.
Perinatal mortality rate[a] (PMR)	The sum of fetal deaths (more than 20 weeks' gestation) plus early neonatal deaths (within the first 28 days after birth) during a year divided by the sum of live births plus fetal deaths during that year. Expressed per 1000 live births plus fetal deaths.
Pregnancy-associated death	Death of a person occurring during or within 1 year of pregnancy but not causally related to pregnancy. This definition contrasts with that of "not pregnancy-associated death"—that is, death of a person with a uterus who has never been pregnant or occurring more than 1year after the end of pregnancy. Reported as the pregnancy-related mortality ratio: pregnancy-related deaths per 100,000 live births.
Pregnancy-related death	Death of a person while pregnant or within 1 year of the end of a pregnancy—regardless of the outcome, duration, or site of the pregnancy—from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes. This includes a pregnancy complication, a chain of events initiated by pregnancy, or the aggravation of an unrelated condition by the physiologic effects of pregnancy.

Stillbirth	Fetal death that occurs ≥ 20 weeks' gestation

[a] There are several definitions of perinatal death rate in use today, including neonatal deaths within 7 days or within 28 days after birth; others use fetal deaths occurring with a gestation of more than 28 weeks.

Based on World Health Organization. Health statistics: maternal mortality ratio. Available at: http://www.who.int/healthinfo/statistics/indmaternalmortality/en/. Accessed December 18, 2017[1]; World Health Organization. *ICD-10 International Statistical Classification of Diseases and Related Health Problems.* Geneva, Switzerland: World Health Organization; 2011[2]; Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. November 9, 2017. Available at: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html. Accessed January 19, 2018.[3]

Table 3A-2 Pregnancy, Fetal, and Infant Death Rates in the United States by Race/Ethnicity

Race/Ethnicity	Infant Mortality Rate: *n*/1000 Live Births[a]	Fetal Mortality Rate: *n*/1000 Live Births and Fetal Deaths[b]	Pregnancy-Related Mortality Ratio[c]: *n*/100,000 Live Births
Non-Hispanic black	11.37	10.53	43.5
American Indian/Native American	8.3	6.2	14.4[d]
Hispanic	5.0	5.22	14.4[d]
Non-Hispanic white	4.9	4.88	12.7
Asian/Pacific Islander	4.2	4.68	14.4[d]
Total	6	5.96	17.3

[a] 2014.

[b] 2013.

[c] 2011–2013.

[d] The pregnancy-related mortality ratio is aggregated for all races excluding non-Hispanic black and non-Hispanic white.

Based on Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. November 9, 2017. Available at: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html. Accessed January 19, 2018[3]; Centers for Disease Control and Prevention. Infant health: protecting our next generation at a glance 2016. Available at: https://www.cdc.gov/chronicdisease/resources/publications/aag/infant-health.htm. Accessed January 20, 2018[4]; MacDorman MF, Gregory EC. Fetal and perinatal mortality: United States, 2013. *Natl Vital Stat Rep.* 2015;64(8):1-10.[5]

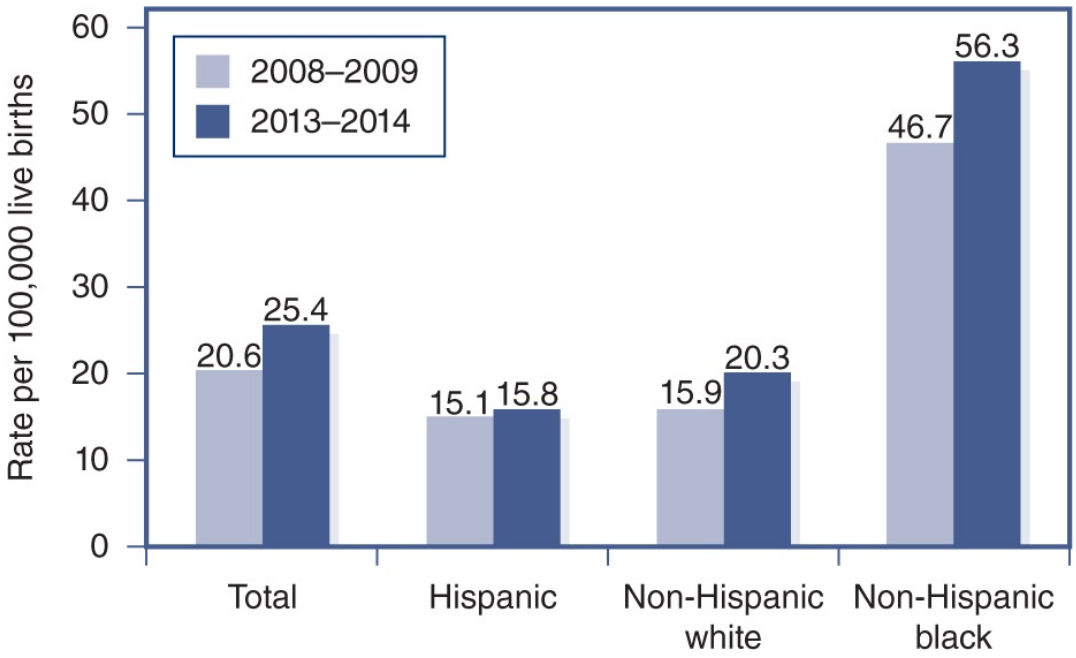


Figure 3A-1 Maternal mortality rates by race and ethnicity, 27 states and Washington, DC, 2008–2009 and 2013–2014.

Reproduced with permission from MacDorman MF, Declercq E, Thomas ME. Trends in maternal mortality by sociodemographic characteristics and cause of death in 27 states and the District of Columbia. *Obstet Gynecol*. 2017;129(5):811-818.[6]

Infant Mortality

In 2014, the overall infant mortality rate (IMR) in the United States was 6 infant deaths per 1000 live births.[4] However, the IMR varies by geographic location and race/ethnicity and is highest among non-Hispanic blacks (Table 3A-2).[3-5,7] The most common causes of infant death are congenital anomalies, preterm birth or low birth weight, maternal complications of pregnancy, sudden infant death syndrome (SIDS), and unintentional injuries.

The IMR has historically been used as an indicator of the health of a population because the health of infants in the first year of life is heavily dependent on factors such as maternal health, quality of health care, socioeconomic conditions (e.g., nutrition, education, wealth), and public health practices (e.g., sanitation, preventive health services).[8] Despite spending larger amounts of money on healthcare services than many other countries, the United States ranks 26th among developed nations, with an IMR that is approximately twice as high as most other developed nations.

The reasons that the IMR in the United States remains high despite higher levels of healthcare spending are complex and not fully understood. International comparisons have found that the higher IMR can be largely attributed to increased rates of preterm birth and higher death rates in infants born after 37 weeks' gestation.[8]

The most modifiable etiology is likely preterm birth.[5] Many studies have explored strategies designed to decrease the risk of preterm birth, including investigation of environmental

epigenetics, suggesting that improving social determinants of health can directly affect perinatal outcomes.[9] Methods for lowering the rate of preterm birth include interventions such as increasing breastfeeding rates, offering more parental support and education, and improving access to healthcare services.

Fetal Death and Stillbirth

States have different reporting requirements for fetal death. Most report fetal deaths that are 20 weeks' gestation or greater and/or 350 grams or greater birth weight, and use 20 weeks' gestation as the gestational age threshold for distinguishing a stillbirth from the product of a miscarriage. A few states report all pregnancy losses as a fetal death regardless of the period of gestation. The fetal death rate in the United States is approximately the same as the IMR, at 5.96 fetal deaths after 20 weeks' gestation per 1000 live births and fetal deaths combined (denominator). The incidence varies by race/ethnicity and is highest in non-Hispanic blacks.

Maternal Mortality

Maternal deaths are evaluated by three different systems in the United States.[10] The National Vital Statistics System reports the maternal mortality rate and ratio based on death certificates using the World Health Organization's International Classification of Disease codes. These statistics do not generate detailed data about the proximate cause of death. The Pregnancy Mortality Surveillance System within the Centers for Disease Control and Prevention (CDC) monitors pregnancy-related and pregnancy-associated deaths via a more complex analysis of death certificates, birth certificates, and pregnancy data that are recorded on both. These statistics generate more detailed data about the causes of death. The third source of information comes from maternal mortality review committees that analyze pregnancy-related deaths in more detail at the local level.

Maternal mortality is much less common than infant or fetal death and is reported as a number per 100,000 live births. A single woman dying in childbirth is a tragedy; an increase in the number of women dying is a societal catastrophe and national embarrassment. Despite advances in standard of living and healthcare technology, the number of women who die from a pregnancy-related cause has increased in the United States from 7.2 deaths per 100,000 live births in 1987 to a high of 17.3 deaths per 100,000 live births in 2013.[3] The maternal mortality rate in non-Hispanic black women is three times higher than that in non-Hispanic white women (40.2 versus 14.1 maternal deaths per 100,000 live births), which is one of the most striking perinatal health disparities in the United States today.[11] Today the United States ranks 60th in the world, with a maternal mortality rate that is higher than that of all other developed nations.[12] It is estimated that approximately 50% of the U.S. maternal deaths are preventable.[13]

Historically, the most common causes of maternal mortality were hemorrhage, infection, hypertension, and anesthesia complications. As the prepregnancy health status of childbearing women has changed over time, so have the causes of mortality and morbidity. The most common direct causes of pregnancy-related death during 2011–2013 are listed in **Table 3A-3**.[3]

Receiving care at an appropriate site may ameliorate risks for an individual, but problems of poverty, access, and insurance can result in women not obtaining the care needed. Specific factors that are associated with an increased maternal mortality ratio include four or fewer prenatal visits, unintended births, cesarean births, unmarried status, and percentage of black women giving birth within a state.[11] The last factor illustrates a continuing challenge associated with racial and ethnic health disparities in the United States.

Table 3A-3	Leading Causes of Maternal Death in the United States, 2011–2013
Cause	**Percentage of Pregnancy-Related Deaths**
Cardiovascular diseases	15.5%
Noncardiovascular diseases (includes drug overdose)	14.5%
Infection or sepsis	12.7%
Hemorrhage	11.4%
Cardiomyopathy	11.0%
Thrombotic pulmonary embolism	9.2%
Hypertensive disorders of pregnancy	7.4%
Cerebrovascular accidents	6.6%
Amniotic fluid embolism	5.5%
Anesthesia complications	0.1%

Based on Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. November 9, 2017. Available at: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html. Accessed January 19, 2018.[3]

The Rising Trend of Maternal Mortality in the United States

Multiple studies have attempted to identify the reasons for the rising incidence of maternal death in the United States. Initially, the increase was deemed temporary and associated with changes in birth certificates, categorization of causes of death, or other record keeping issues.[14,15] Nevertheless, the data collection factors do not explain changes in specific states. For example, the MMR doubled within a 2-year period in one state; such a rise was unexpected in the absence of war, natural disaster, or severe economic disaster.[15] This state and others have now initiated maternal mortality review boards, frequently including midwives as board members, to directly examine records of women who have died. Such groups are not universally available across the country, however.[13]

Prominent among the potential etiologies of pregnancy-related deaths is the fact that care of pregnant women varies dramatically across the country. In many developed countries, best practices are shared and followed regardless of birth site. In the United States, there is no standard approach to treatments for complications of pregnancy or management of women experiencing an emergency in childbirth. This problem is currently the focus of many policy initiatives. For example, the California Maternal Quality Care Collaboration developed

"bundles" or standard practice protocols that were disseminated throughout the state on topics such as postpartum hemorrhage.[16] Studies found that adoption of such bundles was associated with decreases in maternal deaths, prompting the design of the National Partnership for Maternal Safety's bundles, which include one on managing obstetric hemorrhage.[13,17] These practice guidelines are a good example of how statistics drives research that first affects policy and then is translated into clinical practice.

Maternal Morbidity

Maternal morbidity is much more common than maternal mortality. As difficult as it is to obtain accurate maternal mortality data, however, morbidity data collection is even more elusive. Mortality data measure a single event or incidence. Morbidity data, by comparison, are much less likely to be recorded, suffer from a lack of standard case definitions, and frequently involve several comorbidities (e.g., an individual with multiple conditions such as severe anemia, vesico-vaginal fistula, repeated urinary tract infections, and clinical depression).

Near-miss maternal mortality is a life-threatening event that could result in death. Recent efforts to define and track near-misses have been proliferating as a method to further assess the quality of health care and the effectiveness of interventions.[18] Early warning criteria are being instituted as part of patient safety bundles in an effort to decrease the incidence of maternal morbidity.[18]

Reproductive Health: The Global Picture

Although pregnancy-related deaths in the United States are a critical concern nationally, approximately 99% of maternal deaths occur in low-resource nations, as shown in **Table 3A-4**.[19] It is difficult to compare maternal mortality ratios across nations because of inconsistency in definitions and reporting. On a worldwide basis, many women never enter the healthcare system during pregnancy, even when gravely ill, and, therefore, deaths and disabilities are not well captured in vital statistics, surveillance data, or other records. Because the maternal mortality rate is difficult to obtain, the *ratio* is used more frequently. When comparing countries, the maternal mortality ratio is the number of maternal deaths per 100,000 live births in a given period of time.

Table 3A-4 **Estimates of Maternal Mortality Ratio, Number of Maternal Deaths, and Lifetime Risk by United Nations Millennium Development Goal Region, 2013**

Region	MMR: *n*/100,000 Live Births	Range of MMR Uncertainty		Number of Maternal Deaths[a]	Lifetime Risk of Maternal Death: 1/*n*
		Lower Estimate	Upper Estimate		
World	210	160	290	289,000	190
Developed regions	16	12	23	2300	3700
Developing regions	230	180	320	286,000	160

Abbreviation: MMR, maternal mortality ratio.

[a] The MMR, number of maternal deaths, and lifetime risk have been rounded according to the following scheme: < 100, no rounding; 100–999, rounded to nearest 10; 1000–9999, rounded to nearest 100; and > 10,000, rounded to nearest 1000.

Data from World Health Organization, United Nations Children's Fund, United Nations Population Fund, The World Bank, United Nations Population Division. Trends in maternal mortality: 1990 to 2013. Available at: http://www.who.int/reproductivehealth/publications/monitoring/maternal-mortality-2013/en/. Accessed December 12, 2017.[19]

Global Factors Associated with Maternal Mortality

The most common causes of maternal mortality in low-resource nations are hemorrhage, infection, hypertension, complications during birth, and unsafe abortion.[19] Betrán et al. reported findings of a systematic review of maternal mortality in 141 countries in 2005.[20] Using standard regression models, these authors found that (1) the proportion of births assisted by a skilled attendant, (2) the infant mortality rate, and (3) national per capita expenditures on health were three factors strongly related to maternal mortality worldwide.[20]

In addition, a phenomenon known as the "three delays" has been identified as a critical determinant of maternal mortality. Once an emergency occurs in childbirth, its cause can be analyzed within the following framework, whose elements collectively prevent timely, high-quality care from reaching those most in need:

1. Delay in recognizing there is a problem and making the decision to seek care
2. Delay in reaching the appropriate level of care once the problem or complication has been recognized
3. Delay in receiving the appropriate care after arrival at the service site

The Safe Motherhood Initiative

In an effort to address some of the global factors involved in maternal and infant mortality, the Safe Motherhood Initiative held its first meeting in 1987 in Africa.[21] Another group that was organized a few years later but has similar goals is the White Ribbon Alliance. The White Ribbon Alliance includes many of the same members as the Safe Motherhood group, but is more broad based and includes international nongovernmental organizations (INGOs), government agencies, local nongovernmental organizations (NGOs), and community-based organizations in resource-limited countries. The general goals of the alliance members are to promote practices, protocols, and guidelines that facilitate women obtaining high-quality gynecologic, family planning, prenatal, delivery, and postpartum care, so as to achieve optimal

health for the mother, fetus, and infant during the perinatal period. Although the emphasis has been on low-resource countries, the action messages posted on the Safe Motherhood website can be of value to all countries, including the United States (**Table 3A-5**).[22]

Table 3A-5	Safe Motherhood Action Messages
Advance safe motherhood through human rights	
Empower women, ensure choices	
Safe motherhood is a vital economic and social investment	
Delay marriage and first birth	
Every pregnancy faces risks	
Ensure skilled attendance at delivery	
Improve access to quality reproductive health services	
Prevent unwanted pregnancy and address unsafe abortion	

Reproduced with permission from Safe Motherhood action messages. Available at: http://www.safemotherhood.org. Accessed December 12, 2017.[22]

Conclusion

There are multiple complexities involved in the definition, collection, and reporting of vital statistics that make it difficult to rely on exact numbers. Nonetheless, trends in these numbers tell a powerful story. The United States spends more on health care than any other nation in world, and the IMR has decreased in recent years. Yet, the IMR in the United States remains higher than that in many other developed nations, maternal mortality and morbidity are increasing, and racial/ethnic disparities in maternal–child health are striking.

The link between national vital statistics and clinical care of individuals may seem remote but, in fact, these statistics play an important role in each clinical encounter. The underlying etiologies of morbidity and mortality are both social and biologic in nature and, in turn, midwifery care of each woman must address social determinants of health as well as biologic indicators. Midwifery has a long and storied history of providing such care internationally, and today's midwives are charged with continuing that care.

Resources

Organization	Description	Webpage
Cornell University Library	"Finding Health Data and Statistics: Reproductive and Family Health."	http://guides.library.cornell.edu/c.php?g=241897&p=1608216
Lancet Series on Midwifery, June 23, 2014	Special issue of *Lancet* that focuses on the needs of childbearing families internationally and the role of midwives.	http://www.thelancet.com/series/midwifery

References

1. World Health Organization. Health statistics: maternal mortality ratio. Available at: http://www.who.int/healthinfo/statistics/indmaternalmortality/en/. Accessed December 18, 2017.
2. World Health Organization. *ICD-10 International Statistical Classification of Diseases and Related Health Problems*. Geneva, Switzerland: World Health Organization; 2011.
3. Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. November 9, 2017. Available at: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html. Accessed January 19, 2018.
4. Centers for Disease Control and Prevention. Infant health: protecting our next generation at a glance 2016. Available at: https://www.cdc.gov/chronicdisease/resources/publications/aag/infant-health.htm. Accessed January 20, 2018.
5. MacDorman MF, Gregory EC. Fetal and perinatal mortality: United States, 2013. *Natl Vital Stat Rep*. 2015;64(8):1.
6. MacDorman MF, Declercq E, Thomas ME. Trends in maternal mortality by sociodemographic characteristics and cause of death in 27 states and the District of Columbia. *Obstet Gynecol*. 2017;129(5):811-818.
7. MacDorman MF. Race and ethnic disparities in fetal mortality, preterm birth, and infant mortality in the United States: an overview. *Semin Perinatol*. 2011;35(4):200-208.
8. MacDorman MF, Mathews TJ, Mophangoo AD, Zeitlin J. International comparisons of infant mortality and related factors: United States and Europe, 2010. *Nat Vital Stat Rep*. 2014;63(5):1-10.
9. Vick AD, Burris HH. Epigenetics and health disparities. *Curr Epidemiol Rep*. 2017;4(1):31-37.
10. St Pierre A, Zaharatos J, Goodman D, Callaghan WM. Challenges and opportunities in identifying, reviewing, and preventing maternal deaths. *Obstet Gynecol*. 2018;131:138-142.
11. Moaddab A, Dildy GA, Brown HL, et al. Health care disparity and state-specific pregnancy-related mortality in the United States, 2005–2014. *Obstet Gynecol*. 2016;128:869-875.
12. Neggers YH. Trends in maternal mortality in the United States. *Reprod Toxicol*. 2016;64:72-76.
13. Main EK, McCain CL, Morton CH, Holtby S, Lawton ES. Pregnancy-related mortality in California: causes, characteristics, and improvement opportunities. *Obstet Gynecol*. 2015;125:938-947.
14. Joseph KS, Lisonkova S, Muraca GM, et al. Factors underlying the temporal increase in maternal mortality in the United States. *Obstet Gynecol*. 2017;129(1):91-100.
15. MacDorman MF, Declercq E, Cabral H, Morton C. Recent increases in the U.S. maternal mortality rate: disentangling trends from measurement issues. *Obstet Gynecol*. 2016;128(3):447-455.
16. Main EK, Goffman D, Scavone BM, et al. National Partnership for Maternal Safety consensus bundle on obstetric hemorrhage. *J Midwifery Womens Health* 2015;60:458-464.
17. Creanga AA, Syverson C, Seed K, Callaghan WM. Pregnancy-related mortality in the United States, 2011–2013. *Obstet Gynecol*. 2017;130(2):366-373.
18. Mhyre JM, D'Oria R, Hameed AB, et al. The maternal early warning criteria: a proposal from the National Partnership for Maternal Safety. *Obstet Gynecol*. 2014;124:782-786.
19. World Health Organization, United Nations Children's Fund, United Nations Population Fund, The World Bank, United Nations Population Division. Trends in maternal mortality: 1990 to 2013. Available at: http://www.who.int/reproductivehealth/publications/monitoring/maternal-mortality-2013/en/. Accessed December 12, 2017.
20. Betrán AP, Wojdyla D, Posner SF, Gülmezoglu AM. National estimates for maternal mortality: an analysis based on the WHO systematic review of maternal mortality and morbidity. *BMC Public Health*. 2005;12(5):131-143.
21. Islam M. The Safe Motherhood Initiative and beyond. *Bull WHO*. 2007;85(10):735.
22. Safe Motherhood action messages. Available at: http://www.safemotherhood.org. Accessed December 12, 2017.

PART II

Midwives as Primary Care Providers

LISA L. PAINE

The midwife's place in primary care for women and newborns is now well established, bringing with it an awe-inspiring responsibility to do our part to improve the lifelong health and well-being of women, their families, and their communities. Despite state-by-state variations in legislative and regulatory support for this aspect of the midwife's role, as well as irregularities in reimbursement for primary care services, midwives have embraced their role and the responsibilities that come with it.[1]

To some, the primary care provider role for midwives in the United States might appear to be a new phenomenon, but its roots harken back decades, if not centuries. This nation's first midwives were esteemed healers in their communities, and the first nurse-midwives were public health nurses, all providing elements of primary care as we know it today. Their scope of practice included general health and well-being for the whole family.[2] By the early 1970s, content had been added to midwifery curricula regarding family planning, ushering in a role beyond the maternity cycle to include well-woman care before and after pregnancy.[3] By the 1990s, competencies in primary care had been added to midwifery education and practice standards and were made even clearer in subsequent core competency revisions.[4,5]

In 1992, the American College of Nurse-Midwives (ACNM) issued its first formal statement declaring that certified nurse-midwives (CNMs) provide primary care for women and newborns.[6] This statement coincided with publication of results of a landmark prospective study than showed that 7 of 10 visits made to CNMs were by women or infants vulnerable to poor access or disparate outcomes by virtue of their race, ethnicity, age, education, income, immigration status, or place of residence.[7] By the end of the twentieth century, it had become clear that midwives can increase access to primary care services when they are integral members of the primary care workforce.

The U.S. healthcare system remains in flux, despite decades-long efforts to strengthen the

system, including passage of significant legislation in the past decade. The federal government's recognition of the midwife's primary care role is evidenced by primary care workforce expansion efforts that include Medicaid/Medicare program payment for midwifery services, federal funding for education programs that prepare midwives as primary care providers, and federal employee insurance coverage for midwifery services. More recently, however, threats to repeal legislation that has extended health insurance coverage to millions of Americans, as well as state variations in expansion of Medicaid coverage, mean that midwives will be providing primary care in an unsettled environment for years to come.[8] What remains clear, however, is that well-prepared midwives will be needed to meet the nation's substantial primary healthcare needs over time.

Current core competencies for basic midwifery practice specify the primary care content for which midwives are responsible.[4] In 2012, ACNM issued a policy statement that reaffirmed the role of midwives in primary care. This statement also affirmed midwives' roles within the patient-centered medical home, a relatively new model that incorporates improved quality of care strategies, promotes communication, and enhances efficient delivery of care in a multidisciplinary model—with the patient at the center of the partnership.[5]

Midwifery care is focused on normalcy, and the *Introduction to the Care of Women*, *Health Promotion Across the Lifespan*, *Common Conditions in Primary Care*, *Nutrition*, *Mental Health Conditions*, and *Pharmacotherapeutics* chapters in this text present the knowledge needed to help all women of all ages maintain and promote healthy lifestyles. Yet the women who receive primary care from midwives experience a range of inequities that influence their health and well-being and result in health disparities. This reality compels midwives to have an understanding of the social and economic determinants of health, and to explore the primary care role for women with complex social situations, as well as comorbid diseases, mental health conditions, and substance use disorders.[10,11]

The demographic characteristics of the U.S. population are changing, with glaring disparities in health outcomes experienced by women and children of color becoming painfully apparent. Thus attention to racial and ethnic diversity is a priority. Yet other aspects of diversity within populations cared for by midwives are also evident, such that some populations have well-documented inequalities in access and health disparities based on sociodemographic factors: young or old age, low education and low literacy, low income, religious affiliation, disability status, sexual minority status, and place of residence (especially remote, rural, or urban areas). The intersectionality of these population characteristics can further magnify health problems, as in the case of race/ethnic disparities among rural residents.[12]

To bring about equity in terms of healthcare access, outcomes, and quality, midwives must become culturally proficient care providers and, when possible, emerge as leaders in this area.[13] To do so, midwives must embark on a personal and professional journey. Cultivation of cultural humility is an essential first step in the development of the cultural competence skills needed for the practice of effective primary care.[14] Midwives must commit to the practice of cultural humility—a lifelong process of self-critique, self-awareness, and reflection. By examining their own worldview, rather than focusing solely on the client's belief system and

culturally specific traits, the midwife will appreciate those intrinsic qualities that enable individuals to attain health, happiness, and wholeness. With this understanding, the midwife can adapt primary care practice to be more patient centered and, in turn, more effective.

Regardless of their particular practice setting, midwives are certain to encounter women who experience health inequalities in any number of ways, including poor access to primary care and mental health care. Nearly half of all women in the United States live in an area with a shortage of primary care providers.[15] By joining their forebearers who practiced primary care midwifery for disenfranchised women and families with poor or no access to even the most basic health care, midwives today who embrace the primary care role and commit to advancing cultural proficiency can and will make lasting improvements in the health of the entire nation, especially when their work is combined with organized efforts to diversify the midwifery workforce to better serve the needs of all women.[13]

With these imperatives in mind, the chapters in this section present an overview of the common health conditions encountered in primary care. Readers are especially directed to the *Health Promotion Across the Lifespan* chapter, which summarizes current recommendations for primary and secondary prevention—these topics are integral to midwifery practice. It is abundantly clear that more uniform regulatory and practice environments are needed to maximize midwives' contributions to prevention and primary care. Likewise, midwives must be embraced as primary care providers within their practice settings and by the policymakers responsible for ensuring the health of the nation's people. Meanwhile, midwives can and will do their part by becoming fully prepared for the contemporary version of their historical role in primary care, thereby ensuring that the nation's women, families, and communities will be healthier in the future.

References

1. Phillippi JC, Barger MK. Midwives as primary care providers for women. *J Midwifery Womens Heath*. 2015;60:250-257.
2. Varney H, Thompson JB. *A History of Midwifery in the United States: The Midwife Said Fear Not*. New York, NY: Springer; 2016.
3. Rooks JP. *Midwifery and Childbirth in America*. Philadelphia, PA: Temple University Press; 1997.
4. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; December 2012.
5. Phillippi JC, Avery M. The 2012 American College of Nurse-Midwives core competencies for basic midwifery practice: history and revision. *J Midwifery Womens Heath*. 2014;59:82-90.
6. American College of Nurse-Midwives. *Position Statement: Certified Nurse-Midwives and Certified Midwifes as Primary Care Providers/Case Managers*. Washington, DC: American College of Nurse-Midwives; 1992; revised 1994, 1997.
7. Paine LL, Lang JM, Strobino DM, et al. Characteristics of nurse-midwife patients and visits, 1991. *Am J Pub Health*. 1999;89(6):906-909.
8. Antonisse L, Garfield R, Rudowitz R, Artiga S. *Issue Brief: The Effects of Medicaid Expansion under the ACA: Updated Findings from a Literature Review*. Washington, DC: Henry J. Kaiser Family Foundation; September 2017. Available at: http://files.kff.org/attachment/Issue-Brief-The-Effects-of-Medicaid-Expansion-Under-the-ACA-Updated-Findings-from-a-Literature-Review. Accessed December 8, 2017.
9. American College of Nurse-Midwives. *Position Statement: Midwives Are Primary Care Providers and Leaders of Maternity Care Homes: Certified Nurse-Midwives and Certified Midwives as Primary Care Providers/Case Managers*. Silver Spring, MD: American College of Nurse-Midwives; June 2012.
10. Goodman D. Improving access to maternity care for women with opioid use disorders: colocation of midwifery services at an addiction treatment program. *J Midwifery Womens Heath*. 2015;60:706-712.

11. Womack JA, Brandt CA, Justice AC. Primary care of women aging with HIV. *J Midwifery Womens Heath*. 2015;60:146-157.
12. James CV, Moonesinghe R, Wilson-Frederick SM, Hall JE, Penman-Aguilar A, Bouye K. Racial/ethnicity health disparities among rural adults—United States, 2012–2015. *MMWR Surveill Summ.* 2017;66(SS-#23):1-9. http://dx.doi.org/10.15585/mmwr.ss6623a1.
13. Dau KQ. Organizational change in the pursuit of equity. *J Midwifery Womens Health*. 2016;61:685-687.
14. Tervalon M, Murray-Garcia J. Cultural humility versus cultural competence: a critical distinction in defining physician training outcomes in multicultural education. *J Heath Care Poor Underserved*. 1998;9(2):117-125.
15. James CV, Salginicoff A, Thomas M, Ranji U, Lillie-Blanton, Wyn R. *Putting Women's Health Care Disparities on the Map: Examining Racial and Ethnic Disparities at the State Level*. Menlo Park, CA: Kaiser Family Foundation; 2009:1-104.

CHAPTER

4

Introduction to the Care of Women

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Midwife Means “With Woman”

Midwifery is both art and science—and above all midwifery is care for women and their families. As discussed in the first chapter of this text, midwifery in the United States has grown from local efforts to care for women with few resources into a well-respected profession that offers women primary care and gynecologic services, as well as maternity, birth, and newborn care. These services are provided using an interdisciplinary approach because midwives rely on the skills of gynecologists and obstetricians, nurses, social workers, and other health professionals, just as those professionals rely on midwives for their respective expertise. Key to understanding midwifery care is recognition of the interweaving of skills and knowledge from many sources, and the willingness to work with others to achieve the best possible health outcomes for a woman.

The evidence-based studies and expert opinions that underpin the science of midwifery are the same as those from which medicine and nursing draw their understanding of health care. Unfortunately, relatively few of the expert recommendations on which clinicians rely are drawn from high-quality research. In some situations, interventions are adopted before large research studies are conducted. In brief, most clinicians practice similarly, and practice styles are often based on the preferences and experience of those who have taught them.

The flawed perception that guidelines are consistently based on solid evidence and lack bias has been acknowledged.[1] For example, in 2011 the American College of Obstetricians and Gynecologists published the finding that fewer than one-third of its Practice Bulletins were based on “good and consistent” evidence.[2] Additionally, much of pregnancy care cannot be evaluated safely and ethically using the most rigorous types of research. Midwifery research continues to account for only a small percentage of the work being conducted in the area of women’s health, although an increasing number of scholars are contributing knowledge in this area. Examples range from the development of Centering Pregnancy as a model of care to the research done on delayed cord-clamping after birth.[3,4] Deciphering the evidence, acknowledging the quality of information from which recommendations are made, and recognizing biases—both in midwives and in others—are all key components of midwifery care.

Midwifery is distinguished by characteristics that define a partnership with women. A willingness to listen; sensitivity to cultural, sexual, and generational differences; shared decision making; the patience to be “with women”—all combine with professional behaviors to describe midwifery practice. This chapter addresses both clinical tasks and professional behaviors, and its goal is to identify those core skills needed to be a midwife.

Essential skills begin with an understanding of the midwifery management process. Developed by midwifery education program in Mississippi in the early1970s,[5] the seven steps in this process serve as a guide to the process of care at an individual level and offer an opportunity to evaluate the effectiveness of care (**Table 4-1**).

Table 4-1	The Midwifery Management Process

1. Investigate by obtaining all necessary data for complete evaluation of the woman or newborn.
2. Accurately identify problems or diagnoses and healthcare needs based on correct interpretation of the data.
3. Anticipate other potential problems or diagnoses that might be expected because of the identified problems or diagnoses.
4. Evaluate the need for immediate midwife or physician intervention and/or for consultation or collaborative management with other healthcare team members, as dictated by the condition of the woman or newborn.
5. Develop a comprehensive plan of care that is supported by explanations of the valid rationale underlying the decisions made and is based on the preceding steps.
6. Assume responsibility for the efficient and safe implementation of the plan of care.
7. Evaluate the effectiveness of the care given, recycling appropriately through the management process for any aspect of care that has been ineffective.

The midwifery management process emphasizes the midwife's responsibility as an independent care provider and is based on the scientific process. Even while other practitioners may be involved in care, the midwife assumes responsibility for ensuring the woman and her family receive adequate and prompt treatment and health education.

The comprehensive plan noted in the management process focuses first on evidence-based treatment options that promote health and prevent morbidity and/or treat current conditions. These interventions encompass more than simple pharmaceuticals—they also comprise a plan of care customized to meet the woman's needs. Considerations in the design of such a plan include cost, accessibility, and convenience among other factors.

The midwifery management process also acknowledges that most care is provided not in discrete sessions, but rather over time. Optimally, continuity over time is critical in improving quality of care.

Communication

Clear communication is an essential component of any therapeutic interaction and deserves conscious attention if it is to be effective. Several important variables influence the success of communication between a midwife and a woman who seeks healthcare services.

Many characteristics that contribute to a person's identity play an important role in the relationships between women and their midwives. Cultural identity, sex and gender identity, race and ethnicity, immigration status, and socioeconomic status are just a few examples of the factors that influence a person's interactions with others. Health literacy and health numeracy impact how well an individual understands medical communications. In addition, language itself can facilitate or be a barrier to comprehension.

The Role of Culture, Race, Ethnicity, and Identity

Midwives will, over time, meet individuals who are hesitant to share their health concerns because they have learned to distrust how their beliefs and identity may be received. What does this mean for the midwife in practice? Awareness of one's own cultural assumptions, as well as appreciation of one's personal biases and choices about practice parameters, is an essential first step. Unless midwives know themselves, they are at increased risk of unconscious offenses and implicit bias that may hinder the therapeutic relationship, increase reluctance to share important information, or prevent a woman from seeking care. This is a lifelong project. Few, if any, individuals come into adulthood fully sensitive to these topics.

It is important to remember that health care is a cultural construct as well—emerging from beliefs about the characteristics of disease and the impact on the human body. Consequently, culture is a primary factor in the provision of health care that influences both the provider and the receiver of care. This idea extends not only to the care provider, but also to structural and process aspects of care. DeMeester and colleagues describe components of the healthcare system that can improve shared decision making for persons from at-risk communities, focusing on dual-minority individuals (e.g., lesbian and African American). These components include reflecting the community in office workflow, health information technology, organizational culture and structure, the physical environment, resources, staff training, and incentives for positive results.[6] In this light, it is important to note that throughout this book the term "women" is used and may on occasion include reference to individuals who do not self-identify as a "woman." It should be understood that individuals may have a sexual and/or gender identity that is different than the biologic sex at birth, as discussed in more detail in the *Midwifery: Clients, Context, and Care* chapter. Thus, the reader is encouraged to apply the information in this chapter on physical examination as is pertinent for the individual patient.

Addressing Sensitive Concerns

Midwives deal with some of the most intimate and personal aspects of a woman's life. Her choice of partner, her decisions about childbearing, and her sexual experiences constitute just a few of the themes that impact the woman's life. Her risk for being exposed to violence,

whether physically, sexually, or emotionally, is another theme. The choices she has made with regard to lifestyle or habits, exposure to infections, and whether she has basic necessities such as adequate food and shelter are all topics that can and will come up during midwifery care.

Women may experience stigma for many reasons. When a person has been exposed to negative reactions in the community enough times, that individual tends to withdraw from creating opportunities to be stigmatized. It is the midwife's job to create a safe environment, where questions can be asked and answered, and where help can be sought and offered. When greeting a woman or family in the office, when providing care, and when discussing choices, watch for physical or verbal cues that suggest that another topic needs to be addressed.

Use of Language

Learning to listen to one's own words and see one's physical position relative to others is a skill like any other. Both speech and body language affect the relationship between the midwife and the woman. Among other things, this means that word choice needs to be appropriate for the woman's educational and cultural background. The midwife has to move to where the woman is in terms of understanding; by beginning there, both can move together to identify and discuss a problem. The language that any healthcare provider spends years mastering is not the common tongue.

Active listening is an essential skill. It requires the patience to allow a woman to tell her own story where she wishes to start it, with minimal interruptions or directive language. Asking open-ended questions encourages a woman to put into her own words her concerns. When one listens actively, one focuses on what is being said, reflects back what one hears, and verifies that both participants in the conversation share understanding. Waiting silently to encourage additional information; paraphrasing an unclear statement by asking, "I think you mean ____. Is that correct?"; reflecting back, such as by saying, "I hear you saying _____."; and providing reassurance that the woman's information is important are all critical tools. Validating understanding of a problem described by a woman is essential—it is all too easy to misunderstand her actual concern. Further, the woman needs to believe that what she says will be held in confidence, and that she may safely say anything she needs to say. Active listening also incorporates a nonverbal component, including making eye contact, leaning toward a person rather than away from her, having an open body position that suggests acceptance, avoiding closed positioning (e.g., not crossing the arms), and maintaining a professional facial expression.

Another instrument for listening to oneself is to consider how communication in a professional setting affects the woman's ability to hear what is being said. The midwife's words should answer the following four questions. These questions are based on similar concepts that have traditionally been attributed to various individuals and groups, such as the mystical Sufi tradition, Socrates, and more recently, the Quakers. Although their origins may be murky, the concepts remain relevant today. The questions are appropriate when considering how communication in a professional setting affects the woman's ability to hear what is being said. The midwife's words should answer the following four questions:

1. *Is it truthful?* If not, there is no need to say it.
2. *Is it kind?* Many of the topics that need to be discussed require kindness to make it possible for the listener to accept unpalatable advice. Kindness is not ignoring a problem, nor is it patronizing the woman. For example, there are many ways to tell a woman she weighs too much and needs to lose weight. Compare "You're obese and you need to eat less" with "I'm concerned that your weight is harming your health." Which of these statements will be more likely to encourage the woman to accept a referral to a nutritionist?
3. *Is it necessary?* Preventing damage from diabetes or hypertension in this example may hinge on the woman accepting the referral and actively participating in improving her health. Do not ask a question unless the answer changes the woman's management plan, and always be able to explain to the woman why the question is needed.
4. *Is it appropriate?* In one sense, discussing weight management is always appropriate —but what if the woman has come to be treated for a sexually transmitted infection (STI)? Perhaps weight loss is not the concern she needs to focus on today.

Health Literacy and Health Numeracy

The midwife should address the woman in language she can understand, with explanations that are clear and do not omit factual information that is important. Health literacy is defined by the Institute of Medicine as "the degree to which individuals have the capacity to obtain, process, and understand basic health information and services needed to make appropriate health decisions."[7] Approximately half of the U.S. adult population has low health literacy.[8] Low health literacy is associated with more hospitalizations and lower use of preventive services such as mammography and vaccinations. Although educational attainment frequently is used as a proxy for health literacy, many individuals actually read and, more importantly, comprehend at levels below their formal educational level. Research is being conducted into the use in this electronic age of graphics, apps, and digital presentations for individuals who are not highly literate.[9,10]

Health numeracy, which is the degree to which individuals understand quantitative and probabilistic health information, is an important component of health literacy.[11] Strategies to increase the likelihood that a woman will understand the probability of an event include using absolute numbers instead of percentages, relative risks, or risk ratios;[12] avoiding the words "rare," "unlikely," "uncommon," and "unusual," as these are imprecise concepts subject to individual interpretation; and using small denominators and whole numbers.[13] For example, "1 in 4" is more easily understood than "25 out of 100" or "25%." Studies of health literacy and health numeracy often portray these abilities as individual patient skills, but effective communication by the midwife is an equally important factor in ensuring understanding.[14,15] Resources for learning more about health literacy and health numeracy are included in the Resources section at the end of this chapter.

Approaching the Woman

The first steps in any clinical encounter usually occur before the midwife and a woman meet. Somehow, the woman has found the practice, made an appointment, been checked in for her visit, and possibly been seen by a nurse or medical assistant. She has observed whether the setting is professional and the furnishings are in good repair. Although it is not the purpose of this chapter to discuss specific practice management, all of these events will have shaped the woman's first impressions of her midwife and of midwifery. Seemingly simple choices about work flow, such as whether the woman is seen first in an office or undressed on an examination table, say something about mutual respect. Just as when making a home visit, the midwife looks for clues about the woman's lifestyle and health of her family; likewise, the woman seeks clues about the midwife's practice and professionalism.

Before asking any questions, however, mutual introductions should take place. Think of the relationship forged during this encounter as a framework for the care that the woman will receive. The woman should be seated comfortably and with adequate personal space. The midwife's position should promote direct eye contact. Asking, "How are you today?" or "How are you feeling?" establishes concern for the woman as an individual. Listening to her answer assists the establishment of mutual trust and trust in midwifery care.

From a legal compliance or payment perspective, the order of the visit's elements encapsulates the essential components of care. While in this context, *compliance* refers to legal requirements (such as Medicare regulations and not the woman's agreement with the management plan) and *payment* refers to what is required to receive compensation from insurers; both incorporate standards that must be met.

Some visits will be tightly problem focused; some will be comprehensive examinations. Both types of visits follow the same sequence. All begin with the same question: "Why have you come in to see us today?" The answer establishes the chief concern of the woman—that is, the reason for the visit. The terms "concern" and "reason for coming" are less value laden than the older phrase in documentation of "chief complaint." Sometimes other, more pressing problems will become apparent during the conversation or examination, but asking about the woman's initial reason for the visit is always the starting point.

The structural components of the visit include the following: chart review of previous records and test results, history, and review of systems; the examination and any office-based tests; the assessment and diagnosis; and the decision making about future visits, tests, and treatment plus the discussion, teaching, and guidance offered to the woman. **Appendix 4A** lists standard precautions that are an essential component of any healthcare visit.

Collecting the Health History

When collecting a health history, the midwife first considers the purpose of the visit. If the woman has come for a comprehensive or general reason, the history obtained will be broader than if the visit is problem focused. Consider a new prenatal visit, which will include a complete personal, social, and family history, as well as genetic risks. Contrast it with a triage visit for nausea and vomiting of pregnancy, which will focus on the current concern, asking about exposure to spoiled food and infectious contacts, allergies, gastrointestinal disorders, and problems with nausea in any prior pregnancies.

The general principle is to work from the least invasive questions to those that require more personal exposure. When one question at a time is asked, and there is a pause to wait for each response, information is less likely to be confused or omitted. Establishing and maintaining an easy flow of dialogue and a nonjudgmental manner promotes open exchange of information. A woman should be advised that some questions are very personal, and that she is not required to answer ones that she does not wish to discuss. It may be necessary to ask particularly sensitive questions on more than one occasion before the woman is able to give a full answer. An example of a situation that may require such an approach is caring for a woman with prior abuse, as discussed later in this chapter. As the health history is completed, it is wise to ask oneself if any questions are missing or any aspect was not considered. **Appendix 4B** provides a review of the complete health history.

Review of Systems

The review of systems (ROS) bridges the divide between prior health history and the current examination. It includes recent signs and symptoms the woman has noticed, such as burning on urination or a rash. The ROS, in combination with the history, can open up more extensive lines of questioning. In many practices, the ROS is a checklist given to a woman to complete while waiting. During a focused visit, the ROS may be part of the conversation during the examination. When questions are asked well, this part of the encounter can identify further areas of concern or topics for health education.

The Physical Examination

Most examinations in women's health are screening examinations, unless the midwife is serving as the primary care provider (PCP) and the history or ROS has identified additional potential health problems that need to be explored in depth. Screening physical examinations limit the details that can be elicited when every organ system is fully evaluated. **Appendix 4C** outlines a comprehensive physical examination.

By tradition, a woman's initial health assessment, whether she is pregnant or not, is comprehensive, meaning that it includes all the major organ systems. Women who see their midwife regularly and have another provider designated as a PCP can receive a "single system" examination that targets and more completely evaluates the genitourinary and reproductive systems. In that case, the thyroid, breasts, abdomen, and pelvis are examined fully, but other systems are not addressed. Because the breast and pelvic examinations are key to any women's health assessment, these components of the physical examination are reviewed in **Appendices 4D** and **4E**.

In-Office Laboratory Testing

A final part of the office examination is the completion of any in-office tests to be performed. Some tests are obtained and sent for analysis, whereas others are performed in the office and the results determined by the individual performing the test. The latter often are termed point-of-care testing. Among the point-of-care tests commonly performed in midwifery offices are urine dipstick, saline and potassium hydroxide (KOH) slides to test for the presence of yeast, trichomoniasis, or bacterial vaginosis, and pregnancy tests (**Appendix 4F**).

CLIA regulates tests performed in an office (42 CFR part 493).[16] The CLIA regulations are federal regulatory standards for all clinical laboratory tests done on humans in all settings except research protocols. CLIA identifies three categories of laboratory tests based on the complexity of test methodology: (1) waived tests, (2) tests of moderate complexity, and (3) tests of high complexity. Waived tests are those that are accurate (i.e., the likelihood of erroneous results is negligible), pose little risk of harm if performed incorrectly, and have been cleared by the Food and Drug Administration (FDA) to be performed at home. Examples of waived tests and tests of moderate complexity are listed in **Table 4-2**.

Table 4-2	CLIA Testing Categories: Waived and Moderately Complex
CLIA Waived Tests	**CLIA Tests of Moderate Complexity**
Dipstick or tablet reagent urinalysis (nonautomated) for bilirubin, glucose, hemoglobin, ketones, leucocytes, nitrates, pH, protein, specific gravity, and urobilinogen	Provider-performed microscopy: all direct wet-mount preparations for the presence or absence of bacteria, fungi, parasites, and human cellular elements
Fecal occult blood	All KOH preparations
Ovulation tests: visual color comparison tests for luteinizing hormone	Postcoital direct, qualitative examinations of vaginal or cervical mucus
Urine pregnancy tests: visual color comparison tests	Qualitative semen analysis (limited to the presence or absence of sperm and detection of motility)
Blood glucose by glucose monitoring devices cleared by the FDA specifically for home use	Fern tests
Diagnosis of STIs or vaginal conditions: HIV, trichomoniasis, bacterial vaginosis	Pinworm examinations
	Urine sediment examinations
	Nasal smears for granulocytes
	Fecal leukocyte examinations

Abbreviations: Clinical Laboratory Improvement Amendments, CLIA; FDA, U.S. Food and Drug Administration; HIV, human immunodeficiency virus; KOH, potassium hydroxide; STIs, sexually transmitted infections.

Accurate results require correct performance of even the simplest test. When the Centers for Disease Control and Prevention (CDC) surveyed laboratories performing waived tests, high rates of errors in quality control and documentation were noted; a document regarding best practices in laboratory testing was subsequently published.[17]

If *only* waived tests will be performed in an office, then a Certificate of Waiver can be obtained to license the office laboratory. The Centers for Medicare and Medicaid Services (CMS) provides explanations of the waiver process and requirements on its website.[18] CLIA tests that are of moderate and high complexity require further registration and documentation.

Provider-performed microscopy, for example, is considered a subcategory of moderate-complexity testing. Information on obtaining a license for an office laboratory can be found in the current CLIA regulations.[19]

Establishing a Differential Diagnosis: Making an Assessment

When all the information available during the visit has been gathered, the midwife makes an assessment based on a differential diagnosis. The "differential" summarizes the various conditions, disorders, and health problems that might be the cause of each identified concern. It is sometimes very straightforward—for example, assuming a woman has come for an annual examination, she is in good health, and her only questions were about choosing a birth control method, the assessment addresses her normal examination findings and the contraceptive counseling or initiation of a method that is appropriate. At other times, the differential diagnosis can be complex. Right lower quadrant abdominal pain in a woman in early pregnancy may be appendicitis, an ectopic pregnancy, a corpus luteum cyst, or any of many other possible disorders. Those conditions that have the most risk are the first differentials considered, followed by those that are most common. The symptoms, history, and examination findings are considered to determine the most likely cause.

Sometimes the initial differential diagnosis will be descriptive rather than diagnostic. In the second example in the preceding paragraph, ectopic pregnancy will always be the first condition considered, as it can cause irremediable harm to the woman.

The differential diagnosis suggests avenues for further testing or evaluation and helps to direct the plan. Continuing with the example of the woman with right lower quadrant (RLQ) pain in early pregnancy, the initial diagnosis is descriptive: RLQ pain during pregnancy. The first tests ordered should be a pelvic ultrasound and a quantitative level of serum human chorionic gonadotropin (hcG).

Designing a Plan

The plan should always include any lab work or procedures ordered, any medications prescribed, and the date of the next visit. For midwives, however, the plan never includes just these three items. Among the *Hallmarks of Midwifery* are health education, counseling, and guidance.[20] Every plan for every woman includes documentation ensuring that these aspects of care were addressed. The reputation of midwifery rests in part on midwives' ability to be available for support, knowledge, and clarity of information. The education and counseling sometimes include just-in-time teaching, such as what a group B *Streptococcus* (GBS) screening culture is and why it is recommended at the 36-week prenatal visit; at other times, they consist of information that addresses long-term plans—for example, new Pap testing guidelines and why the intervals are changing. The plan also includes information that will lead to shared decision making.

Shared Decision Making

The midwife is responsible for providing evidence-based information; the woman is responsible for considering the alternatives, asking questions, and making the decision. There are only rare occasions where professional judgment can override a woman's consent; most of these involve potentially fatal emergencies requiring rapid action. Even then, there are limitations on action—some decisions require consent of a family member or even court approval.

Shared decision making is different from presenting risks and benefits. Woman-centered decision making can occur only when risk, benefits, and the range of available options are clearly communicated. When information is presented in this way, the woman is able to make her decision within the context of her personal values and beliefs and preferences.

Midwives have the responsibility to verify that women are pleased with the amount and type of information received, and feel that their needs have been addressed during a visit. *Informed consent* and *informed refusal* are the traditional terms for documenting a woman's agreement or disagreement with a procedure or plan of care, whereas *shared decision making* encompasses both types of informed decisions but does not imply that a plan of action is simply presented to the individual. The term "informed consent" continues to be a common term in practice, however, and has been codified in several legal documents, especially those stating that the healthcare provider must legally and/or ethically obtain this type of consent before administering a treatment or procedure.[21,22]

Educational and financial status have been shown to affect an individual's preferences for shared or directed decision making: As the disparity between the care provider and the individual widens, the information provided and the path to a decision change.[23]

Studies have concluded that care that uses evidence-based, shared decision making has the potential to improve quality and outcomes.[24,25] For the most effective results, the process should be thought of in terms of shared decision making, as such a process allows for the woman to question and/or decline treatment in whole or part (i.e., make an informed consent or informed refusal). **Table 4-3** summarizes the essential components of informed decision

making.

Table 4-3	Essential Elements to Make an Informed Decision
The known or possible diagnosis	
The nature and purpose of the proposed treatment or procedure	
The benefits and risks associated with the recommended or usual plan	
Complications and side effects (include both common and severe)	
The likelihood of success for this individual	
Reasonable alternatives available, including no treatment	
Benefits and risks associated with the alternatives	
Possible consequences of not following the proposed plan of care	
Assessment of the person's understanding and agreement	

Ethical behavior requires that no harm be done (nonmaleficence), that the midwife acts for the good of the family or community involved (beneficence), that all persons are treated equally (justice), and that the woman be able to make a choice (autonomy). It is sometimes difficult to accept that the choice a woman makes is not the one a midwife is comfortable with, or even an option under initial consideration.

When a midwife cannot or does not provide a requested service, the obligation is to advise the woman about resources in an unbiased manner. The woman's ability to choose is impaired when the line between personal beliefs and professional actions is not clearly drawn. No midwife should act in a way that violates her or his understanding of safety for the woman and evidence-based care. Likewise, no midwife should withhold information or care based on her or his personal beliefs. When a woman's chosen action is outside the scope of midwifery care, whether because of its complexity or because it implies a lack of safety for the woman (or her embryo/fetus if she is pregnant), then the midwife is obligated to refer the woman to another provider.

Conclusion

The expert midwife has mastered the skills of history taking, examination, diagnosis, and treatment. She or he has become comfortable teaching, guiding, and caring for women throughout their lives. Using solid evidence as a base for decisions and focusing on safety for women will have become second nature. Equally important, the expert midwife has moved beyond simple performance of the necessary elements of care to a level where each woman is viewed as an individual and as a member of her community, to respect different cultures and become culturally competent in delivering healthcare services, and to truly believe that all women deserve midwifery care.

Resources

Organization	Description	Webpage
Centers for Disease Control and Prevention (CDC)	Health literacy for public health professionals: Web-based continuing education course designed for public health professionals about health literacy and their role in providing health information.	https://www.train.org/cdctrain/course/1057675/
U.S Department of Health Resources and Services Administration (HRSA)	Health Literacy: Website has multiple resources for healthcare providers on how to address health literacy. Includes essential tools for health literacy.	https://www.hrsa.gov/publichealth/healthliteracy/
	Culture, language, and health literacy: Resources for recognizing and addressing unique culture language and health literacy of diverse communities.	https://www.hrsa.gov/culturalcompetence/index.html

References

1. Sniderman AD, Furberg CD. Why guideline-making requires reform. *JAMA*. 2009;301(4):429-431.
2. Wright JD, Pawar N, Gonzalez J, et al. Scientific evidence underlying the American College of Obstetricians and Gynecologists' Practice Bulletins. *Obstet Gynecol*. 2011;118(3):505-512.
3. Rising SS, Quimby CH. *The Centering Pregnancy model*. New York NY: Springer; 2017.
4. Mercer JS, Erickson-Owens DA, Collins J, Barcelos MO, Padbury JF. Effects of delayed cord clamping on residual placental blood volume, hemoglobin and bilirubin levels in term infants: a randomized controlled trial. *J Perinatol*. 2017;37(3):260-264.
5. Kane Erwin D, Hosford B. Demystifying the nurse-midwifery management process. *J Nurse- Midwifery*. 2011; 32(1):26-32.
6. DeMeester RH, Lopez FY, Moore JE, Cook SE, Chin MH. A model of organizational context and shared decision making: application to LGBT racial and ethnic minority patients. *J Gen Intern Med*. 2016;31:651.
7. Institute of Medicine. *Health literacy: a prescription to end confusion*. Washington, DC: National Academies Press; 2004. Available at: https://www.nap.edu/catalog/10883/health-literacy-a-prescription-to-end-confusion. Accessed June 5, 2017.
8. Nelson W, Reyna VF, Fagerlin A, Lipkus I, Peters E. Clinical implications of numeracy: theory and practice. *Ann Behav Med*. 2008;35(3):261-274.
9. Kim H, Xie B. Health literacy in the eHealth era: a systematic review of the literature. *Patient Educ Couns*. 2017;100(6):1073-1082.
10. Eyler RF, Cordes S, Szymanski BR, Fraenkel L. Utilization of continuous "spinners" to communicate risk. *Med Decision Making*. 37(6):725-729.
11. Golbeck AL, Ahlers-Schmidt CR, Paschal AM, Dismuke SE. A definition and operational framework for health numeracy. *Am J Prev Med*. 2005;29(4):375-376.
12. Galesic M, Gigerenzer G, Straubinger N. Natural frequencies help older adults and people with low numeracy to evaluate medical screening tests. *Med Decis Making*. 2009;29(3):368-371.
13. Ancker JS, Kaufman D. Rethinking health numeracy: a multidisciplinary literature review. *J Am Med Informatics Assoc*. 2007;14(6):713-721.
14. Baker DW. The meaning and the measure of health literacy. *J Gen Intern Med*. 2006;21(8):878-883.
15. Edwards A, Elwyn G, Mulley A. Explaining risks: turning numerical data into meaningful pictures. *BMJ*. 2002;324(7341):827-830.
16. Centers for Disease Control and Prevention. Clinical Laboratory Improvement Amendments: CLIA law and regulations. Available at: https://www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNProducts/downloads/CLIABrochure.pdf. Accessed May 29, 2017.
17. Centers for Disease Control and Prevention. Good laboratory practices for waived testing sites: survey findings from testing sites holding a certificate of waiver under the Clinical Laboratory Improvement Amendments of 1988 and Recommendations for Promoting Quality Testing. *MMWR*. 2005;54(RR-13):1-21.
18. Centers for Medicare and Medicaid Services. Clinical Laboratory Improvement Amendments (CLIA): how to obtain a CLIA Certificate of Waiver. Available at: https://www.cms.gov/Regulations-and-Guidance/Legislation/CLIA/CLIA_Brochures.html. Accessed January 9, 2017.
19. Centers for Disease Control and Prevention. Provider-performed microscopy (PPM) procedures. Available at: https://www.cms.gov/regulations-and-guidance/legislation/clia/downloads/ppmplist.pdf. Accessed May 29, 2017.
20. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
21. Lipkin M. Shared decision making. *JAMA Intern Med*. 2013;173(13):1204-1205.
22. King JS, Moulton BW. Rethinking informed consent: the case for shared medical decision-making. *Am J Law Med*. 2006;32(4):429-501.
23. Verlinde EE, De Laender N, De Maesschalck S, Deveugele M, Willems S. The social gradient in doctor–patient communication. *Int J Equity Health*. 2012;11:12.
24. Moore JE, Titler MG, Kane Low L, Dalton VK, Sampselle CM. Transforming patient-centered care: development of the evidence informed decision making through engagement model. *Womens Health Issues*. 2015;25(3):276-282.
25. Moore JE. Women's voices in maternity care: the triad of shared decision-making, informed consent, and evidence-based practices. *J Perinat Neonatal Nurs*. 2016;30(3):218-223.

APPENDIX

4A

Standard Precautions

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Healthcare-associated infections (HAIs) are any infections that individuals acquire while receiving direct services. It has been estimated that HAI rates range from 5% to 35%.[1] Although activities designed to prevent HAIs were originally targeted toward protection for consumers, healthcare workers are also at risk for HAIs. Therefore, knowledge and institution of standard precautions are a core standard of practice to protect both the women and families for whom midwives provide care, as well as the midwives themselves.

Standard precautions are the essential activities that should be employed by all healthcare workers in all healthcare settings for the purpose of preventing HAIs without regard to specific infection risks. Standard precautions are the most basic level of infection control and have four components: (1) hand hygiene, (2) use of personal protective devices, (3) safe injection practices, and (4) respiratory hygiene/cough etiquette.

Standard precautions[2] are sometimes viewed as interfering with the close relationship valued both by midwives and by women. However, they are also the most effective method of preventing *both* parties from accidentally being exposed to infection. On some occasions it is the midwife who is more at risk when caring for a woman with an infection than the woman herself. Standard precautions are not limited to the hospital environment, although some accommodations to precautions are observed in ambulatory settings.[3] For example, complete contact precautions are not always possible when a woman presents with an open lesion that was not previously disclosed.

Standard Precautions

1. Hand hygiene is to be performed before and after every encounter with an individual, regardless of the healthcare environment—hospital, home, birth center, or office. Alcohol-based cleaners are effective against a wider spectrum of pathogens than soap and water and are preferred unless the hands are visibly soiled. Soap and water are

always used when any dirt, blood, or body fluids are visible.[4]

2. Gloves are worn for vaginal examinations; collection of cultures; birth; phlebotomy, finger sticks, and heel sticks; handling the newborn before the baby is washed and dried; and handling of any sanitary or bed pads, clothing, linens, or other items soiled with body fluids. Hand hygiene is always performed after glove removal.
3. Personal protective equipment (PPE) is used to prevent exposure of skin and mucous membranes (eyes, nose, mouth) to blood, amniotic fluid, vaginal secretions, semen, repeated contact with breast milk, and body fluids or secretions containing visible blood. PPE can include gloves, cover gowns or aprons, masks, protective eyewear, foot covers, and mouthpieces or other barriers for use during resuscitation.
4. Safety needles have mechanisms built in to protect providers from accidental needle punctures. Blunt-tipped needles are advised for drawing up fluids from a vial to minimize needle-sticks associated with sharp needles. Both safety syringes/needles and blunt needles should be used whenever available. Needles are *not* to be recapped, removed from disposable syringes, bent, broken, or otherwise manipulated by hand after use. All sharp needles and disposable instruments should be placed in puncture-resistant disposal containers located in the immediate area, but outside the reach of visiting children.
5. Respiratory hygiene is observed to protect against respiratory and other droplet infections. This includes provision of tissues, masks, and hand cleaner in all healthcare settings; a reminder to women and their family members who are ill to cover coughs or sneezes and utilize the provided supplies; and seeing that all healthcare staff also observe these precautions.

References

1. Flodgren G, Conterno LO, Mayhew A, Omar O, Pereira CR, Shepperd S. Interventions to improve professional adherence to guidelines for prevention of device-related infections. *Cochrane Database Syst Rev.* 2013;3:CD006559. doi:10.1002/14651858.CD006559.pub2.
2. Siegel JD, Rhinehart E, Jackson M, Chiarello L, Healthcare Infection Control Practices Advisory Committee. 2007 guideline for isolation precautions: preventing transmission of infectious agents in healthcare settings. Available at: https://www.cdc.gov/hicpac/pdf/isolation/isolation2007.pdf. Accessed January 9, 2017.
3. Centers for Disease Control and Prevention, National Center for Emerging and Zoonotic Infectious Diseases. Guide to infection prevention for outpatient settings: minimum expectations for safe care 2011. Available at: https://www.cdc.gov/HAI/settings/outpatient/outpatient-care-guidelines.html. Accessed January 9, 2017.
4. Centers for Disease Control and Prevention. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC /IDSA Hand Hygiene Task Force. *MMWR.* 2002;51(RR-16):1-56. Available at: http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf. Accessed January 9, 2017.

APPENDIX

4B

Collecting a Health History

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In women's health, the chief concerns, or reasons for a visit, are frequently focused on reproductive or gynecologic concerns. This does not mean that the personal health or family history can be omitted. A woman's overall health influences healthcare decision making, as prior illnesses and surgeries may affect which type of examination is indicated and which medications can be prescribed. Family history may open up a discussion about future health risks. For midwives with an active primary care practice, many general health concerns, elicited in the history and review of systems, may also be addressed during routine examinations.

History of the Present Illness

After determining the chief concern for a visit, the next step is to inquire about the history of the present illness (HPI). This is often a misnomer—for example, the reason for a visit of "I need my Pap test and birth control pills" leads to an HPI that addresses how the woman is managing her contraception and her satisfaction with her current method (and possibly to a discussion of screening recommendations). A chief concern that begins with abnormal uterine bleeding leads to the more traditional assessment of the history of the woman's current symptoms. A common mnemonic for the questions asked about a health problem is OLD CARTS (**O**nset, **L**ocation/radiation, **D**uration, **C**haracter, **A**ggravating factors, **R**elieving factors, **T**iming, and **S**everity). These questions can be followed by "What has changed now that made you come in?" or "How did you decide it was time to come in?"

Past Health History

A past health history includes review of all the organ systems, mental health, common infections, blood transfusions, injuries and traumas, and surgeries including any complications

(**Table 4B-1**). The list in Table 4B-1 is not comprehensive, but does include questions that are frequently or specifically germane in a woman's health examination. Clearly, any other problems that are identified should also be investigated.

Table 4B-1 Past History

1. Neurologic	**2. Skin**	**3. Respiratory**	**4. Cardiovascular**
Migraine headaches	Chronic skin conditions	Asthma	Hypertension
Other types of headaches		Tuberculosis	Hyperlipidemia
Epilepsy or seizure disorders			CVA
Multiple sclerosis			Myocardial infarction
5. Breast	**6. Gastrointestinal**	**7. Genitourinary**	**8. Musculoskeletal**
Regularity of self-breast examinations	GERD	Frequent UTIs	Arthritis
Biopsy, cyst or adenoma removal	Chronic diarrhea or constipation	Genital tract infections	Any limitations of motion
Breast enlargement, reduction, or reconstruction	Cholecystectomy	STIs	
Other breast problems	Appendectomy	Hysterectomy, myomectomy, oophorectomy	
	Bariatric surgery	Post childbirth repair	
		Cervical LEEP, cone biopsy	
		Maternal DES exposure	
9. Hematologic	**10. Endocrine**	**11. Infections**	**12. Psychological**
Sickle cell, hemoglobinopathies	Thyroid disorders	Childhood infections	Depression
Anemia	Diabetes	Vaccinations, especially TDaP and influenza	Postpartum depression
Bleeding disorders		Chronic illnesses—HIV, hepatitis B or C, herpes	Other mental disorders
		PID	
		Endometritis	
13. Allergies	**14. Medications**	**15. Other**	**16. Risk of violence**
Drug	Prescription	Surgeries	Physical, sexual, or emotional
Environmental	Vitamins	Physical trauma	Violence: current/past
Food	Over the counter	Injuries	Abusive behavior: current/past
	Herbal, homeopathic, nutritional, or other supplements		

Abbreviations: CVA, cerebral vascular accident; DES, diethylstilbestrol; GERD, gastroesophageal reflux disease; HIV, human immunodeficiency virus; LEEP, loop electrosurgical excision procedure; PID, pelvic inflammatory disease; STI, sexually transmitted infection; TDaP, tetanus, diphtheria, pertussis vaccine; UTI, urinary tract infection.

Some commonly asked questions include the following:

1. Have you ever had a major illness—for example, any breathing problems, stomach or liver problems, or any bladder infections?
2. Are there any other problems you can think of?
3. Have you ever had to have any special tests or procedures?

4. Have you ever been admitted to the hospital because you were sick? When was that? Why were you in the hospital?
5. Who has been taking care of you for (problem)? Have they suggested you see anyone else?

For the woman who is seen on a regular basis, asking whether there has been any change in her health, and possibly reminding her of what she previously reported, can assist with time management during the visit.

Social History

The social history, like the sexual history, brings up topics that may be embarrassing or even threatening to some women (**Table 4B-2**). Ask these questions in a quiet, professional tone, and respect the woman's need to avoid certain answers. Providing information "why" the information is needed often reassures the woman and may help the midwife feel comfortable asking for it. Obviously if there is no reason for eliciting sensitive information, asking it should be omitted. Relationship questions should be asked in a pattern that allows women to reveal relationships that are more complicated than "single" or "married with children."

Table 4B-2 Social History

1. Relationship status	2. Diet	3. Substance use	4. Employment/student status
With a partner (male or female) Married Single Widowed, separated, or divorced	Eating habits, history of eating disorders Diet recall Any food restrictions—allergies, vegetarian or vegan, religious, cultural limitations	Caffeine Tobacco Alcohol Marijuana Illicit drugs Use of others' prescriptions	
5. Hazardous exposures at work or home	**6. Physical activity, types and frequency**	**7. Seat belt use**	**8. Guns or weapons in home**

Gynecologic and Pregnancy History

This area is a natural progression after the general history is taken, and some of the items mentioned previously need not be re-asked if the answer is already recorded. Listen for answers to questions not yet asked and note them (**Table 4B-3**).

Table 4B-3 Gynecologic and Pregnancy History

1. Last menstrual period and last regular menstrual period	**2. Age at menarche, timing of menstrual cycles** Any irregularity of timing Amount and duration Premenstrual symptoms Dysmenorrhea Endometriosis	**3. Perimenopausal symptoms** Age at menopause—surgical or natural Use of hormone therapy	**4. Pap test history** Abnormal Pap test Colposcopy Cervical treatment or surgery
5. Contraceptive history[a] Types used Duration of use Any problems	**6. Age at onset of sexual activity** Additional information in the sexual history in Table 4B-4	**7. Pregnancy** G/P-TPAL Gravida (number of pregnancies) Para (number of births); T: term; P: preterm; A: spontaneous or elective abortions; L: number of living children Ectopic and multiple pregnancies Problems with pregnancies, births, or recovery Genetic testing Postpartum depression	**8. Other** Infertility Reproductive life plan or childbearing plans

[a] If a woman has had only women partners or is abstinent, she may have had no need for contraception, but may have still chosen to use hormonal contraception for other benefits.

Sexual History

Core questions, such as age at first coitus, number and gender of partners, and if the woman is currently sexually active or not, are asked as part of a general gynecologic history. Nevertheless, the Centers for Disease Control and Prevention recommends obtaining a complete sexual history during an initial visit, as part of routine preventive examinations, and for any woman who presents for care of possible sexually transmitted infections.[1,2] The sexual history is based on the "5 P's"—**p**artner, **p**ractices, **p**rotection from STIs, **p**ast history of STIs, and **p**revention of pregnancy (**Table 4B-4**). Because this topic can be difficult, it may help to introduce it by telling the woman that these questions are asked of all adults and the information is kept in confidence. The CDC also has a Brief Sexual History Tool that can be filled out privately by the woman and then reviewed during the visit.[2]

Table 4B-4	**Sexual History**
1. Partners	
Are you sexually active? If no: Have you ever been sexually active?	

If yes: Are your sexual partners men, women, or both? (If the individual answers “both,” ask the next questions about partners for each sex and gender partner.)
In recent months, how many sex partners have you had?
What is the sex and gender of your previous sexual partners, including men, women, and transgender individuals?
Do you have any problems with your sexual partner, or does your sexual partner have any problems you want to discuss?
2. Practices
Have you ever had oral sex, meaning mouth on penis or vagina sex? How many times have you had oral sex without a condom or dental dam? Do you have receptive oral sex, meaning when the male ejaculates into your mouth?
Have you ever had vaginal sex, meaning any type of sex that involves insertion of objects into your vagina? Which objects are inserted into your vagina? Are you the receptive partner in female–male intercourse, meaning that the male ejaculates while in your vagina? How many times have you had vaginal sex without a condom or used safe sex practices?
Have you ever had anal sex, meaning penis in rectum/anus sex? Are you the receptive partner, meaning that the male ejaculates into your rectum? How many times have you had anal sex without a condom?
Are your sexual practices more painful than you would like?
If yes: Explore for sexual violence, dyspareunia, and other sources of pain.
3. Protection from Sexually Transmitted Infections
Do you use anything to protect yourself from STIs like HIV?
If no: Can you tell me the reason?
If yes: Which kind of protection do you use and how often do you use it?
Do you have any questions about methods to protect against STIs?
4. Past History of STIs
Have you ever had an STI?
If yes: How were you treated for it?
Have you ever been tested for HIV or other STIs? Would you like to be tested?
Have you had any recurring symptoms?
Has your current sexual partner or former partners ever had an STI, including HIV?
If yes: Were you tested at the same time?
5. Prevention of Pregnancy
Are you currently trying to conceive a child?
Are you concerned about getting pregnant?
Are you using any form of birth control? Do you need information about birth control?
6. Additional Questions
What other things about your sexual health or sexual practices would you like to discuss?
Do you have any other concerns or questions?

Abbreviations: HIV, human immunodeficiency virus; STI, sexually transmitted infection.

Family History

Family histories assist in identifying risk factors and genetic concerns. Some concerns may be a disease or condition; other factors may relate to psychological or social concerns. The health histories of first- and second-degree relatives are most important. Minimum components include the elements listed in **Table 4B-5** for parents, siblings and grandparents.

Table 4B-5 **Family History (Three Generations)**

1. Parents and siblings	2. Chronic disorders	3. Genetic problems
Living or dead	Heart disease—especially coronary artery disease	Birth defects
Age at death	Diabetes	Mental handicaps
Cause of death	Cancer—especially breast, reproductive, or colon	Behavioral conditions

The final question in the history taking should be some variant of "Is there anything else I should have asked you today?" or, alternatively, "Is there anything else I should know or that you want to share with me?"

Review of Systems

The review of systems (ROS) acts as a bridge from the past to the present. It is a structured inquiry about current symptoms or concerns related to each body system. This part of the examination serves as a check for symptoms that the woman may be experiencing but has not yet mentioned. **Table 4B-6** is an example of a self-administered ROS form that women can complete while waiting for the visit with the midwife. In some practices, a standard screening tool for depression is also provided for women to fill out privately before the visit. Alternatively, an ROS may be completed with the physical examination, asking the woman about symptoms as the examination progresses.

Table 4B-6 **Sample Review of Systems for a Woman to Complete**

Please mark any of the following that are bothering you now or have bothered you within the last 2 weeks.

1. Constitutional
- ❑ weight loss
- ❑ weight gain
- ❑ fatigue
- ❑ fever
- ❑ change in appetite

2. Neurologic
- ❑ dizziness
- ❑ seizures
- ❑ numbness
- ❑ trouble walking
- ❑ memory problems
- ❑ headaches
- ❑ fainting

3. Skin
- ❑ rash
- ❑ sore
- ❑ dry skin
- ❑ moles
- ❑ acne
- ❑ eczema

4. Eyes
- ❑ double vision
- ❑ vision changes
- ❑ spots before eyes
- ❑ glasses/contacts

5. Ears, Nose, Throat
- ❑ sinus problems
- ❑ hearing problems
- ❑ earaches
- ❑ ringing in ears
- ❑ sore throat
- ❑ mouth sores
- ❑ dental problems

6. Respiratory
- ❑ painful breathing
- ❑ wheezing
- ❑ short of breath
- ❑ chronic cough
- ❑ spitting up blood

7. Cardiovascular
- ❑ chest pain or pressure
- ❑ leg swelling
- ❑ difficulty breathing when active
- ❑ rapid or irregular heartbeat

8. Breasts
- ❑ pain in breast
- ❑ breast lump
- ❑ nipple discharge

9. Gastrointestinal
- ❑ frequent diarrhea
- ❑ bloody stools
- ❑ nausea/vomiting
- ❑ constipation
- ❑ passing gas or stool involuntarily

10. Genitourinary

- ❑ blood in urine
- ❑ pain with urination
- ❑ frequent urination
- ❑ incomplete emptying of bladder
- ❑ unintended urine leaking
- ❑ leaking urine with cough or lifting
- ❑ abnormal vaginal bleeding
- ❑ painful periods
- ❑ pain with sex
- ❑ premenstrual symptoms (PMS)
- ❑ abnormal vaginal discharge
- ❑ hot flashes

11. Muscle and skeletal

- ❑ muscle weakness
- ❑ spasms
- ❑ muscle or joint pain
- ❑ frequent falls

12. Hematologic and lymphatic

- ❑ frequent bruising
- ❑ enlarged lymph nodes
- ❑ cuts that do not stop bleeding

13. Endocrine

- ❑ hair loss
- ❑ heat/cold intolerance
- ❑ abnormal thirst
- ❑ hot flashes

14. Mental Health

- ❑ mood swings
- ❑ frequent crying
- ❑ anxiety
- ❑ trouble sleeping
- ❑ thoughts of hurting yourself or someone else

15. Allergies

- ❑ hay fever
- ❑ hives
- ❑ seasonal allergies
- ❑ latex allergy
- ❑ food allergies

References

1. Centers for Disease Control and Prevention. A guide to taking a sexual history. Available at: https://www.cdc.gov/std/treatment/sexualhistory.pdf. Accessed May 30, 2017.
2. Centers for Disease Control and Prevention. Brief sexual history tool. Available at: https://www.cdc.gov/actagainstaids/pdf/campaigns/hssc/hssc_sexualhistorytool_v4.pdf. Accessed May 30, 2017.

APPENDIX

4C

The Physical Examination

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As with the health history, this description of the physical examination focuses on those aspects most significant for women's health. Be alert for any inconsistency between the woman's history and the physical examination.

Review of General Principles

1. As with every healthcare visit, hand washing is essential immediately prior to beginning the examination. Alcohol-based gels and/or foams are appropriate. Hand washing within the woman's vision may be reassuring to her.
2. Ask the woman whether she has any area of her body in particular that she wants to have examined.
3. Drape the woman in such a way that only the area being examined is exposed. The approach can demonstrate respect for her body as well as respect for her right to modesty and privacy.
4. The examination should progress from head to toe, and minimize the number of times the woman has to change position (**Table 4C-1**).

Table 4C-1	Physical Examination	
1. Constitutional Findings	**2. Neurologic**	**3. Skin**
Measured height and weight, calculation of BMI	Orientation to time, place, and person	Normal tone and turgor
Vital signs: blood pressure, pulse, respiratory rate, temperature	Cranial nerves (visible alterations)	Rashes, boils, or lesions
General appearance, grooming, cleanliness	Mood and affect Depression score (if formal assessment done)	

4. Head and Neck	5. Respiratory	6. Cardiovascular
Eyes with PERLA	Lung sounds	Heart rate and rhythm
Dental care needed	Respiratory effort	Audible murmurs or extra heart sounds
Thyroid		Pulses
Enlargement of lymph nodes		Varicosities
7. Gastrointestinal	**8. Genitourinary**	**9. Musculoskeletal**
Abdominal tone, guarding, rigidity	CVAT (as illustrated in *Common Conditions in Primary Care* chapter)	Spinal deformity
Bowel sounds	Suprapubic tenderness	Range of motion
Size of liver and spleen, masses	See Appendix 4E	DTR
Hernias, inguinal lymph nodes		Clonus (as described in detail in *Complications During Labor and Birth* chapter)
Rectum (hemorrhoids, fissure)		

Abbreviations: BMI, body mass index; CVAT, costovertebral angle tenderness; DTR, deep tendon reflex; PERLA, pupils equal and responsive to light and accommodation.

5. Talk as the examination proceeds, both to let the woman know what is happening next and to share reassuring findings. Alert the woman in advance if any part of the examination will be uncomfortable.
6. Use a touch that is as firm as needed to elicit accurate information.
7. Share the findings with the woman. If she is anxious about something that is normal, describe why the examination is normal.
8. Discuss physical changes that may be abnormal with the woman briefly during the examination and in more detail after she is dressed at the end of the visit.

APPENDIX

4D

Breast Examination

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Clinical examination of the breasts takes place while taking into consideration any concerns expressed by the woman, any relevant personal or family history, and review of any prior records or reports. Formal breast self-examination is no longer recommended, but rather has been replaced by "breast awareness" as a way to encourage women to recognize abnormal changes between clinical evaluations. There is no consensus about the clinical breast examinations.[1-5] Neither the American Cancer Society[2] nor the U.S. Preventive Services Task Force[3,4] recommends regular clinical breast examination for asymptomatic women. The American College of Obstetricians and Gynecologists continues to recommend offering clinical breast examinations to women at average risk for breast cancer every 1 to 3 years for women aged 25 to 39 years and annually starting at age 40 years.[5] More information on breast evaluation and diagnosis is found in the *Breast Conditions* chapter.

Procedure for Breast Examination

1. Wash hands prior to beginning the examination.
2. The woman should be seated on the examining table so that she is facing the examiner. Her chest area should be entirely exposed. Throughout the examination, use the drape or gown to cover any parts of the woman's body not being examined.
3. Have the woman sit erect, facing the examiner. Look at the breasts with her arms loose at her sides, raised overhead, and then with her hands on her hips so that her elbows are extended 90 degrees from the plane of her abdomen. Ask her to lean forward to check that the breasts hang freely.
   a. With her arms raised, the pectoral fascia is elevated. If there is a carcinoma that has attached to the fascia, the breast may show an indentation in the contour or skin retraction. When her hands are pressed against her hips, the pectoral muscles contract and if there is a carcinoma that is fixed to the underlying fascia, the breast

may elevate more than expected or skin dimpling or nipple deviation may occur. Similarly, when leaning over, the breasts will normally fall freely away from the chest but may exhibit asymmetry or retraction if the fibrosis of a breast lesion is present.

b. Note any visible scars.

4. Palpate the lymph nodes above and below the clavicle on both sides.

5. Ask the woman to lie supine on the table, and have her raise one arm and fold it behind her head or across her forehead.

a. If she has expressed concern about a possible mass or lesion, the opposite breast should be examined first.

6. Gently palpate the axillary lymph nodes. The palpating hand should be moved within the axilla to press anteriorly for the pectoral nodes, posteriorly for the subscapular nodes, along the upper arm for the lateral brachial nodes, and deep in the middle for the central axillary nodes (**Figure 4D-1**).

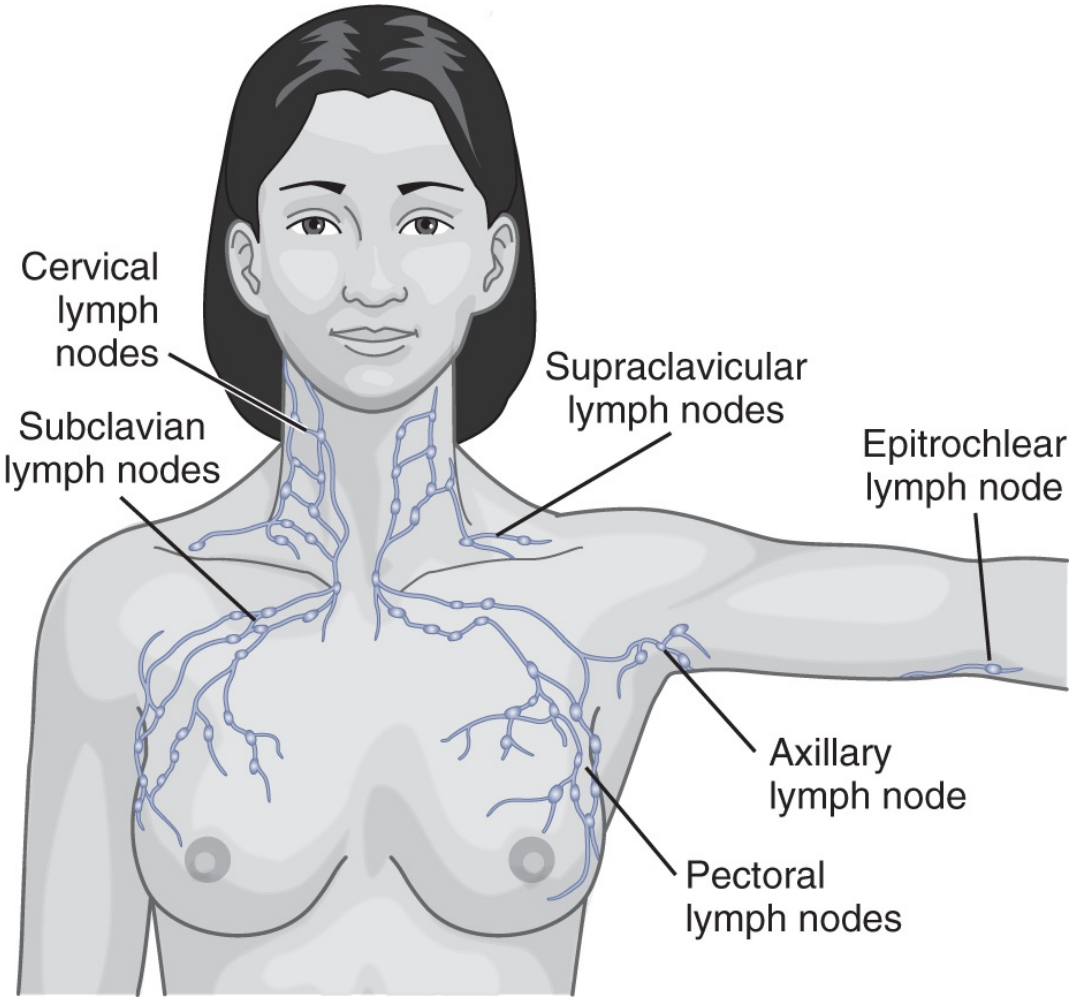


Figure 4D-1 Lymph nodes in the superficial region of the chest and the axillary region.

a. Small isolated lymph nodes that are palpable may reflect irritation from shaving or a localized infection. They should be reevaluated within 1 month.

7. Inspect the appearance of the nipples and areolae.
   a. Nipples may be erect, flat, or inverted. The appearance changes with reproductive maturity, pregnancy, breastfeeding, and aging.
   b. Spontaneous discharge, cracking, lesions, and bleeding are abnormal.
   c. Do not squeeze the nipple in an attempt to elicit discharge.
8. Inspect the appearance of the breasts.
   a. Skin texture and appearance change over time.
   b. Edema, redness, retracted or collapsed areas, visible sores, and masses are all abnormal observations.
9. The most effective pattern for clinical breast examination works up and down the breast, beginning under the axilla and working toward the sternum, and from the clavicle to below the inframammary ridge (**Figure 4D-2**).

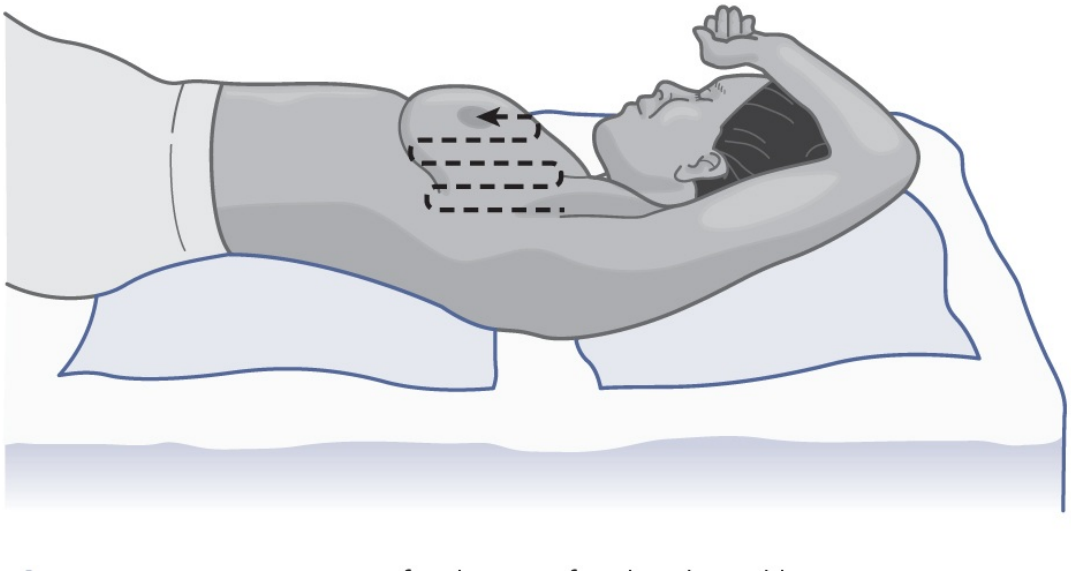


Figure 4D-2 Direction of palpation for the clinical breast examination. Begin under the axilla and move fingers down the length of the breast tissue from the clavicle to the inframammary ridge then up the length of the breast tissue from the inframammary ridge to the clavicle gradually moving toward the sternum until all the breast tissue has been palpated to three different levels of pressure.

10. Palpate each breast for texture and masses. Using the flat surface of the fingers, gently palpate each area being assessed, as illustrated in **Figure 4D-3**. A circular motion is used each time the fingers are placed on the breast. This should not be confused with an older method of breast examination in which the direction of palpation was circular—that method is no longer recommended.

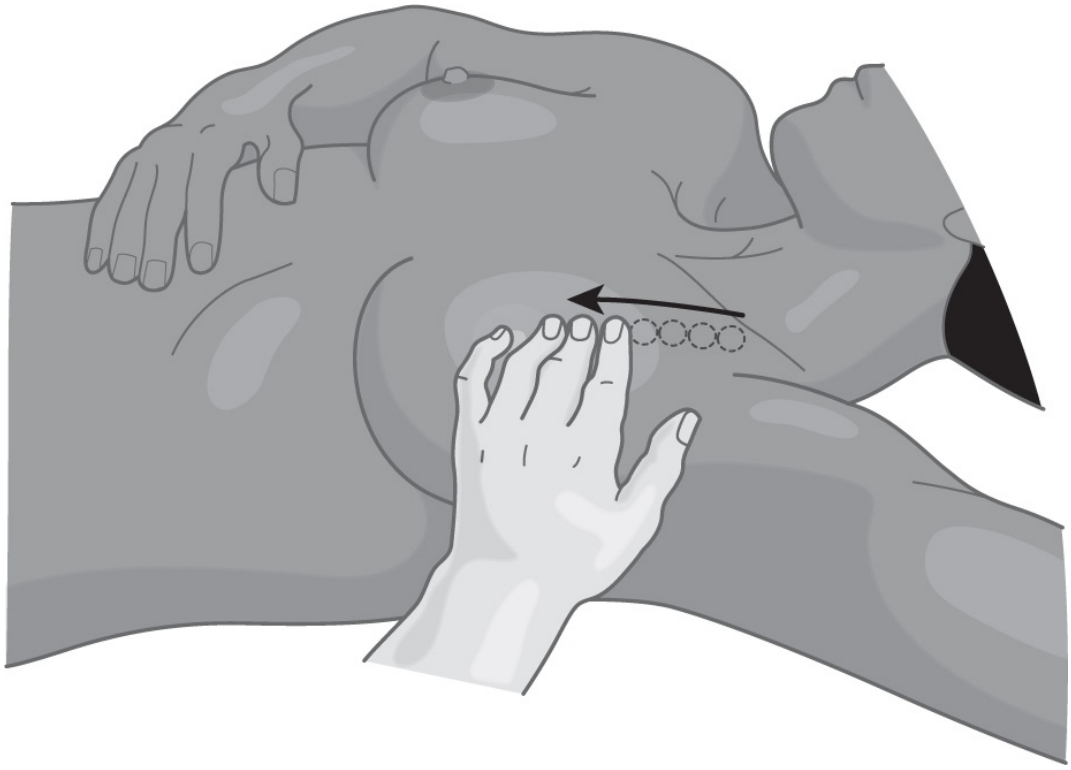


Figure 4D-3 Palpation technique for clinical breast examination.

11. The full depth of the breast to the underlying rib cage is examined (**Figure 4D-4**).

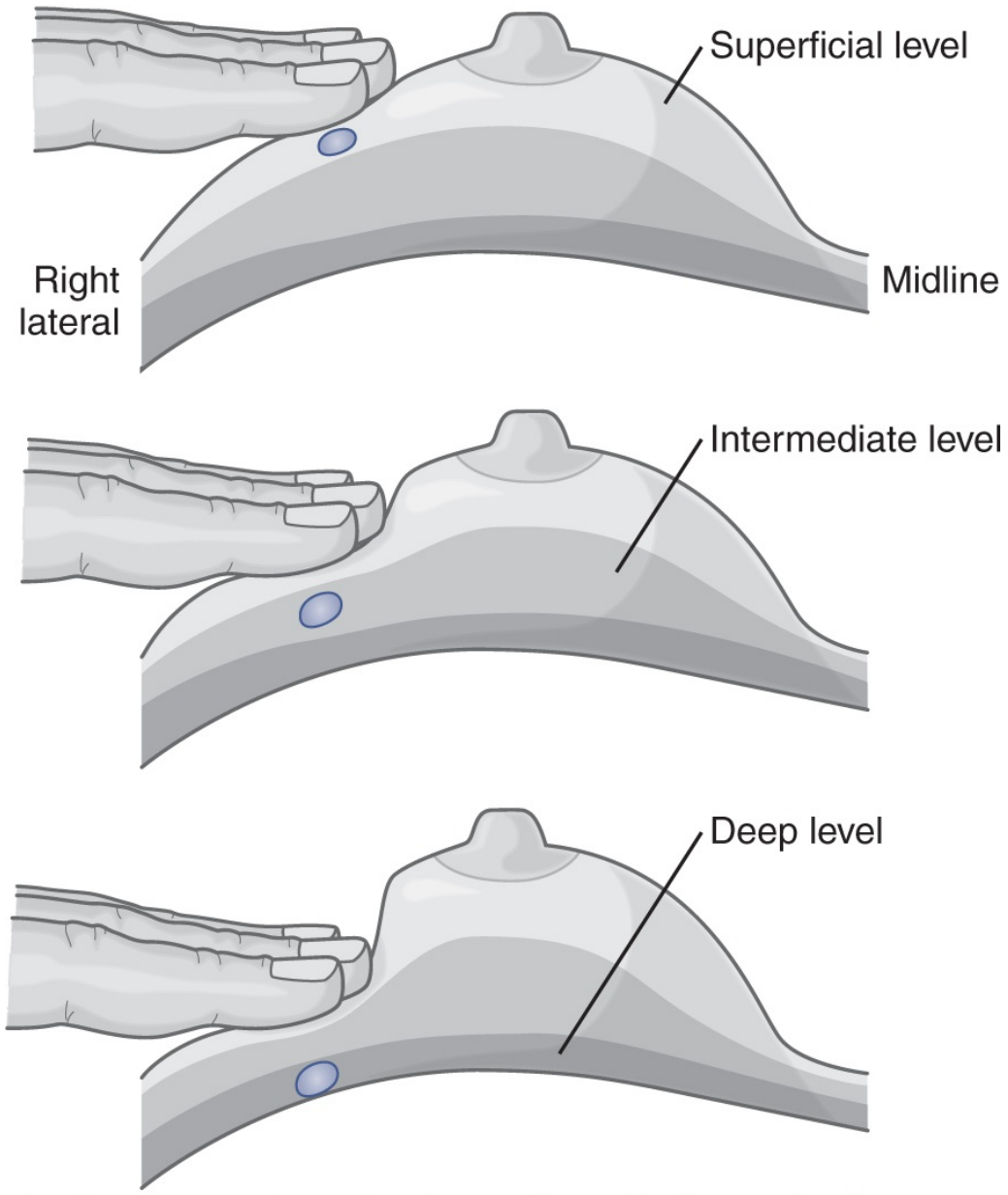


Figure 4D-4 Palpating breast tissue to three different levels of pressure.

a. Breast tissue has texture. Some young women will have very smooth tissue, while an older woman who has breastfed may have an all-over nodular texture. The texture of the breast should be consistent.

b. Prior to the menses, coarse nodularity or firmness may be more noticeable.

c. Palpable masses of any kind need to be evaluated further (**Figure 4D-5**).

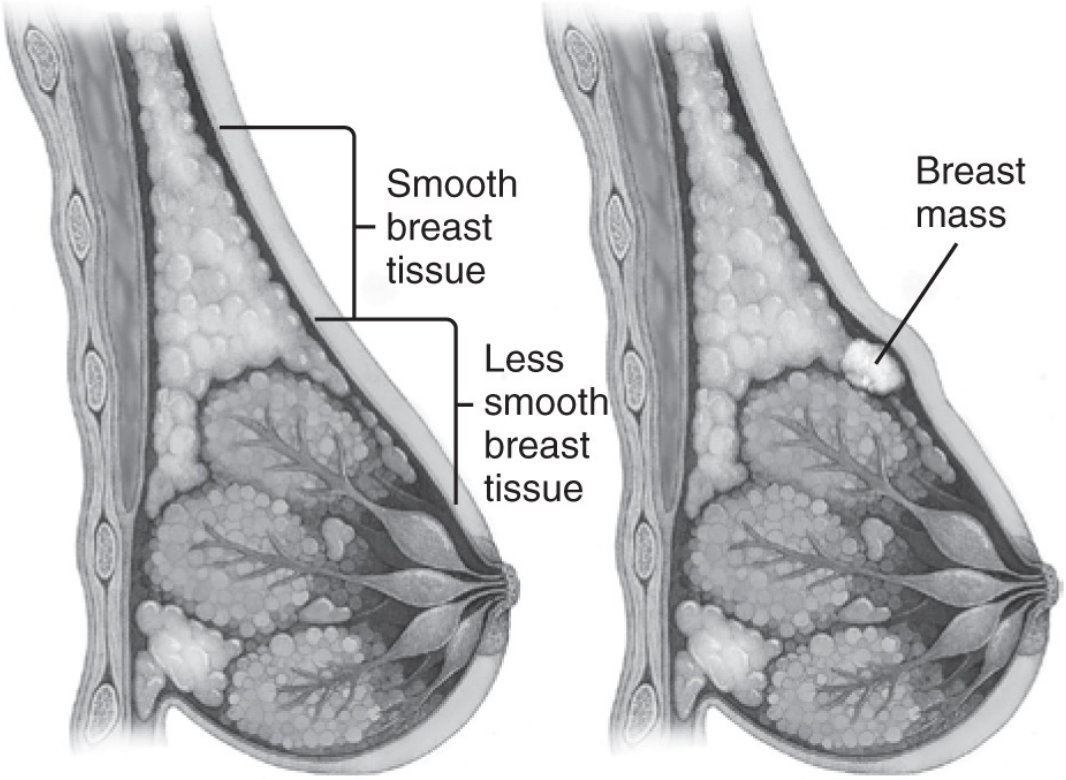


Figure 4D-5 Nodular breast texture versus a mass.

12. While performing the breast evaluation, the examiner describes what is being felt and explains how a woman can recognize breast changes. If the woman wishes to learn self-examination, this is the appropriate time to illustrate the procedure.

References

1. American College of Obstetricians and Gynecologists. Practice Bulletin No. 122: breast cancer screening. *Obstet Gynecol.* 2011;118(2 pt 1):372-382.
2. American Cancer Society. American Cancer Society recommendations for the early detection of breast cancer. Available at: https://www.cancer.org/cancer/breast-cancer/screening-tests-and-early-detection/american-cancer-society-recommendations-for-the-early-detection-of-breast-cancer.html. Accessed July 20, 2017.
3. U.S. Preventive Services Task Force. Screening for breast cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2009;151(10):716-726, W-236.
4. Siu AL, on behalf of the U.S. Preventive Services Task Force. Screening for breast cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2016;164:279-296.
5. American College of Obstetricians and Gynecologists. Practice Bulletin No. 179: breast cancer risk assessment and screening in average-risk women. *Obstet Gynecol.* 2017;130:241-243.

APPENDIX

4E

Pelvic Examination

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The pelvic examination is performed based on a woman's reported concerns, personal history, and family history. Prior to beginning the pelvic examination, the midwife should determine whether the woman previously has had a pelvic examination, and if she has any concerns, questions, or history of problems during a pelvic examination. Prior to any procedure, discussing what is involved is essential. Although this appendix will describe both a pelvic examination and a speculum examination, the midwife should remember that not all women will need a speculum examination with a pelvic examination.

During and after a pelvic examination, the choice of words and personal facial expressions should be used with intention. Helping the woman to remain calm and presenting a professional demeanor throughout are important behaviors. A woman's face usually will convey her level of comfort and sometimes if she has questions. Therefore, visualization of both faces, midwife and woman, should be unobstructed throughout the procedure. It is always preferable to have a chaperone when performing a pelvic examination regardless of gender of the provider; this person assures the woman that no untoward or unprofessional activity is likely. Pragmatically the chaperone also can hand equipment and receive specimens to facilitate the procedure.

1. Before beginning an examination, the woman should be offered the opportunity to empty her bladder.
2. It is assumed that, similar to prior to any physical examination, the midwife performs hand hygiene, preferably in front of the woman to reassure her. To minimize the risk for contamination of the room with vaginal secretions, assemble all necessary equipment prior to beginning the examination. This includes taking lids off containers and opening any packages needed.
3. When the woman is gowned for the examination, she should rest her feet in the stirrups or on the lowered section of the table and move so that her buttocks extends just beyond the end of the table. Thus, her perineum is at the edge of the table, enabling easier insertion of the speculum (**Figure 4E-1**). The woman's arms should be relaxed at her

sides or across her abdomen. Some women will be more comfortable in a semi-reclining position. The drape sheet should be adjusted as needed for modesty and some midwives advocate draping so that the woman's knees are covered and only the pelvic area uncovered.

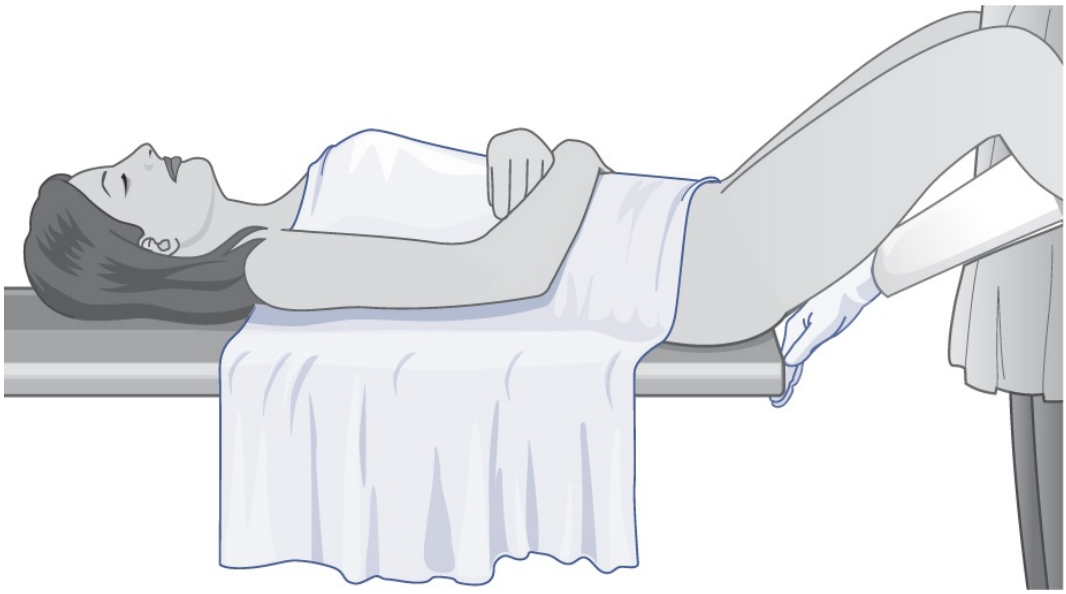


Figure 4E-1 Positioning a woman for a pelvic examination.

- **a.** Before proceeding further with the examination, as well as at any point during the procedure when the woman demonstrates potential discomfort, the midwife should verify that the woman does not feel physically uncomfortable.
- **b.** Note that this is the usual positioning for a woman in North America. In Europe, providers often examine a woman when she is in a lateral position.[1]
- **c.** Women with a physical or mental disorder may require different positioning for a pelvic examination.

4. Adjustment of the light is usually done before gloving and speculum selection. The speculum should be the correct size and mechanically functional.

- **a.** Place the speculum and any needed supplies for the examination on a clean surface. In some practices, the speculums are placed on clean areas with heating pads beneath them to warm, but not heat, the instruments.
- **b.** The means of gloving for this examination is controversial, and no single method has been demonstrated to be superior:
  - **i.** Some midwives double-glove both hands and remove the outermost glove if it touches the woman or any secretions.
  - **ii.** Others double-glove only the hand that will be used for the internal examination. The first glove is then removed after internal contact.
  - **iii.** In the past, it was common to use one glove only, which was placed on the hand that performed the internal examination. However, with the advent of universal precautions, most midwives also glove the hand that performs the

external/abdominal portion of the examination.

iv. The important factor is that the midwife continuously is aware of hand cleanliness. Cross-contamination can occur easily, especially by touching equipment, the light, or the clean area on the table after touching the woman's perineum. It is also helpful to be cautious with language and avoid using terms such as "clean" and "dirty" to designate the hand used for either equipment or direct contact with the woman. The word "dirty" as applied to the woman can be distressing if she hears it.

5. Correct choice of a speculum can both facilitate visualization and decrease the woman's discomfort. Specula come in many sizes and designs. Pederson specula are straight sided; Graves models have a "duck-billed" shape that increases visualization of the vaginal vault and fornices when lax musculature or submucosal fat impedes visualization of the upper vagina. Narrow (virginal) specula and shorter pediatric specula are also available for use whenever conditions require a smaller device. **Figure 4E-2** shows the variety of specula available. Disposable plastic specula are similar in shape to the Pederson metal specula.

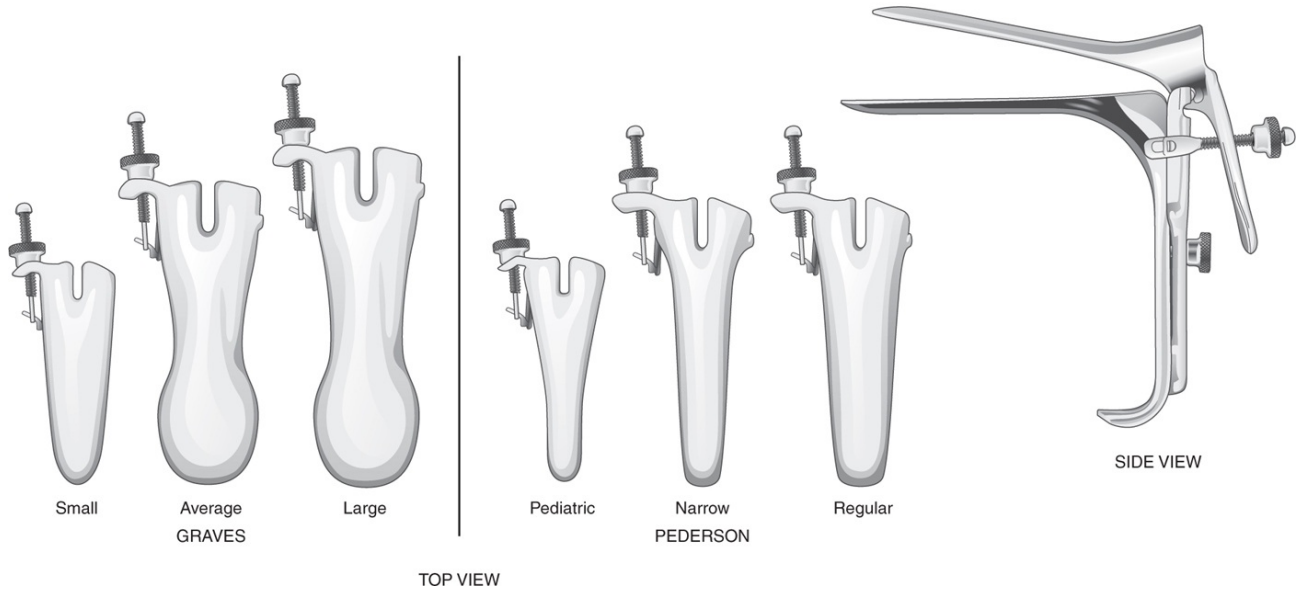


Figure 4E-2 Common vaginal speculum types.

External Inspection of the Genitalia

1. Ask the woman to allow her knees fall apart, and to relax her hip and thigh muscles.
   a. When the legs and buttocks are relaxed, there is less likelihood that the vaginal muscles will be tightened against the speculum.
   b. Remind the woman that she should speak immediately if she is having pain or desires the examination to stop.
   c. Gently touch the inside of the woman's thigh with the back of the gloved hand

intended for the internal examination before proceeding with the examination and let her know before the introitus or vagina are touched.

2. Inspect the external genitalia (**Figure 4E-3**). To do so, separate the labia majora and inspect the labia minora. Then separate the labia minora and inspect the clitoris, the inside of the labia minora, vestibule, urethral orifice, and vaginal introitus. **Table 4E-1** lists observations to be noted.

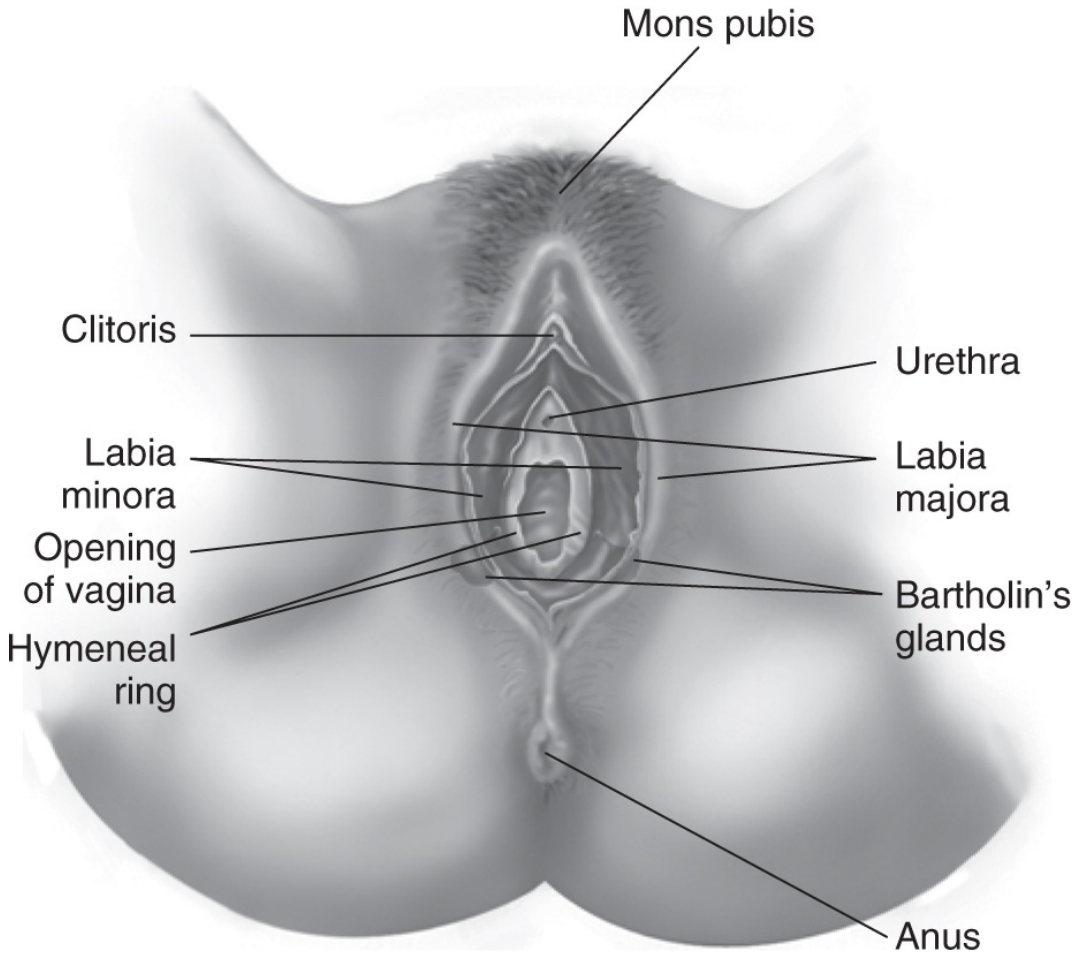


Figure 4E-3 Normal external genitalia.

Table 4E-1	Observations of the External Genitalia
During examination of the external genitalia, many observed changes provide information of clinical significance. Assess the following:	
Pattern of hair growth	Discoloration or bruising
Size and shape of each area	Cysts, polyps, condyloma, or other growths
Appearance of the introitus	Lesions, fissures, rashes, ulcerations, or crusting
Clitoral enlargement	Adhesion of tissues

Inflammation or irritation	Fistula
Swelling or edema	Uterine prolapse
Scarring	Varicosities

a. Inspect the Skene's glands and urethra for normal appearance, irritation, swelling, lesions, redness, or discharge. Separate the labia; insert one finger into the vagina, palm up; and sweep down the Skene's glands at each side of the urethra toward the introitus. Pressing directly upward onto the urethra, again sweep from the apex of the vagina toward the introitus to elicit discharge from the urethra if present.
b. Inspect the Bartholin's glands for masses, fluctuation, redness, heat, or pain, with one finger in the vagina and the other fingers and thumb outside the vagina. Palpate the entire area, usually by gently palpating the tissue between the thumb and the index finger, both sides of the vaginal opening in turn. Pay particular attention to the posterolateral portion of the labia majora.
c. Inspecting the glands before performing the speculum examination increases the likelihood that any discharge will be noted.
d. This sequence also allows for smooth transition to the speculum examination.

Speculum Examination

1. Start with use of the hand to be designated for use with equipment. Verify that the speculum previously chosen is appropriate in size and that it is warm (skin temperature). If not, discard the glove on the hand that palpated the external genitalia, correct the speculum size or temperature, and reglove.
2. If needed, lubricate the warm speculum with water, as other lubricants can interfere with specimen interpretation. Check the speculum before using it to make sure the locking knobs (metal speculum) or latch (plastic speculum) work correctly.
3. While holding the speculum by the handle and securing the two blades together with the index finger across the top blade to avoid inadvertent opening and increasing the size/diameter of the device, have the two fingers that remained in the vagina after palpation of the labia positioned in the posterior introitus (**Figure 4E-4**). If needed, the fingers may be slightly spread apart to create a visible space for insertion of the speculum.

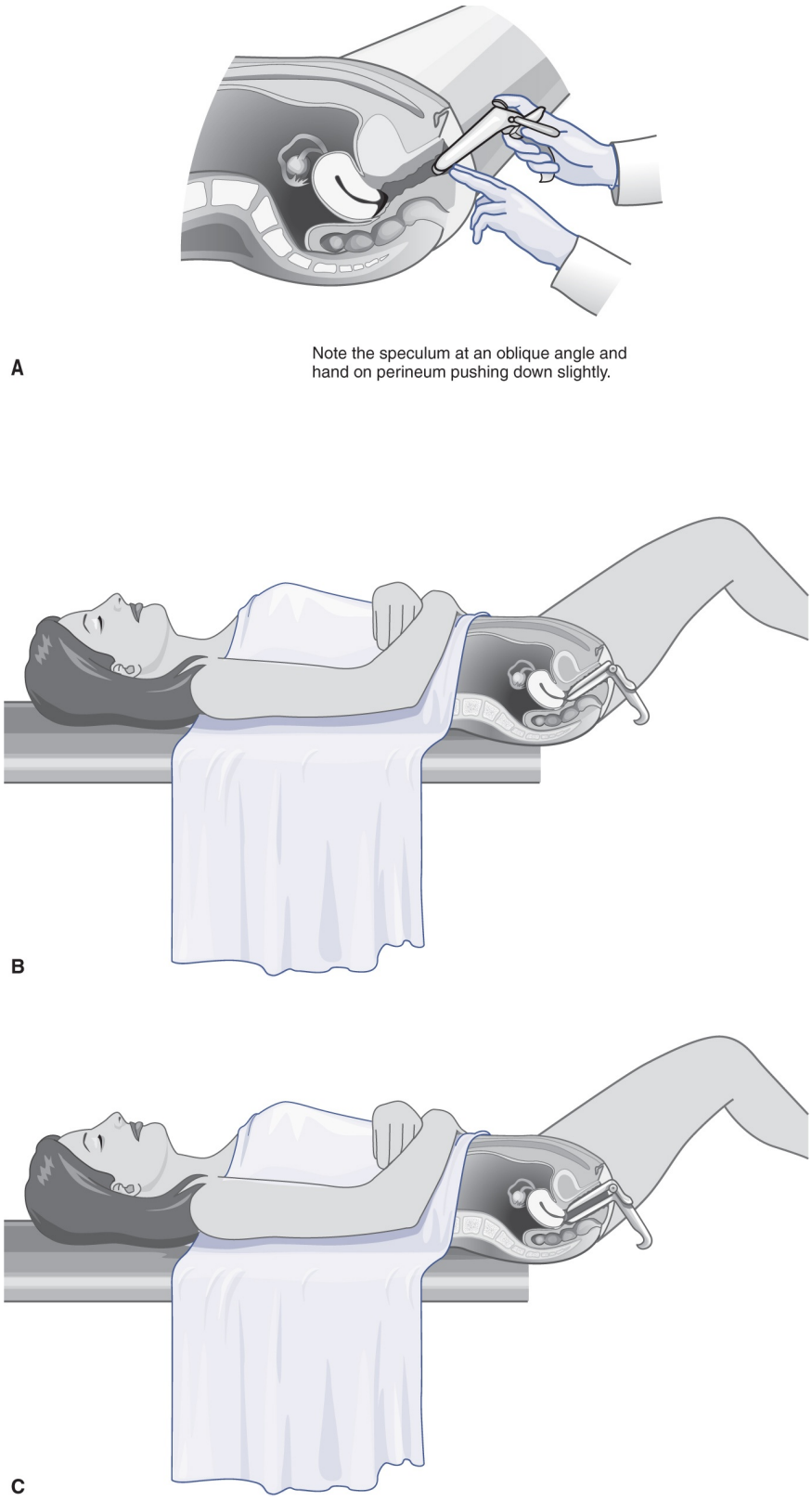


Figure 4E-4 Speculum examination. **A.** Position of the hands and speculum at insertion (speculum at oblique angle). **B.** Speculum inserted along the posterior wall of the vagina. **C.** Speculum open to visualize cervix.

Modified with permission from Schuiling KD, Likis FE, eds. *Women's gynecologic health.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017.[2]

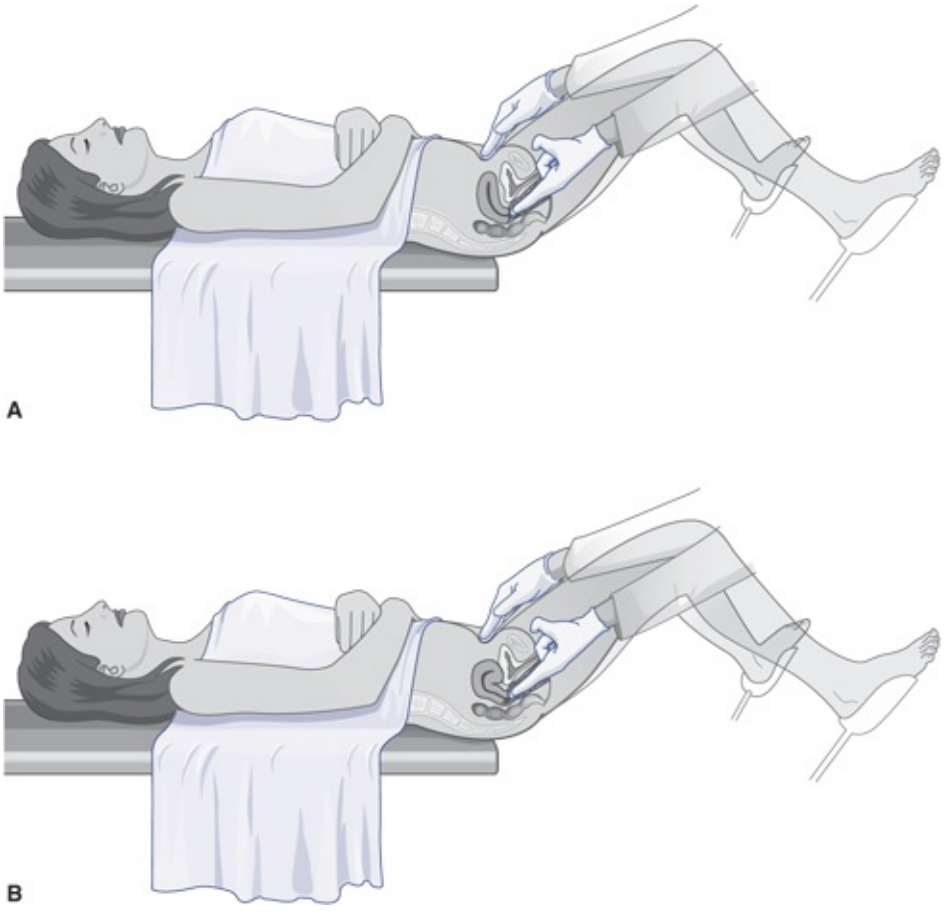
4. Provide gentle downward pressure with the two fingers and guide the speculum blades between the two fingers into the vagina. The speculum should enter and be removed from the vagina at a 45-degree angle so the speculum avoids touching the sensitive anterior structures (e.g., urethra, clitoris). As the speculum enters the vagina, at a 45-degree angle, slowly begin to remove the fingers as the speculum slides downward along the posterior vaginal wall.
5. Rotate the speculum to a horizontal position as it moves toward the posterior portion of the vagina. Do not open the bills until the speculum is fully inserted.
6. Maintaining downward pressure, withdraw the speculum slightly and open it only as much as is needed to visualize the cervix using the thumb piece.
    a. The most common cervical position is tilted slightly posterior, so placing the speculum behind the cervix and gently opening it is the technique most likely to identify the cervix with minimal discomfort for the woman.
7. When the cervix is visible, insert the speculum slightly deeper to stabilize and improve visibility, and then use the screws or latch (on a plastic speculum) to fix the open position of the speculum. The anterior blade of a metal speculum can be adjusted if a larger area needs to be created.
    a. Note any discharge, cysts, polyps, lesions or masses, vascularity, erosion, or eversion.
8. It is at this point that specimens are collected if needed.

After collecting specimens, rotate the speculum to visualize the anterior and posterior walls of the vagina. Many midwives will perform this inspection as they slowly remove the speculum. Note that the speculum blades can be allowed to fall together after clearing the cervix. The speculum should be rotated back at a 45-degree angle as it nears the vaginal introitus and then gently removed. The woman may give a gentle push that is unnecessary, but may make her feel more in control.

Bimanual Examination

1. If necessary, discard gloves and don a new one/pair. Apply water-based gel to the fingers of the gloved hand that will be inserted into the vagina.
2. This portion of the examination is performed with two fingers in the vagina, unless the introitus is too tight or the woman too uncomfortable to tolerate more than one finger.
    a. Keep the thumb of the examining hand tucked to one side or folded into the palm to avoid unintentional pressure on the clitoris and to allow the fingers to reach farther into the vagina.
3. Pressing gently downward, insert two fingers and place them along the posterior wall of the vagina.
4. Press downward firmly to open the vagina and ask the woman to bear down, observing for the bulge of a cystocele or urethrocele. Observe for uterine descent associated with prolapse.

5. Spreading the examining fingers apart, the woman should be asked to bear down a second time, and posterior bulging associated with a rectocele or enterocele is observed if present.
6. Ask the woman to tighten her vaginal muscles around the vaginal fingers.
   a. Kegel's exercises can be taught at this point.
7. Sweep the vaginal fingers around the walls to assess for masses or lesions such as cysts, polyps, or condyloma.
8. When the fingers reach the cervix, they should be moved circumferentially around the cervix.
   a. Assess for size, consistency, smoothness, shape, mobility, and dilation.
9. Move the cervix gently side to side between two fingers to assess for cervical motion tenderness.
10. Place the external hand above the symphysis and press downward and forward toward the vaginal hand with the palmar surface of the fingers. With the vaginal hand, lift upward directly against the cervix to bring the uterine fundus in contact with the hand on the abdomen. Press both hands together gently to outline an anteverted or anteflexed uterus. The uterus should move smoothly between the hands. If necessary, reposition the abdominal hand farther up the abdomen to locate the fundus.
11. Note the position of the uterus (anteverted, retroverted, anteflexed, retroflexed, midline). Also note the shape, size, consistency, mobility, tenderness, or presence of any masses.
12. If the uterus is not identified, repeat the maneuver with the vaginal fingers on either side of the cervix (**Figure 4E-5** illustrates uterine positions).



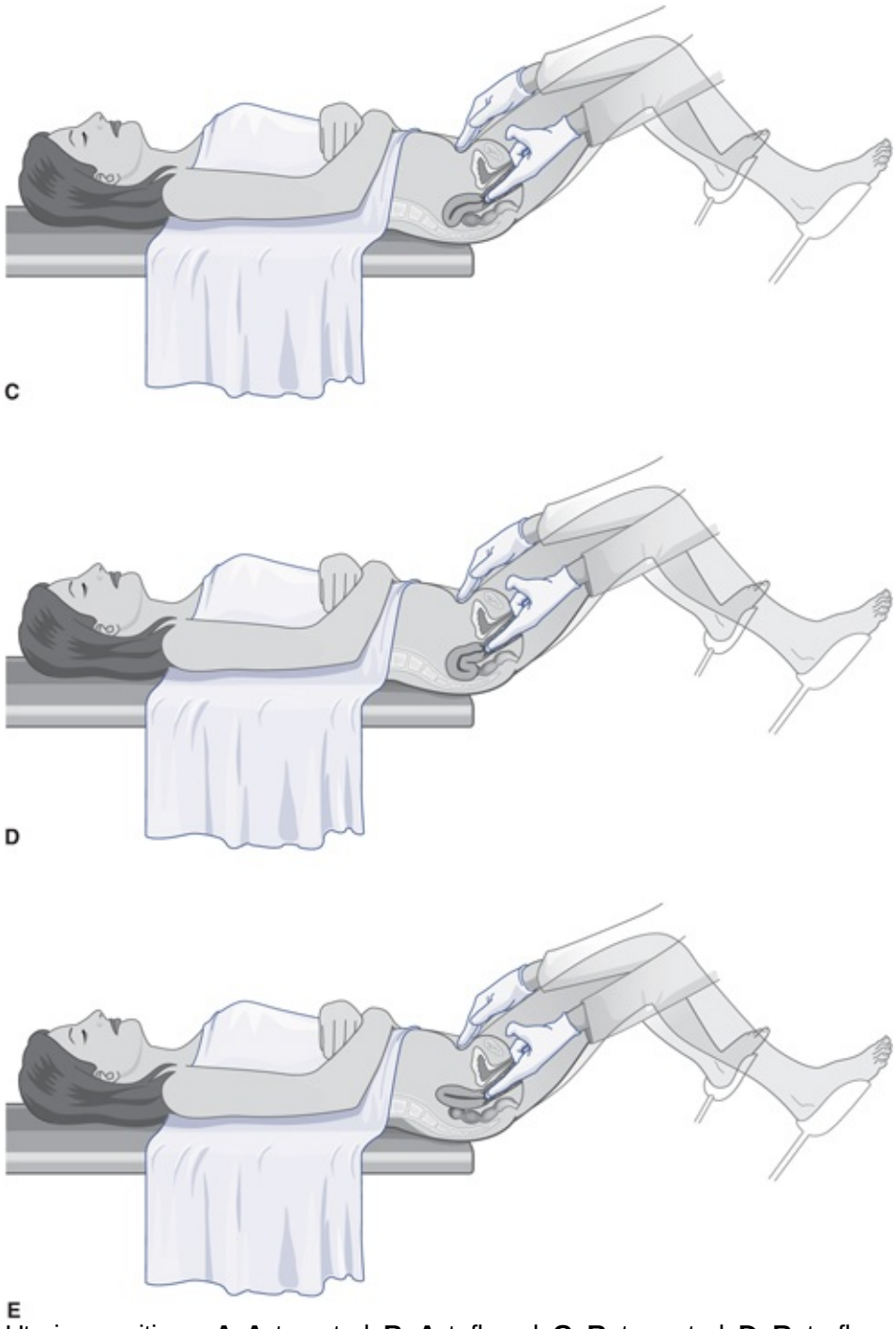


Figure 4E-5 Uterine positions. **A. A**nteverted. **B. A**nteflexed. **C. R**etroverted. **D. R**etroflexed. **E. M**idline. Modified with permission from Schuiling KD, Likis FE, eds. *Women's gynecologic health.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017.[2]

13. If the uterine position is still not palpated, it may be in a midline or posterior position. With fingers in the vagina above and below the uterus, press inward with the external hand and assess as much of the lower portion of the uterus as is possible.
14. To assess the adnexa, place the external hand in the area between the iliac crest of the innominate bone and the abdominal midline midway between the level of the umbilicus and the symphysis pubis. Use the flats of the palmar surface of fingers on the abdomen to press deeply downward and obliquely toward the symphysis pubis and toward the fingers in the vagina.

15. With the internal hand in the vagina, the palm should face upward. Both of the examining fingers in the vagina are placed in the lateral vaginal fornix corresponding to the side (right or left) that the abdominal hand is positioned to examine. Press the fingers deeply inward and upward toward the abdominal hand as far as possible.
16. Palpate the entire area between the uterus and the pelvic sidewalls with a sliding, gentle, but firm touch, pressing the internal and abdominal hands toward each other as they synchronously move together from the abdominal area above the pelvic brim downward toward the symphysis.

Rectovaginal Examination

1. Not all regular examinations include a rectovaginal examination. Before attempting a rectovaginal examination, the midwife should explain why the examination is indicated —to further evaluate the uterus and adnexa or to check for fistula. Offer reassurance that if the woman can relax, the examination should not be painful.
2. Change current gloves and lubricate the examining fingers.
3. Insert a gloved index finger into the woman's vagina. Ask the woman to bear down, and gently insert the middle finger into the rectum.
4. Palpate the area of the anorectal junction and just above it. Ask the woman to tighten and relax her rectal sphincter.
   a. This position allows for assessment of sphincter tone and for internal hemorrhoids.
5. As both vaginal and rectal examining fingers slide as far as they will reach, palpate half of the rectal wall, sweeping the examining finger back and forth as the distance is covered methodically. Asking the woman to bear down extends the area that can be reached and makes the procedure more comfortable for her.
6. When a uterus is retroverted or retroflexed, the posterior side of the uterus may be palpated. Palpate as much of the posterior side of the uterus as possible with the rectal examining finger.
7. As the vaginal and rectal fingers are withdrawn, the other half of the rectal wall is examined as in step 5.
8. Remove gloves and discard them. Perform hand hygiene.
9. Assist the woman to a sitting position and offer her cleansing wipes and tissues with which to clean herself.

References

1. William A, Williams M. A guide to performing pelvic speculum exams: a patient-centered approach to reducing iatrogenic effects. *Teach Learn Med*. 2013;25:383-391.
2. Schuiling KD, Likis FE, eds. *Women's gynecologic health*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017.

APPENDIX

4F

Collecting Urinary, Vaginal, Cervical, and Rectal Specimens for Testing and Interpretation of Saline and KOH Slides

JAN M. KRIEBS

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Specimens collected during pelvic examinations are used to test for infections, risk of preterm labor, rupture of the membranes, and cervical cancer. In all cases, standard precautions are used to prevent provider exposure to blood and body fluids.[1] The order of specimen collection is determined by the purpose and type of specimen collected.

General Principles for Specimen Collection

1. Cross-contamination should be carefully avoided—either of the equipment and supplies or of the woman and clinical specimens—by the appropriate use of gloves and clean or sterile technique, and by attention to surroundings.
2. Avoiding the use of gel when collecting these specimens improves detection. The gel can interfere with testing by obscuring the collection site, altering the pH of a specimen, and making microscopy less accurate. Only warm water should be used to moisten a speculum.
3. The usual order of specimen collection is as follows:
   a. Voided urine
   b. Vaginal swab for culture/nucleic acid amplification tests (NAATs)
   c. Vaginal swab for pH, saline, or KOH
   d. Endocervical swab for NAAT or culture
   e. Papanicolaou (Pap) testing

Procedure for Collecting Urine Specimens

Urine specimens can be used for urinalysis, urine culture, and NAAT for *Neisseria gonorrhoeae* and *Chlamydia trachomatis*.

1. Determine whether a catheterized specimen is necessary before collecting the specimen. Examples of times when this might be necessary are after rupture of the membranes, during the early postpartum period, when it will be difficult for the woman to adequately clean the area, or whenever a definitive measurement of urinary protein, leukocytes, or erythrocytes is necessary.
2. If performing a bladder catheter collection:
   a. Open the sterile catheter and leave it in the sterile wrap.
   b. Don sterile gloves.
   c. Place the end of the catheter where the urine will come out into a sterile collection device.
   d. Holding the labia away from the urethra, cleanse the periurethral and vaginal area with soap (not disinfectant, which may interfere with culture). Wipe from front to back, and repeat twice, using a clean pad each time.
   e. Using aseptic technique, insert the catheter tipped with water-based lubricant into the urethra until urine is returned (usually 4–5 cm).
   f. Remove the catheter from the urethra when an adequate specimen is obtained or when urine no longer drains, depending on the purpose of the procedure.
   g. Cap the sterile collection device without contaminating the inside by touching it.
   h. Remove gloves.
   i. Label the sterile collection device.`

Procedure for Collecting and Interpreting Saline and Potassium Hydroxide Specimens for Vaginal Infections[1,2]

In the United States, microscopy for the diagnosis of infection or rupture of the amniotic membranes falls under the Clinical Laboratory Improvement Amendments of 1988 (CLIA). These federal regulations, which mandate improved quality controls, include requirements for competence in provider-performed microscopy. The Centers for Medicare and Medicaid Services is responsible for these regulations, which are published online through the CDC.[3] As with any procedure that has the potential for exposure to blood or other body fluids, standard precautions are always observed. The first step is knowing how to use the microscope (**Table 4F-1**).

Table 4F-1	Use of the Optical Microscope

An optical microscope can be used to facilitate diagnosis in the clinical setting.

Proper microscope maintenance—minimizing exposure to dust by covering the microscope when not in use, cleaning the eyepiece and objective lenses with compressed air and a lint-free paper wipe, and wiping down mechanical components with a microfiber or other lint-free cloth—will preserve the quality of images.

Prior to using a microscope for the first time, familiarity with the light source, adjustment of the fine- and coarse-focus knobs, technique for moving the slide on the mount, and the powers available for viewing a specimen is necessary.

Most modern microscopes will have a 10× eyepiece lens and 4×, 10×, and 40× objective lenses. The oil immersion lens is not used for these procedures.

Saline specimens are used for the direct visualization of vaginal yeasts, trichomonads, and clue cells typical of bacterial vaginosis. Assessment of the presence or absence of lactobacilli and white blood cells can also aid diagnosis.

A sample placed in potassium hydroxide (KOH) is used to visualize yeasts. The KOH destroys the cell walls of bacteria and epithelial cells, but not the cell walls of fungi. KOH can also be collected for use in the "whiff" test, to identify release of amines when a bacterial vaginosis specimen is exposed to an alkaline solution that also releases a strong odor.

1. Obtain dropper bottles of normal saline and KOH. Saline is preferred over distilled water to help preserve the living specimen long enough for examination.
2. Insert a moistened speculum into the vagina and visualize the cervix.
3. As the speculum is inserted and positioned, note the appearance of the perineum, vaginal mucosa, cervical epithelium, and any discharge. The discharge is categorized by quantity, color, consistency, and odor.
4. Using a sterile cotton swab, collect a sample of fluid from the posterior fornix and/or from the vaginal wall. If necessary, a second swab may be used to obtain an adequate sample.
5. Gently roll the swab across a glass microscope slide. Repeat the smear on a second slide. The specimen smear should be thin, not clumpy.
6. If gloves are contaminated, don a new set of nonsterile gloves. Place a single drop of saline onto the sample on the slide.
7. Place a single drop of KOH solution onto the sample on a second slide. It is preferable to use a second slide when collecting a sample for KOH, to prevent mixing with the saline specimen.
8. Place a cover slip over each specimen. Hold the cover slip by the edges. The cover slip should be placed at a 45-degree angle and lowered slowly, to minimize air bubbles (**Figure 4F-1**).

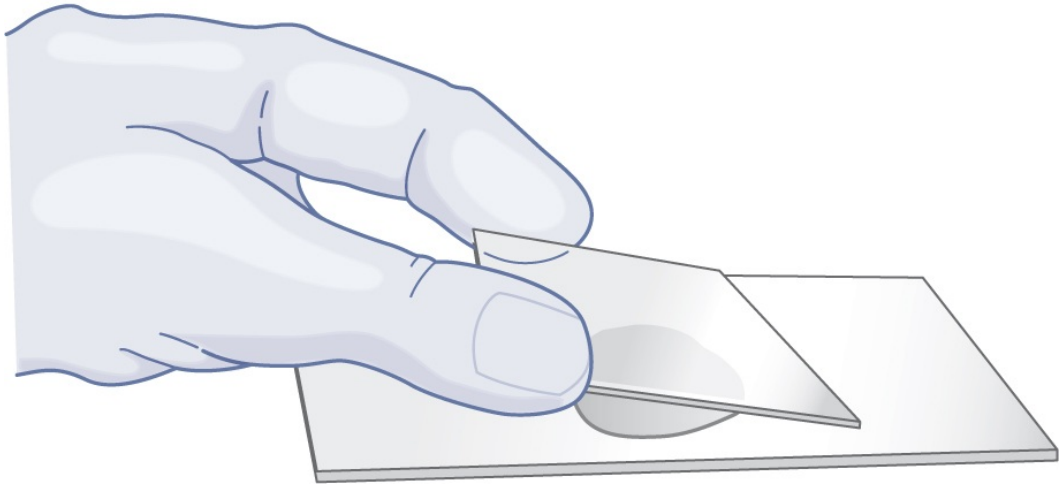


Figure 4F-1 Cover slide placement.

9. If the cover slip is floating because the specimen is too thick, a small piece of lint-free paper can be used to absorb some of the excess fluid. Place the edge of the paper at the edge of the cover slip, and allow the paper to wick away excess fluid.
10. The saline slide is inspected first, followed by the KOH slide if needed. If slides are not labeled, the difference between the two can be seen. Cells are visible on the saline slide, whereas a KOH slide looks empty or has remnants of epithelial walls. Yeast hyphae remain visible after KOH application.
11. Focus on the edge of the cover slip using the low-power lens (10×). At this point, the fine adjustment should be all that is necessary to bring specimens into focus. Fingerprints, lint, and dust can be misidentified as part of the specimen. Keeping the slide as clean as possible and carefully focusing on the edge of the cover slide will assist in finding the correct viewing plane.
12. Examine the specimen by moving the slide back and forth, using the knob or handle provided for that purpose. Enough of the slide should be inspected to provide a correct diagnosis. The entire specimen may need to be viewed because the specimen will not be distributed equally across the slide.
13. Specimens of vaginal secretions are inspected under 40× power. The samples are read as positive or negative, with details of the specific findings as needed. While the 10× lens gives an overview of cells and fungal material present, the 40× lens enables the examiner to identify blood cells, clue cells, bacteria, trichomonads, and yeast hyphae or spores accurately (**Figure 4F-2**). A more detailed discussion is found in the *Reproductive Tract and Sexually Transmitted Infections* chapter regarding the diagnosis of vaginal infections and STIs.

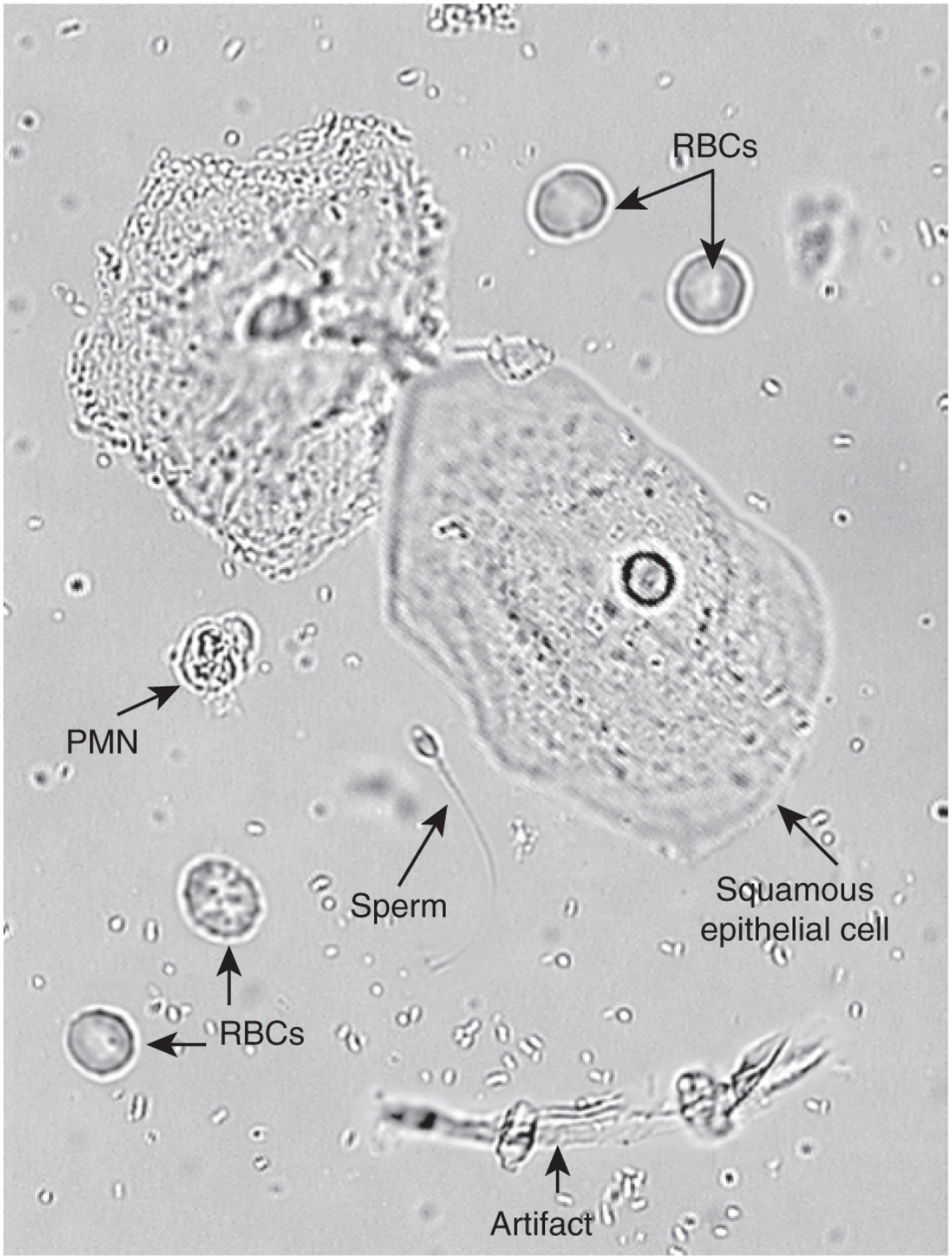


Figure 4F-2 Vaginal slide prepared with saline.

Abbreviations: PMN, polymorphonuclear; RBCs, red blood cells.

Reproduced with permission from Centers for Disease Control and Prevention. CDC STI slide set. Available at: https://www2a.cdc.gov/stdtraining/ready-to-use/Manuals/Vaginitis/vaginitis-slides-2013.pptx. Accessed April 1, 2017.[4] Used with permission from Seattle STD/HIV Prevention Training Center and Cindy Fennell, MS, MT, ASCP.

14. Use the knob to move the stage away from the optic so as to minimize the risk of scratching the lens. Turn off the microscope light, clean up any spilled material, and

properly discard the sample to complete the procedure.

Procedure for Collecting Vaginal or Cervical Specimens For *N. gonorrhoeae* and *C. trachomatis*

Most laboratories now use NAAT or similar DNA-based testing for evaluation of mucopurulent cervicitis and for screening for *Neisseria gonorrhoeae* and *Chlamydia trachomatis* infection.[5,6] These tests are the most sensitive available, and their specificity is similar to that of culture. If the product being used is approved for vaginal testing, these specimens are as accurate as cervical ones, and more accurate than urine specimens in women. A provider can collect vaginal swabs, or such samples may be self-collected by the woman. Culture for *N. gonorrhoeae* is utilized as an alternative when these tests are not available or when resistance testing is needed. *Chlamydia* cultures have poor sensitivity and specificity.

1. Insert a moistened speculum into the vagina and visualize the cervix.
2. Note the presence of any discharge or cervical irritation.
3. For a vaginal specimen, swab the upper vaginal wall and posterior fornix for 10–30 seconds with a Dacron swab.
4. For cervical specimens, first use a large swab to remove any mucus discharge obscuring the cervical opening.
5. Insert a sterile Dacron swab into the cervix approximately 2 cm and rotate for 5 seconds. The specimen collected for liquid cytology Papanicolaou tests is adequate for this purpose allowing the single specimen to be used for Pap, chlamydia, and gonorrhea testing.
6. Do not allow a cervical swab to touch the vaginal walls while it is being removed.
7. Place the specimen in the transport medium.

Procedure for Performing a Papanicolaou Test

Liquid-based specimen collection, in which the specimen is collected at the cervix, has become the standard where available, due to improved ability to interpret abnormalities. This material can also be used for human papillomavirus (HPV) testing.[7] In a conventional Papanicolaou (Pap) test, a slide is made directly from the specimen collected at the cervix. An extended-tip spatula plus endocervical brush technique or broom plus endocervical brush has been shown to provide the most accurate results, and should be used whenever available,[8-10] A discussion of frequency of cervical cancer testing can be found in the *Health Promotion and Health Maintenance* chapter.

1. Insert a moistened speculum into the vagina and visualize the cervix.
2. If only a Pap test is being performed, a small amount of water-based gel can be used to lubricate the speculum.

3. Using a spatula or broom, sweep the cervix, rotating the spatula or broom 360 degrees to cover the entire ectocervix. An extended-tip spatula is the preferred device. If the squamocolumnar junction is visible, it should be included.
4. Remove the device without touching the vaginal sidewalls; rotate it gently in liquid medium to dislodge the cells, or roll it across a clean slide, depending on whether a liquid or dry collection method is used.
5. For dry specimens only: Place one flat side on the top half of the slide and stroke once to the end of the slide. Then turn the spatula or brush over and place the other flat side on the bottom half of the slide and stroke once to the end of the slide. If the specimen is too thick, take the edge of the device and, with a single light stroke down the slide, remove the excess.
6. Insert a cervical brush into the endocervix approximately 2 cm and rotate through 90 to 180 degrees.
7. Repeat the procedure for processing as in steps 4 and 5.
8. If a dry technique is used, the specimen should immediately be fixed before transport to preserve cellular integrity.

References

1. Centers for Disease Control and Prevention. Self-study STD modules for clinicians: vaginitis. Available at: https://www2a.cdc.gov/stdtraining/self-study/vaginitis/vaginitis_diagnosis_self_study_from_cdc.html. Accessed January 9, 2017.
2. Lowe S, Saxe JM. *Microscopic procedures for primary care providers*. Philadelphia, PA: Lippincott Williams & Wilkins; 1999.
3. Centers for Disease Control and Prevention. CLIA law and regulations. Available at: https://wwwn.cdc.gov/CLIA/Regulatory/default.aspx. Accessed January 9, 2017.
4. Centers for Disease Control and Prevention. CDC STI slide set. Available at: https://www2a.cdc.gov/stdtraining/ready-to-use/Manuals/Vaginitis/vaginitis-slides-2013.pptx. Accessed April 1, 2017.
5. Centers for Disease Control and Prevention. Sexually transmitted disease treatment guidelines, 2015. Available at: https://www.cdc.gov/std/tg2015/default.htm. Accessed January 9, 2017.
6. Association of Public Health Laboratories. Recommendations for the laboratory-based detection of *Chlamydia trachomatis* and *Neisseria gonorrhea* - 2014. Available at: https://www.aphl.org/programs/infectious_disease/std/Pages/N-gonorrhoeae-and-C-trachomatis.aspx. Accessed July 19, 2017.
7. Hoda RS, Loukeris K, Abdul-Karim FW. Gynecologic cytology on conventional and liquid-based preparations: a comprehensive review of similarities and differences. *Diagn Cytopathol.* 2013;41(3):257-278. doi:10.1002/dc.22842.
8. Martin-Hirsch PL, Jarvis GG, Kitchener HC, Lilford R. Collection devices for obtaining cervical cytology samples. *Cochrane Database Syst Rev.* 2011;4.
9. Davis-Devine S, Day SJ, Anderson A, et al. Collection of the BD SurePath Pap Test with a broom device plus endocervical brush improves disease detection when compared to the broom device alone or the spatula plus endocervical brush combination. *CytoJournal.* 2009;6:4. Available at: http://www.cytojournal.com/text.asp?2009/6/1/4/45495. Accessed January 9, 2017.
10. Marchand L, Mundt M, Klein G, Agarwal SC. Optimal collection technique and devices for a quality Pap smear. *Wisc Med J.* 2005;104(6):51-55.

CHAPTER

5

Health Promotion Across the Lifespan

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Introduction

Health promotion is the process of enabling individuals to increase control over and improve their health. Providing a person with the tools necessary to maintain optimal health is the essence of health promotion, and is done primarily through counseling, education, and shared decision making within a therapeutic relationship. Maintaining optimal health also requires the conduct of specific screening at various intervals throughout life to ensure early detection of the onset of disease states. This chapter provides information about primary and secondary health promotion and disease prevention services that aid in promoting and maintaining health for women across the lifespan. Reproductive life planning includes both primary and secondary prevention strategies and, therefore, is included in a final section. Education and counseling that are recommended for a preconception care visit are reviewed in **Appendix 5A**.

The Midwife's Role in Health Promotion

Health promotion is an essential component of midwifery practice. The International Confederation of Midwives' (ICM) *International Definition of the Midwife* articulates the key role that midwives play in health education and counseling for women, their families, and the community.[1] Similarly, the American College of Nurse-Midwives (ACNM) has identified that wellness counseling, health promotion, and disease prevention are part of the scope of practice of certified nurse-midwives (CNMs) and certified midwives (CMs).[2] Both ACNM and ICM address elements of health promotion and disease prevention in their essential/core competencies for entry-level midwifery practice,[3,4] and ACNM recognizes health promotion, disease prevention, and health education as hallmarks of the art and science of midwifery.[3]

One of the roles of the midwife is to help individuals in becoming empowered to be active participants in maintaining their health.[3,4] Furthermore, aiding women to become empowered to maintain their health and the health of their families, within the context of the communities in which women live, requires that midwives remain aware of the health status of the communities and advocate for policies that promote healthy communities.

Definition of Terms

Participating in health promotion and health maintenance first requires an understanding of key concepts and terms.

Health is defined in a number of ways, based largely on the context in which it is used. The World Health Organization (WHO) defines *health* as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity."[5] The Centers for Disease Control and Prevention (CDC) defines *health* as "a human condition with physical, social, and psychological dimensions, each characterized on a continuum with positive and negative poles. Positive health is associated with a capacity to enjoy life and withstand challenges; it is not merely the absence of disease. Negative health is associated with illness, and in the extreme, with premature death."[6] If health is conceptualized as occurring along a continuum, the goal of the midwife in health promotion is to identify each individual's health

status and provide the preventive services necessary to keep that status moving in a positive direction.

Disease, for the purpose of this chapter, is defined as any disruption in the physical, emotional, or social functioning of a human being that interferes with that person's capacity to maintain optimal physical, emotional, or social health.

Primary prevention refers to the delivery of healthcare services that focus on preventing disease from occurring in a given population and in individuals.[7] Examples of primary preventive services include immunizations, counseling, and education. At the population/community level, primary preventive services include the implementation of policies that promote clean air, clean water, and workplace safety.

Secondary prevention refers to the delivery of healthcare services aimed at early detection of disease states as well as interventions that limit severity and resulting morbidity.[7] Identification of risk factors through a thorough health history and the conduct of regular screening tests at appropriate intervals in a woman's life are perhaps the most powerful secondary preventive services provided by midwives.

Tertiary prevention refers to the delivery of healthcare services that restore optimal function, improve health status, and limit long-term disability following the identification of disease.[7] Although midwives are involved in the delivery of tertiary preventive services (the treatment of women with diseases), this chapter focuses on the midwife's role in the provision of primary and secondary preventive services for individual women. Delivery of tertiary preventive services is addressed in other chapters throughout this text.

Screen is a term that can be used as a noun or a verb. As a verb, midwives screen women for certain disease states as a component of secondary prevention. As a noun, it is the screen that is evaluated during testing. Readers will see mention of screening tests and diagnostic tests throughout this text. In general, *screening tests* are laboratory (or other) tests conducted on asymptomatic, healthy women for the purpose of early detection of health problems. *Diagnostic tests* are laboratory (or other) tests conducted to confirm a diagnosis, often following a positive screen.

Health Promotion: Primary Prevention

Preventing the onset of chronic disease through primary preventive strategies is the leading goal of health promotion. The United States spends more money on health care than any country in the world.[8] In 2015, an average of $9990 per person was spent on health care, representing approximately 18% of the U.S. gross domestic product.[9] Despite the fact that healthcare spending in the United States far exceeds that of other nations, the United States has poorer health outcomes on most measures than any other developed nation.[8] Reasons for the rapid growth in U.S. healthcare spending are multifaceted, although research suggests that the costs associated with the treatment of individuals with chronic diseases are a key contributor.[8-10]

The causes of poor health outcomes in the United States are equally complex. Public health leaders have identified the lack of attention paid to health promotion as a primary contributor.[8,9] Chronic diseases, many of which are preventable, are currently the leading cause of death and disability for adults in the United States. An estimated 50% of adults living in the United States have one or more chronic health problem.[11] Rising rates of chronic disease include conditions that are largely consequences of modifiable health risk behaviors such as poor nutrition and sedentary lifestyle.[11] Associated conditions such as type 2 diabetes, hypertension, and heart disease place a financial burden on healthcare delivery systems and increase the risk of decreased quality of life and premature death for the individual.[8-11] A substantial body of evidence demonstrates that increasing preventive health services to improve population health can lead to reduced healthcare spending,[8,9] increased productivity, and improvements in quality of life.[8-11]

Education and Counseling

The leading causes of death in women—which include heart disease, cancer, stroke, chronic respiratory disease, and diabetes[12]—are strongly associated with lifestyle-related behaviors. In turn, modifying behaviors such as inactivity, poor dietary habits, and tobacco use can often reduce a woman's risk for disease-related morbidity and early death. To achieve this goal, midwives and other healthcare providers use counseling and education aimed at improving personal health practices as tools for health promotion.[13] It is important to implement these interventions in such a way that the counseling and education are meaningful to each individual.[14] An individualized education and counseling plan is based primarily on the findings of a thorough health history and physical examination during which the midwife identifies the health risks that need to be addressed.

Learning Theory

Most persons are unlikely to benefit from provider-driven teaching, in which the midwife decides what the individual needs to know and when that person needs the information. Instead, all persons are more likely to understand and implement the information provided during a teaching/counseling session when the learning is person centered—in other words,

when that person has identified a need to know and demonstrates a readiness to learn, and when the learning builds on prior experience(s).

The principles of adult learning provide a framework within which health education can be customized to meet each woman's needs. The primary goal of adult learners is to acquire skills and resources that can be applied in their daily lives, rather than simply to acquire information. Adolescents who have begun to take responsibility for making their lifestyle decisions will also benefit from the self-directed learning that may be facilitated using the principles of adult learning, as summarized in **Table 5-1**.[15] For adults, learning is a process for which the "teacher" becomes a facilitator.

Table 5-1	**Principles of Adult Learning Theory**
Adults need to know why they need to learn something before learning it.	
The self-concept of adults is heavily dependent upon a move toward self-direction.	
Prior experiences of the learner provide a rich resource for learning.	
Adults typically become ready to learn when they experience a need to cope with a life situation or perform a task.	
Adults' orientation to learning is life centered; education is a process of developing increased competency levels to achieve their full potential.	
The motivation for adult learners is internal rather than external.	

Based on Knowles MS, Holton EF, Swanson RA. *The Adult Learner: The Definitive Classic in Adult Education and Human Resource Development.* 8th ed. New York, NY: Routledge; 2015.[15]

Recognizing the external motivators for adult learners is an important first step in establishing a health promotion plan. By investigating *why* the person wants to change behavior —that is, what she or he deems to be the benefit of behavior change—the midwife gains valuable insight into *which types* of interventions will be most effective in helping someone achieve their goals. The person's talents and skills, combined with their existing beliefs, can be incorporated into the approach used to achieve behavior change. For example, when establishing a plan to increase physical activity, it is important to understand that adolescents may be interested in skill acquisition, helping others, making friends, or physical appearance. Women of all ages may view physical activity as an outlet for stress; as a way to lose weight, prevent weight gain, and tone muscles; as a way to maintain or increase physical strength; as a means by which to meet new friends; or as the best prevention for diseases such as diabetes or hypertension. Within the parameters of these motivations, the midwife can work with each individual to identify types of exercise or activity that are likely to be incorporated into that person's life.

An important component of any teaching and counseling plan is the provision of anticipatory guidance. The advent of the Internet and today's ready access to multiple sources of health information have led to a wide range of personal knowledge about health. Some individuals will be well informed but others do not have access to health information. It is, therefore, incumbent upon the midwife to anticipate the healthcare needs of each person (based on risk factors) and provide individualized counseling about appropriate health services across the lifespan.

Content to Address During Counseling and Education

After determining how to present the information, the next important step is to identify which content to address during a given clinical encounter. Again, using the principles of adult learning, this step is completed with the individual and the midwife acting in partnership. Women often present to a clinical encounter with specific questions for their healthcare providers. In those instances, it is easy to design a teaching plan based on the explicitly expressed needs of the woman. Whether the woman presents for a lengthy appointment for a full physical examination or for a brief appointment and targeted examination for a specific concern, it is important for the midwife to honor (and respond to) the learning needs that are identified by the woman, especially when the time spent with the woman is limited.

Time is a precious commodity in a busy practice setting. When designing a teaching/counseling plan, only information about topics for which evidence of effectiveness exists should be included. Spending time on the delivery of care or services that are not evidence based means that less time can be devoted to care and services for which there is evidence of effectiveness. Moreover, in 2003, the Institute of Medicine issued a report recommending that all health professionals be educated to deliver patient-centered care with an emphasis on evidence-based practice.[16]

Evidence-Based Counseling Interventions

For the past 2 decades, healthcare providers have been strongly encouraged to participate in evidence-based practice (EBP), referring to the delivery of evidence-based care. One of the earliest definitions of evidence-based practice was "the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence-based medicine means integrating individual clinical expertise with the best available clinical evidence from systematic research."[17] ACNM has recognized the importance of providing care based on scientific knowledge of best practices and defines evidence-based practice as "the integration of the best research evidence with clinical expertise and client values."[18] Values and preferences include anything that makes an individual woman unique, such as religious and cultural beliefs, literacy level, and the woman's prior experience and knowledge.

U.S. Preventive Services Task Force

Professional organizations such as the American Congress of Obstetricians and Gynecologists and the American Cancer Society have been advancing recommendations regarding preventive services for several decades. However, it was not until the 1989 publication of *Guide to Clinical Preventive Services* by the U.S. Preventive Services Task Force (USPSTF) that healthcare professionals were provided with recommendations that were based on an unbiased review of scientific evidence. The mission of the USPSTF is "to improve the health of all Americans by making evidence-based recommendations about clinical preventive services and health promotion. These recommendations, which are developed for primary care clinicians are grounded in science, include screening tests, counseling about healthful behaviors, and preventive medications."[19] The recommendations of the USPSTF are considered the "gold standard" for clinical preventive services.[19]

Adopting evidence-based practices is not always easy. One of the fundamental ethical principles upon which healthcare delivery is based is the principle of nonmaleficence, or "doing no intentional harm." Consequently, accepting new evidence that suggests a previously accepted clinical practice may be harmful can be challenging. For example, in 2009, the USPSTF published new recommendations for breast cancer screening, which included a recommendation *against* teaching women without known risk factors for breast cancer to do breast self-examination: The scientific evidence revealed moderate certainty that the harms associated with teaching breast self-examination outweigh the benefits in this population.[20] Many midwives have been teaching breast self-examination since they began clinical practice and can cite examples of women who were adamant that their lives were saved because they found a mass during self-examination. However, implementing evidence-based preventive services requires that midwives establish individualized plans for health promotion that are based on best evidence rather than what they "believe" is best practice. At best, time spent teaching breast self-examination is an inefficient use of time that could have otherwise been spent on the delivery of care that is evidence based; at worst, time spent teaching breast self-examination may actually be harmful.

To aid clinicians in selecting evidence-based approaches, the USPSTF provides recommendations for clinical preventive services, including counseling interventions. The USPSTF recommendations reflect the strength of the available evidence and the magnitude of net benefit from the intervention (i.e., benefit minus harms). Professionals may find minor differences in the grading of evidence by the USPSTF based on the year in which the recommendations were made. Recommendations from 2012 to the present (**Table 5-2**) are based on categories that were slightly modified from the pre-2007 scale.[21] Grades A and B remain interventions that should be offered to women. **Table 5-3** identifies the level of certainty regarding the net benefits associated with healthcare practices.[21] Both the grading scale and level of certainty are used to evaluate specific therapeutic interventions or techniques.

Table 5-2 **U.S. Preventive Services Task Force Grade Definitions After July 2012**

Grade	Definition	Suggestions for Practice
A	The USPSTF recommends the service. There is high certainty that the net benefit is substantial.	Offer or provide this service.
B	The USPSTF recommends the service. There is high certainty that the net benefit is moderate or there is moderate certainty that the net benefit is moderate to substantial.	Offer or provide this service.
C	The USPSTF recommends selectively offering or providing this service to individual patients based on professional judgment and patient preferences. There is at least moderate certainty that the net benefit is small.	Offer or provide this service for selected patients depending on individual circumstances.
D	The USPSTF recommends against the service. There is moderate or high certainty that the service has no net benefit or that the harms outweigh the benefits.	Discourage the use of this service.
I Statement	The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of the service. Evidence is lacking, of poor quality, or conflicting, and the balance of benefits and harms cannot be determined.	Read the clinical considerations section of USPSTF Recommendation Statement. If the service is offered, patients should understand the uncertainty about the balance of benefits and harms.

Abbreviation: USPSTF, U.S. Preventive Services Task Force.

Reproduced with permission from U.S. Preventive Services Task Force. Grade definitions. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Name/grade-definitions. Accessed June 5, 2017.[21]

Table 5-3 **Levels of Certainty Regarding Evidence of Net Benefit**

Level of Certainty[a]	Description
High	The available evidence usually includes consistent results from well-designed, well-conducted studies in representative primary care populations. These studies assess the effects of the preventive service on health outcomes. This conclusion is therefore unlikely to be strongly affected by the results of future

	studies.
Moderate	The available evidence is sufficient to determine the effects of the preventive service on health outcomes, but confidence in the estimate is constrained by such factors as: The number, size, or quality of individual studies. Inconsistency of findings across individual studies. Limited generalizability of findings to routine primary care practice. Lack of coherence in the chain of evidence. As more information becomes available, the magnitude or direction of the observed effect could change, and this change may be large enough to alter the conclusion.
Low	The available evidence is insufficient to assess effects on health outcomes. Evidence is insufficient because of: The limited number or size of studies. Important flaws in study design or methods. Inconsistency of findings across individual studies. Gaps in the chain of evidence. Findings not generalizable to routine primary care practice. Lack of information on important health outcomes. More information may allow estimation of effects on health outcomes.

[a] The USPSTF defines certainty as "likelihood that the USPSTF assessment of the net benefit of a preventive service is correct." The net benefit is defined as benefit minus harm of the preventive service as implemented in a general, primary care population. The USPSTF assigns a certainty level based on the nature of the overall evidence available to assess the net benefit of a preventive service.

Reproduced with permission from U.S. Preventive Services Task Force. Grade definitions. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Name/grade-definitions. Accessed June 5, 2017.[21]

Although the USPSTF has provided many recommendations to implement preventive services that are evidence based (A and B grade services), there remain some services for which there is insufficient evidence to make a recommendation (I grade) and some services for which there is a moderate degree of certainty that the net benefit is small (C grade). Decisions to use those services are reached through shared decision making, in which the midwife and the individual decide together whether to use a given service. When these services are used, the recipient of care should be informed about the lack of available evidence (or conflicting evidence) that supports use of the service.

The USPSTF recommendations are frequently updated. As new evidence regarding preventive health services emerges, recommendations are modified to reflect the current body of evidence. Presented in this chapter are the major counseling interventions recommended by the USPSTF, at the time of this text's publication. Readers are advised to regularly review the USPSTF website for updates to these recommendations, and for the addition of new recommendations for preventive services. Links to this website are provided in the Resources section at the end of the chapter.

Behavioral Counseling Interventions

Behavioral counseling interventions include education, counseling, and other interventions provided in primary care settings that are focused on assisting individuals to adopt new behaviors, change existing behaviors that lead to poor health outcomes, and maintain behaviors

that improve health status.[22] Most of the counseling interventions that are recommended for women involve the implementation of some type of lifestyle change or avoidance of risky behaviors.

Alcohol Misuse

Alcohol is the third leading cause of preventable deaths in the United States,[23] and diseases caused by alcohol misuse, including fetal alcohol syndrome, are preventable. Risks associated with excessive alcohol use in women include liver disease, heart disease, brain damage, some forms of cancer, sexual assault, unintended pregnancy and sexually transmitted infections, accidental injury, and premature death. According to the CDC, 53.6% of nonpregnant women and 10.2% of pregnant women use alcohol.[24] An estimated 18.2% of nonpregnant women and 3.1% of pregnant women report participating in episodic (binge) drinking, which the CDC defines as the consumption of more than three alcoholic beverages on one occasion (for women).[24] For women who are not pregnant, an occasional drink is generally considered safe. However, no level of alcohol use is considered safe during pregnancy. Complete abstinence from consuming alcohol is recommended for women who are pregnant or trying to become pregnant.[24,25] For pregnant women, any consumption of alcohol may cause fetal alcohol syndrome, other disorders along the fetal alcohol spectrum, and/or other adverse fetal/neonatal outcomes.[24,25]

The USPSTF uses the term “alcohol misuse” to describe a range of behaviors that includes risky or hazardous drinking, harmful alcohol use, alcohol abuse and alcohol dependence.[24] Screening for alcohol misuse and provision of counseling interventions to reduce alcohol misuse is recommended for all adults in all primary care settings. To date, the USPSTF has found insufficient evidence to recommend for or against screening and counseling interventions for adolescents.[24]

Levels of drinking behavior are also defined by various groups or organizations. For example, the *Dietary Guidelines for Americans* (developed by the U.S. Department of Health and Human Services and the U.S. Department of Agriculture) define moderate drinking as “up to 1 drink per day for women and up to 2 drinks per day for men.”[25] Binge drinking is defined by the National Institute on Alcohol Abuse and Alcoholism as alcohol consumption that results in a blood alcohol concentration to 0.08 g/dL, which typically happens after consumption of 5 drinks for men and 4 drinks for women over a 2-hour time period.[25]

These definitions also illustrate the difference between screening and assessment: The purpose of *screening* is to identify persons who may have a problem with alcohol misuse based on their level of drinking behavior, while the purpose of *assessment* is to identify the extent of the alcohol problem for persons who have a positive screen.[26] As primary care providers, the recommendation for midwives is to periodically screen all clients and provide a brief intervention to reduce alcohol misuse; women who continue to misuse alcohol may be alcohol dependent and may need to be referred for further assessment and treatment.[24,26]

Ideally, screening for alcohol misuse should take place in settings that allow for assessment and follow-up of persons with a positive screen. Midwives who work in settings without

resources for follow-up care will need to identify healthcare providers in the community (or surrounding area) to whom individuals with a positive screen can be referred.

Screening for alcohol misuse begins by asking the woman how much alcohol she drinks. If the woman indicates that she never consumes any alcohol, the screen is complete. Documentation in the medical record should include the finding of a negative screen for alcohol misuse and a plan to rescreen in one year. If the woman indicates that she consumes any alcohol, the screen continues using a reliable screening instrument.

The USPSTF recommends using the Alcohol Use Disorders Identification Test (AUDIT), Alcohol Use Disorders Identification Test—Consumption (AUDIT-C), or a single-question screen to assess for alcohol misuse in primary care settings.[23] The easiest of these instruments to administer is the single-question screen, in which the midwife simply asks a woman about how many times during the past year she consumed 4 or more drinks in one day; a response of one or more times in the last year is considered a positive screen and signals the need to complete the full AUDIT.[27] The full AUDIT screen is presented in **Table 5-4**.[28] The AUDIT-C (**Table 5-5**[29]) is an abbreviated version of the AUDIT and takes just 1 to 2 minutes to complete. After assuring the woman that the information she shares will remain confidential, the midwife can administer any of these instruments during the collection of a complete health history or separately for a brief assessment of alcohol misuse. As is true for the entire health history, it is important to remain nonjudgmental when asking questions about sensitive topics such as alcohol use.

Table 5-4 **The Alcohol Use Disorders Identification Test (AUDIT)**

1. How often do you have a drink containing alcohol?
   - (0) Never (Skip to Questions 9–10)
   - (1) Monthly or less
   - (2) 2 to 4 times a month
   - (3) 2 to 3 times a week
   - (4) 4 or more times a week
2. How many drinks containing alcohol do you have on a typical day when you are drinking?
   - (0) 1 or 2
   - (1) 3 or 4
   - (2) 5 or 6
   - (3) 7, 8, or 9
   - (4) 10 or more
3. How often do you have six or more drinks on one occasion?
   - (0) Never
   - (1) Less than monthly
   - (2) Monthly
   - (3) Weekly
   - (4) Daily or almost daily
6. How often during the last year have you been unable to remember what happened the night before because you had been drinking?
   - (0) Never
   - (1) Less than monthly
   - (2) Monthly
   - (3) Weekly
   - (4) Daily or almost daily
7. How often during the last year have you needed an alcoholic drink first thing in the morning to get yourself going after a night of heavy drinking?
   - (0) Never
   - (1) Less than monthly
   - (2) Monthly
   - (3) Weekly
   - (4) Daily or almost daily
8. How often during the last year have you had a feeling of guilt or remorse after drinking?
   - (0) Never
   - (1) Less than monthly
   - (2) Monthly

4. How often during the last year have you found that you were not able to stop drinking once you had started?
   (0) Never
   (1) Less than monthly
   (2) Monthly
   (3) Weekly
   (4) Daily or almost daily
5. How often during the last year have you failed to do what was normally expected from you because of drinking?
   (0) Never
   (1) Less than monthly
   (2) Monthly
   (3) Weekly
   (4) Daily or almost daily
9. Have you or someone else been injured as a result of your drinking?
   (0) No
   (2) Yes, but not in the last year
   (4) Yes, during the last year
10. Has a relative, friend, doctor, or another health professional expressed concern about your drinking or suggested you cut down?
   (0) No
   (2) Yes, but not in the last year
   (4) Yes, during the last year

Add up the points associated with answers. A total score of 8 or more indicates harmful drinking behavior (a positive screen).

Reproduced with permission from Substance Abuse and Mental Health Services Administration, HRSA Center for Integrated Health Solutions. The Alcohol Use Disorders Identification Test (AUDIT). Available at: http://www.integration.samhsa.gov/clinical-practice/screening-tools#drugs. Accessed June 8, 2017.[28]

Table 5-5 **AUDIT-C Questionnaire**

1. How often do you have a drink containing alcohol?
   a. Never
   b. Monthly or less
   c. 2 to 4 times a month
   d. 2 to 3 times a week
   e. 4 or more times a week
2. How many standard drinks containing alcohol do you have on a typical day?
   a. 1 or 2
   b. 3 or 4
   c. 5 or 6
   d. 7 to 9
   e. 10 or more
3. How often do you have six or more drinks on one occasion?
   a. Never
   b. Less than monthly
   c. Monthly
   d. Weekly
   e. Daily or almost daily

The AUDIT-C is scored on a scale of 0–12. For each question, a = 0 points; b = 1 point; c = 2 points; d = 3 points; e = 4 points.

In women, a score of ≥ 3 is considered a positive screen.

Reproduced with permission from Substance Abuse and Mental Health Services Administration, HRSA Center for Integrated Health Solutions. AUDIT-C Overview. 2014. Available at: http://www.integration.samhsa.gov/clinical-practice/screening-tools#drugs. Accessed June 8, 2017.[29] Scoring matrix modified with permission from Substance Abuse and Mental Health Services Administration.

Most women who use alcohol do so within the limits that are considered safe. For these women, the goal of behavioral counseling is to prevent the onset of risky or hazardous drinking. Counseling should include reminders about safe use limits and recommendations for abstinence when necessary (such as when pregnant or planning a pregnancy, or when medication use or other health conditions warrant abstinence).[26] Documentation in the health record should include the finding of a negative screen for alcohol misuse and plans to rescreen in one year.

The USPSTF recommends behavioral counseling interventions for women with a positive screen for risky or hazardous drinking and referral for further assessment and treatment for women in whom alcohol abuse or dependence is suspected.[23] For women with a positive screen, several counseling interventions are available that are designed for use in primary care settings. The brief intervention recommended by the National Institute on Alcohol Abuse and Alcoholism[26] is based on the *5 A's* construct (assess, advise, agree, assist, arrange), as outlined in **Table 5-6**.[30] The same approach may also be of value when identifying the use of other substances separate from or in addition to alcohol. Additional resources for clinicians who conduct brief interventions in clinical settings are provided in the Resources section at the end of this chapter.

Table 5-6	5 A's for Alcohol Misuse

Assess or ask the person about alcohol use and desire to change behavior.

Advise the person to change risky behavior using a personalized approach that includes information about the harmful effects of alcohol use and benefits of changing behavior.

Agree upon treatment goals and approaches to changing behavior.

Assist the person in achieving agreed-upon goals by providing education, support and encouragement, and formal counseling when necessary. This may include helping the individual identify sources of social support for change.

Arrange follow-up visits for ongoing assistance with behavioral change and referral to treatment when necessary.

Note: Although these questions have been developed to conduct a brief intervention for alcohol misuse, they can be modified for use with brief interventions to aid women with other behavioral changes such as tobacco use or weight loss.

Based on Whitlock EP, Orleans CT, Pender N, Allan J. Evaluating primary care behavioral counseling interventions: an evidence-based approach. *Am J Prev Med.* 2002;22(4):267-284.[30]

Informing a woman about the finding of a positive screen and the need for referral must be done with sensitivity. The best approach is to tell the woman that her responses to the screening questions raise a concern that her alcohol use may be harmful and that she would benefit from meeting with a provider who specializes in alcohol counseling. For example, the

midwife might address the referral in the following way:

> *One of the things we know about alcohol is that it can cause serious health problems for women who drink more than what is considered safe. For women, having more than seven drinks per week or more than three drinks on one occasion is considered "risky or hazardous" drinking.*[23,26] *Based on your response to the questions I asked about alcohol use, your use of alcohol may fall into the "harmful" category. It may be that this is not the case, but just to be sure, I would like you to see one of my colleagues who is very skilled at assessing alcohol use and at helping women make changes in their alcohol use before it has a negative effect on their health.*

Documentation in the woman's health record includes the finding of a positive screen for alcohol misuse, a description of the behavioral counseling intervention that was provided and/or referrals that were made, and the plan for return visits for ongoing support.

Breastfeeding

The benefits of breastfeeding for women and infants are well documented. Although breastfeeding rates are increasing,[31] the United States has not yet met the *Healthy People 2020* goals for infant feeding, which include increasing the proportion of infants who are ever breastfed to 81.9%, the number of infants breastfed at 6 months to 60.6%, and the number of infants breastfed at 1 year to 34.1%.[32]

Interventions in primary care settings to promote breastfeeding result in increased rates of breastfeeding initiation as well as increased duration of breastfeeding. Therefore, the USPSTF recommends interventions during pregnancy and the postpartum period to promote breastfeeding and provide ongoing support for women who choose to breastfeed.[33] The most effective interventions use combined approaches to breastfeeding support, such as promoting the benefits of breastfeeding, providing practical advice and formal education for women and families, direct support, health professional training in breastfeeding support, and peer support and counseling.[33] Breastfeeding education and counseling is reviewed in the *Prenatal Care* and *Breastfeeding and the Mother–Newborn Dyad* chapters.

Dental Health

Dental health is often not considered by primary care providers, yet dental disease can have profound adverse effects on health such as increasing risk of cardiovascular disease among all individuals and increasing the risk of preterm birth for women who are pregnant. The last USPSTF recommendations for counseling interventions to prevent dental disease were established in 1996. Those recommendations were to counsel individuals to floss and brush their teeth daily with a fluoride-containing toothpaste and to regularly see a dental provider.[34] Since that time, there has been no new evidence regarding the primary provider's role in promoting oral health, so the USPSTF has elected to maintain the 1996 recommendation and

not modify it.[35] Recommendations of the CDC aimed at maintaining good oral health include drinking fluoridated water, brushing teeth with fluoridated toothpaste and flossing, visiting the dentist regularly, not using tobacco, limiting alcoholic beverages, maintaining control of diabetes and other chronic diseases, avoiding medications that cause dry mouth, and drinking plenty of water if dry mouth cannot be avoided.[36]

Dietary Counseling

The benefits of healthy eating and the risks associated with an unhealthy diet are well documented. Of the 10 leading causes of death in women, four are related to diet—namely, heart disease, certain forms of cancer, stroke, and diabetes.[12] Assessing the dietary counseling needs of individual women first requires the collection of a 24- to 48-hour recall diet history to evaluate nutritional and caloric intake, determination of height and weight for calculation of body mass index (BMI), and obtaining a health history for identification of risk factors that may be related to diet.

A BMI of less than 18.5 is considered "underweight"; a BMI in the range 18.5–24.9 is considered "normal"; a BMI in the range 25–29.9 is considered "overweight"; and a BMI of 30 or higher is considered "obese."[37] Women whose weight is categorized as underweight, overweight, or obese are at increased risk for weight-related morbidity and mortality. Poor dietary intake is a modifiable risk factor for several diseases, including certain forms of cancer and coronary artery disease. As such, improving dietary intake can result in improvements in overall health status, especially for women with additional risk factors (e.g., hypertension, dyslipidemia, impaired fasting glucose, metabolic syndrome, and family history) for diet-related diseases.[38] The USPSTF also recommends screening all women for obesity (using BMI) and offering women with a BMI of 30 or higher intensive, multicomponent behavioral interventions.[39] Intensive behavioral counseling interventions that address diet and physical activity are also recommended for adults who are overweight or obese and who have additional risk factors for cardiovascular disease.[38] The decision to offer diet counseling to women who are not obese and do not have risk factors for coronary artery disease should be made on an individual basis based on the healthcare needs of the woman.[39]

The *Nutrition* chapter contains a detailed description of dietary recommendations and healthy eating. Similar to the brief intervention for alcohol misuse, the 5 A's construct can provide a framework for behavioral counseling interventions for women who are overweight or obese.[40] The 5 A's for alcohol misuse can be modified for use with women who are overweight or obese. Midwives may also consider referral to programs or clinicians who specialize in intensive, counseling interventions.[38] Documentation in the health record should include findings of the dietary assessment, health risk assessment, BMI, and a description of the counseling and/or referral that was provided.

Genetic Assessment for Cancer Risk

Breast cancer is a leading cause of death for women in the United States. In 2014, nearly 237,000 women were diagnosed with breast cancer in the United States, and more than 41,000

women died from breast cancer in that same year.[41] The two most influential risk factors for breast cancer are gender and age; most breast cancers are discovered in women who are 50 years or older. Another important risk factor for breast cancer is a harmful mutation of the breast cancer susceptibility genes (*BRCA1* or *BRCA2*).[41] Mutations of the *BRCA1* and *BRCA2* genes are primarily associated with significant increases in risk for breast, ovarian, and fallopian tube cancers, but are also related to other forms of cancer.[42]

Collection of a complete health history should include a thorough assessment of a woman's family history, including her family's history of cancer. More specifically, a thorough family history includes identification and documentation of which family members were affected, the specific type of cancer and cancer site, age at diagnosis and gender of affected family members, and any family members with more than one type of cancer.[42] Seven findings in a family history are associated with an increased likelihood of harmful BRCA mutations. Findings associated with an increased risk for BRCA mutation include a family history of (1) breast and ovarian cancer, (2) breast cancer diagnosis prior to age 50 years, (3) bilateral breast cancer, (4) multiple cases of breast cancer in the family, (5) breast cancer in one or more male family members, (6) one or more family members with two types of BRCA-related cancer, and (7) Ashkenazi Jewish ethnicity.[42] Women whose family history includes one or more of these findings should be offered additional screening for genetic risks.

One of several familial risk stratification tools that can reliably detect women who should be offered genetic counseling can be used.[42] Previously validated screening tools include the Ontario Family History Assessment Tool, Manchester Scoring System, Referral Screening Tool, Pedigree Assessment Tool, and the Family History Screen—7 (FHS-7). The easiest of these instruments to use in primary care settings are the Referral Screening Tool and the FHS-7.[42] Both tools can be found online, and the links are included in the Resources section at the end of this chapter. The USPSTF further recommends that women with a positive screen be referred to a healthcare provider who is trained to conduct genetic counseling for additional assessment and counseling and genetic testing if indicated and if the woman choses to obtain genetic testing.[42]

Midwives can initiate the process of screening and counseling as part of the well-women history and physical examination. Counseling should include reassurance that the findings of the health history provide a basic screen for cancer risk, and that although most women have one or more risk factors, most women do not develop cancer.[41] Documentation in the health record should include findings related to the family history and, if used, findings from the validated screening tool as well as a description of the counseling and referral that was provided for women with a positive screen. A detailed discussion about BRCA screening can be found in the *Breast Conditions* chapter.

One of the challenges in the many steps of screening and counseling about BRCA-related mutations can be the (lack of) availability of trained genetic counselors. Furthermore, even when genetic counseling is available, this service may not be covered by insurance and may present a significant cost for women whose initial screening indicates the need for referral. Midwives are advised to familiarize themselves with the availability and costs associated with genetic counseling services. These services include those available in the woman's

community and surrounding areas as well as genetic counseling services available by telephone and online, which are recommended for women who would otherwise lack access to genetic counseling services.[42] A link to genetic counseling resources can be found in the Resources list at the end of this chapter.

Injury Prevention

Unintentional injury is the sixth leading cause of death in women in the United States.[12] Injuries related to motor vehicle crashes are the leading cause of death for individuals age 1–54 years.[43] Although such injuries are usually related to accidents and, therefore, are difficult to control, the USPSTF recognizes several interventions that can effectively limit the risk for unintentional injury. There is insufficient evidence to assess the balance of benefits and harms of counseling individuals about proper use of seat belts and child safety seats, however.[43]

One reason for this equivocation is that legislative action and community-based intervention, combined with counseling in the primary care setting, have already led to substantial increases in the number of persons in the United States who properly use motor vehicle restraints.[44] Approximately 87% of front-seat occupants now use seat belts, and the rate of seat belt use for rear-seat occupants is estimated at 78%.[44] Whether regularly counseling women in primary care settings would increase proper use rates even further is largely unknown. Nevertheless, the USPSTF and the CDC recognize that harms from counseling about proper motor vehicle safety restraints are minimal to absent.[45]

Improved seat belt use and reduced rates of alcohol-impaired driving would likely have the greatest effect in lowering rates of motor vehicle fatalities.[44] Therefore, counseling women to use motor vehicle restraints properly is likely to cause no harm and may lead to further reductions in motor vehicle–related injury and death. The CDC recommends that healthcare providers counsel parents and caregivers of children about the importance of using age- and weight-appropriate car seats and restraints at all times. The CDC also recommends providing resources about safe teen driving for parents of teenagers.[43]

Proper use of adult restraints includes wearing the seat belt at all times and making sure that the lap and shoulder belts are properly positioned across the pelvis and ribcage. The shoulder belt should be placed across the middle of the chest and away from the neck; the lap belt should be secured below the stomach and across the hips.[46] Women who are pregnant should be advised to wear a seat belt with the shoulder strap laying across the chest (between the breasts) and away from the neck. The lap belt should be secured below the abdomen so that it fits snugly across the hips and pelvic bone, as illustrated in **Figure 5-1**. Pregnant women should be further advised to adjust the seat as far back as possible, such that the foot pedals can be comfortably operated and at least 10 inches is maintained between the chest and the steering wheel.[47]

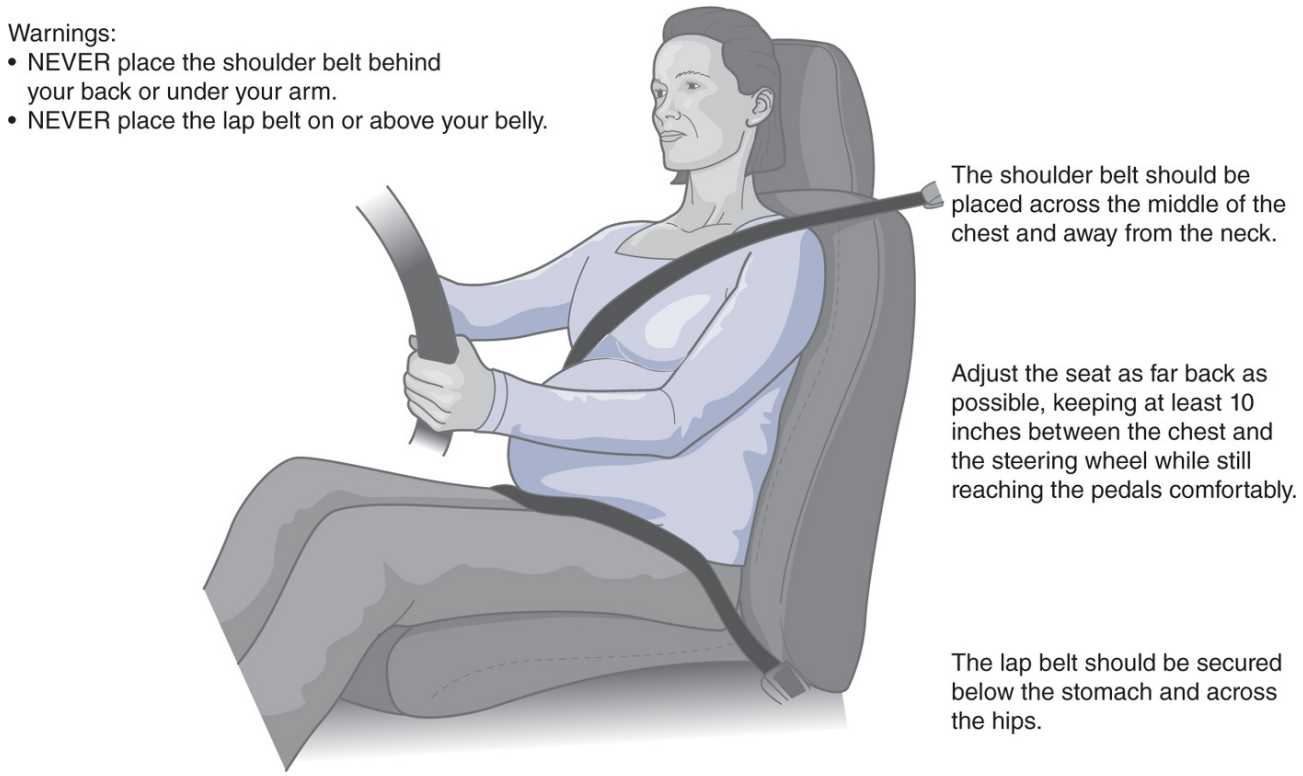


Figure 5-1 Proper placement of seat belt for women during pregnancy.

One third of automobile accident–related deaths in the United States involve drunk driving, and there are no identified harms associated with counseling about driving and drinking.[43] Counseling to reduce the incidence of driving under the influence of alcohol or other drugs is similar to counseling about the use of motor vehicle safety restraints. One approach is to include information about "drinking and driving" in behavioral counseling interventions for individuals who screen positive for alcohol misuse.[24] Counseling should include a discussion about determining a designated driver and establishing a plan for alternative transportation when in the company of an impaired driver.

Distracted driving is a relatively newly identified phenomenon, and interventions to prevent morbidity and mortality related to this practice have not been evaluated by the USPSTF. *Distracted driving* refers to driving while doing other things—most specifically, talking on a cell phone and sending/reading text messages. In 2015, nearly 3500 persons were killed and 391,000 persons were injured in the United States in motor vehicle accidents caused by distracted driving.[48] As is true for counseling about the dangers associated with drinking and driving, counseling about the dangers associated with distracted driving, with strong recommendations to refrain from talking on hand-held cell phones and sending/reading text messages, may result in fewer distracted driving–related automobile accidents.

For women age 65 years and older, falls are the leading cause of injury-related death and the most common cause of nonfatal injuries and trauma-related hospitalization.[49] The USPSTF recommends regular exercise or physical therapy and vitamin D supplementation for falls prevention in women age 65 years and older who are at increased risk for falls.[50] One way to assess a woman's fall risk is to ask her three questions:

1. Have you fallen in the past year?
2. Do you feel unsteady when standing or walking?
3. Do you worry about falling?

A "yes" response to one or more of the questions signals a positive screen.[51]

Recommendations for exercise include participation in weight-bearing exercises, leg-strengthening exercises, and exercises that improve balance (such as Tai Chi) at home or in group exercise classes.[50] The recommended dose of supplemental vitamin D for individuals at increased risk for falls is 800 IU per day.[50] Additional recommendations to reduce falls in women age 65 years and older include the identification of, and warning women about, medications that cause dizziness or drowsiness; reducing tripping hazards in the home, along with the addition of grab bars and improved lighting; and yearly eye examination with update of corrective lenses as needed.[51]

Physical Activity

Adequate physical activity is a factor in the prevention of heart disease, type 2 diabetes, obesity, infertility, some cancers, osteoporosis, osteoarthritis, falls, anxiety and depression, and premature death for both women and men.[52] During the collection of a complete health history, specific questions about activity interests and motivation can facilitate discussion about types of activities in which a person regularly participates. The midwife is then able to compile a comprehensive picture of nutrition status, activity levels, and lifestyle choices, as well as risk factors for certain diseases that may be minimized with physical activity.

Recommendations for counseling about physical activity are primarily based on risk factors. Intensive behavioral counseling interventions that include physical activity and diet are recommended for persons who are overweight or obese and who have additional risk factors for cardiovascular disease.[38]

Multicomponent recommendations for women with a BMI of 30 or higher and women who are overweight and have risk factors for cardiovascular disease include several behavioral management activities, such as goal setting, individual and group counseling and/or exercise sessions, making dietary and physical activity changes, recognizing and addressing barriers to change, and long-range planning to maintain lifestyle changes.[39] Although much of the counseling aimed at improving physical activity can begin with the primary care provider, the most effective interventions are delivered by specially trained professionals such as exercise professionals, physical therapists, or health educators.[39]

For women who are not obese and who do not have risk factors for cardiovascular disease, the USPSTF found a positive but small net benefit from exercise counseling conducted by primary care providers.[40] In the absence of risk factors for cardiovascular disease, the decision to provide exercise counseling is based on the individual woman's healthcare needs. Women who are ready to make behavioral changes are most likely to benefit from such counseling.[40]

To help women increase their physical activity level, it is necessary to know about the resources available in the community and to think creatively about ways to encourage effective

physical activity. While some women may have access to gyms or exercise studios with certified fitness professionals, many other women will prefer to use resources available in their neighborhood. Walking or running in the local neighborhood, along with participating in community, school, or workplace activity groups, may be among the most effective methods of increasing physical activity for most women. It is important that midwives understand the safety parameters of the communities in which they live and work so that they will be prepared to recommend safe spaces for women to exercise. When helping women with special needs (e.g., acute or chronic illnesses), knowledge of group exercise programs designed for individuals with specific health conditions can be invaluable.

For a woman with an established exercise routine, the goal is to support and encourage her participation in a beneficial exercise regimen. For women who participate in little or no physical activity, the challenge is to help them make small, incremental steps toward a more active lifestyle. Physical activity recommendations for women across the lifespan are presented in **Table 5-7**.[52] The CDC has additional information about various forms of physical activity, such as aerobic versus strength training, and ways to measure the intensity of physical activity.[53]

Table 5-7 Physical Activity Recommendations for Women Across the Lifespan

Age	Recommendation
Adolescents (age 17 years and younger)	≥ 60 minutes of moderate or vigorous aerobic activity each day Include muscle strengthening activities in the 60 minutes at least 3 days per week Include bone strengthening activities in the 60 minutes at least 3 days per week
Adults (age 18–64 years)	150 minutes[a] of moderate-intensity aerobic activity each week and muscle-strengthening activities that work all major muscle groups on ≥ 2 days per week OR 75 minutes[a] of vigorous-intensity aerobic activity each week and muscle-strengthening activities on ≥ 2 days per week OR An equivalent mix of moderate- and vigorous-intensity aerobic activity each week and muscle-strengthening activities that work all major muscle groups on ≥ 2 days per week
Older adults (age 65 years and older)[b]	150 minutes[a] of moderate-intensity aerobic activity each week and muscle-strengthening activities that work all major muscle groups on ≥ 2 days per week OR 75 minutes[a] of vigorous-intensity aerobic activity each week and muscle-strengthening activities on ≥ 2 days per week OR An equivalent mix of moderate- and vigorous-intensity aerobic activity each week and muscle-strengthening activities that work all major muscle groups on ≥ 2 days per week
Pregnant and postpartum women	150 minutes[a] of moderate-intensity aerobic activity per week Healthy women who participate in vigorous-intensity aerobic activity prior to pregnancy may continue to do so during and after pregnancy as long as they stay healthy and discuss their exercise activity with their healthcare provider.

[a] May be broken into increments as small as 10 minutes at a time.

[b] Who have no limiting health conditions and are generally fit.

Based on Centers for Disease Control and Prevention. Physical activity basics. 2015. Available at: https://www.cdc.gov/physicalactivity/basics/index.htm. Accessed July 11, 2017.[52]

Documentation in the health record should include findings of the health history, health risk assessment, and BMI. Documentation should also include a description of the counseling and/or referral that was provided and plans for follow-up.

Sexually Transmitted Infections and Risk for Unplanned Pregnancy

Sexually transmitted infections (STIs) are a preventable cause of morbidity and mortality in the United States, with an estimated 20 million new STIs diagnosed each year, almost half of which are diagnosed among individuals age 15–24 years.[54] Recognizing that all sexually active adolescents are at risk for STIs, the USPSTF recommends intensive behavioral counseling for STI prevention for all sexually active adolescents[54] and for all adults at increased risk for STIs. Women at risk for STIs include those with a current STI or an infection within the past year, women who have multiple sexual partners, women who do not consistently use condoms, and women who live in communities with high prevalence rates of any STI.[54]

The most effective counseling interventions are those that are delivered over the course of 2 or more hours, although sessions as brief as 30 minutes can be effective. Effective counseling sessions can be provided in group or individual settings, and delivered over single or multiple sessions. The most effective counseling interventions provide information about STIs and their transmission, assessment of STI transmission risk, and training in essential skills such as condom use, talking about safer sex, goal setting and problem solving.[54] The *Reproductive Tract and Sexually Transmitted Infections* chapter contains a detailed discussion of STIs and STI prevention.

The USPSTF has not made recommendations or addressed the prevention of unplanned pregnancy. However, the most recent data indicate that 45% of pregnancies in the United States are unintended, down from 51% in 2008.[55] To meet the *Healthy People 2020* goal of decreasing the rate of unintended pregnancy to 44%,[56] it is likely that some form of intervention in the primary care setting will be necessary. Prior to initiating a discussion about pregnancy prevention, the midwife asks the woman about her plans regarding pregnancy. A review of contraceptive options is conducted for women who are not planning a pregnancy. A detailed discussion of contraceptive methods is presented in the *Family Planning* chapter. For women who are planning a pregnancy, the midwife completes the implementation of the health promotion plan as described in this chapter with a focus on topics of particular import for pregnant women as reviewed in Appendix 5A. Documentation in the health record should include findings of the sexual health history and a description of the counseling and/or referral that was provided.

Skin Cancer Prevention

Skin cancer is the most common cancer in the United States. Basal and squamous cell carcinomas, which are the two most common forms, are highly curable.[57] By comparison,

melanomas, most of which are caused by ultraviolet (UV) light exposure,[57] result in more than 9000 deaths in the United States each year.[58] Fair-skinned adolescents and young adults through the age of 24 years should be counseled about minimizing their risk for skin cancer by decreasing exposure to UV light.[59] Because the lifetime risk for skin cancer is strongly linked to UV exposure early in life, the USPSTF was unable to determine the net effect of counseling individuals older than 24 years. However, it found little harm associated with such counseling and recommends that decisions to provide counseling about UV exposure be made based on risk factors.[59]

In addition to reviewing the risks for skin cancer associated with UV exposure and avoiding sun exposure between the hours of 10:00 a.m. and 4:00 p.m., use of a broad-spectrum sunscreen with a sun-protection factor (SPF) of 15 or more and use of protective clothing, a wide-brimmed hat, and sunglasses are recommended. All persons should also be counseled to avoid using indoor tanning facilities.[59,60]

Tobacco Use

The top four leading causes of death in women—heart disease, cancer, chronic respiratory disease, and stroke—can all be linked to tobacco use.[12] Although tobacco use among both women and men has declined over the past several decades, smoking remains the leading cause of preventable death in the United States.[61] Surpassing breast cancer in 1987, lung cancer is now the leading cause of cancer death in women in the United States.[61] In addition to lung and several other forms of cancer, the health risks associated with tobacco use include cardiovascular disease; diseases related to the endocrine system, including diabetes; menstrual dysfunction, including early menopause; osteopenia/osteoporosis; and premature death.[61]

Women who smoke during pregnancy are at increased risk for ectopic pregnancy and spontaneous abortion, preterm labor and birth, preterm premature rupture of membranes, placental abruption, placenta previa, intrauterine growth restriction, and perinatal mortality.[62-64] Infants born to women who smoke during pregnancy are at increased risk for neonatal death, sudden infant death syndrome (SIDS), and low birth weight.[62-64] Finally, environmental exposure to cigarette smoke on the part of nonsmokers—a phenomenon characterized as *secondhand* or *passive smoke*—contributes to early death in almost 41,000 nonsmoking adults and 400 infant deaths each year.[65] Some authorities are studying smoke retained in cars, rooms, and clothing, termed *thirdhand smoke*, to ascertain whether risks exist in those situations.

Smoking cessation at any age is associated with reductions in smoking-related disease and early death, and women who stop smoking before or during pregnancy can significantly reduce their risk for adverse pregnancy outcomes.[64] Therefore, primary preventive strategies focus on assisting current smokers to stop and reducing the number of individuals who have ever smoked.

Healthcare providers should ask all patients, including pregnant women, about tobacco use. Individuals who use tobacco are advised to stop and provided with behavioral interventions aimed at cessation of tobacco use.[64] It is also recommended that pharmacotherapy approved by the U.S. Food and Drug Administration (FDA) be provided for all nonpregnant women and

men who use tobacco. There is insufficient evidence to assess the balance of benefits and harms related to pharmacotherapy for smoking cessation in pregnant women.[64]

One of the most effective and efficient behavioral counseling interventions for women who use tobacco is based on the 5 A's construct (ask, advise, assess, assist, arrange), as shown in **Table 5-8**.[66] Convincing evidence indicates that even brief counseling interventions (lasting 3 to 10 minutes), either alone or in combination with pharmacotherapy, can effectively increase the number of smokers who quit and remain abstinent for one year.[64] Pharmacotherapy for smoking cessation includes nicotine replacement therapy (found in gums and lozenges) as well as the medications bupropion (Wellbutrin) and varenicline (Chantix). The effectiveness of and harms associated with these agents during pregnancy is not well documented.[62,63]

Table 5-8 **Modified 5 A's for Smoking Cessation**

Ask every woman about tobacco use.

Advise every woman who smokes to stop smoking and provide information about the risks associated with continued use.

Assess every woman's readiness and/or willingness to stop smoking. This includes identifying barriers and motivating factors to stop smoking.

Assist women to stop smoking. This includes setting goals to cut down/quit, providing support, identifying sources of social support, and dealing with motivational barriers.

Arrange for regular follow-up visits with the midwife and/or refer to additional healthcare providers for more intensive treatment if necessary.

Based on Agency for Healthcare Research and Quality. Five major steps to intervention (the "5 A's). 2017. Available at: https://www.ahrq.gov/professionals/clinicians-providers/guidelines-recommendations/tobacco/5steps.html. Accessed June 11, 2017.[66]

Smoking cessation interventions may be briefly reviewed in midwifery education programs. One survey of CNMs and CMs found that approximately 50% of the respondents were aware of the USPSTF guidelines and 5 A's intervention.[67,68] However, a recent pilot study found that midwife-delivered 5 A's smoking cessation was effective in decreasing the number of cigarettes smoked per day and increasing the number of women who stopped smoking during pregnancy.[69] Although this was a small pilot study, the findings indicate that use of the modified 5 A's smoking cessation program can be incorporated into midwifery practice.[62,68] In addition, it is essential to conduct this kind of counseling in a nonjudgmental way, to individualize counseling interventions during pregnancy, and to include information about health risks for both the woman and her child.[64]

Most women who smoke are fully aware of the health risks associated with smoking and experience feelings of guilt and shame about continuing the unhealthy behavior. These feelings are especially prevalent among women who smoke during pregnancy and among women who relapse after a cessation attempt. Counseling interventions for women are most effective when the relationship between the midwife and the woman is based on mutual trust and respect and when the intervention is woman centered. Rather than attempting to manipulate pregnant women to stop smoking by instilling feelings of guilt and fear about the fetal effects of smoking, a more effective approach is to engage women in an open and respectful dialogue, where the

primary focus of the discussion is on maintaining and promoting the personal health of the woman. Documentation in the health record should include findings of the health history regarding tobacco use, a description of the counseling and/or referral that was provided, and plans for follow-up.

Immunizations

Immunization against several disease states is a primary preventive strategy for women across the lifespan.[70],[71] General immunization recommendations from the CDC for adolescents, women of childbearing age, and women of perimenopausal/menopausal age are summarized in **Table 5-9**.[70] However, it is important to understand that these recommendations are frequently updated so that timing and doses may change. Among the changes in the revised 2018 recommendations will be additional information about vaccines in pregnancy. Readers are advised to consult the CDC website frequently for the current recommendations. A link to the CDC immunization schedules can be found in the Resources section at the end of this chapter. An app is available for clinicians who need this information at their fingertips. Midwives who administer immunizations are also encouraged to consult the relevant Advisory Committee on Immunization Practices (ACIP) statements, available on the CDC's website, for recommendation updates, special recommendations for women with immunocompromising conditions, and guidelines for vaccinations for healthcare workers, and travelers. Adverse events following vaccination should be reported to the Vaccine Adverse Event Reporting System (800-822-7967). Links to vaccine-related resources, including vaccine schedule apps, are provided in the Resources section at the end of this chapter.

Table 5-9 **Immunization Schedule for Women Across the Lifespan[a]**

Vaccine	Adolescents	Women Age 19–41 Years	Pregnant Women	Women Age ≥ 41 Years	Dose/Series, Timing and Other Considerations
Hib (*Haemophilus influenzae* type b conjugate vaccine)	Recommended only for adolescents with anatomic or functional asplenia (including sickle cell disease) or HIV infection who have not been previously immunized	Recommended only for women with certain immunocompromising conditions	Recommended only for women with certain immunocompromising conditions	1 or 3 doses depending on the indication for vaccination	See CDC guidelines for additional information.
HepA (hepatitis A)[b]	Recommended only for adolescents who live in areas with vaccination programs targeting older children, adolescents at increased risk for hepatitis A, and for adolescents for whom immunity is desired	Only women who have not been previously immunized and who are at increased risk for infection or desire protection[b]		Only women who have not been previously immunized and who are at increased risk for infection or desire protection[b]	Single antigen hepatitis A vaccine (HepA; Havrix) given as a two-dose series with 6–12 months between doses (Havrix) or a two-dose series with 6–18 months between doses (VAQTA). Alternatively, a three-dose series of combined hepatitis A and hepatitis B vaccine (HepA–HepB) may be used; the second dose is given 1 month following the first dose and the third dose is given 6 months following the first dose.
HepB (hepatitis B)	All adolescents not previously immunized	Only women who have not been previously immunized and who are at increased risk for infection or desire protection (see CDC guidelines)		Only women who have not been previously immunized and who are at increased risk for infection or desire protection (see CDC guidelines)	Complete vaccination requires a three-dose series: second dose given 4 weeks following the first dose; third dose given 8 weeks after the second dose (and at least 16 weeks after the first dose). Two-dose series (separated by 4 months) of adult formulation *Recombivax HB* may be used in girls 11–15 years.
Herpes zoster vaccine	NA	NA	NA	All women ≥ 60 years	One-time dose; no need for revaccination. **Contraindicated for persons with certain medical conditions** (see CDC guidelines).

HPV (human papillomavirus) vaccines:[c] Gardasil (4vHPV) Gardasil 9 (9vHPV)	All adolescents 11 or 12 years Women 13–26 years if not previously immunized	All women < 26 years if not previously immunized Pregnancy testing not required prior to administration	HPV vaccines are not recommended in pregnancy, though there is no evidence that the vaccine poses harm. If pregnancy is discovered after the first dose is given, completion of the series should be postponed until the woman is no longer pregnant; no additional intervention is necessary	All women < 26 years if not previously immunized	Complete vaccination requires a two-dose series: second dose given 6–12 months following the first dose. Adolescents initiating the vaccine at ≥ 15 years should receive three doses; administer the second dose 1–2 months after the first dose and the third dose 6 months after the first dose.
Influenza IIV (inactivated influenza)	All girls ≥ 9 years LAIV is not recommended for individuals < 18 years	All women	All adults, including pregnant women, can receive IIV	All women	Inactivated influenza vaccine comes in two formulations: inactivated influenza and recombinant influenza. Both are safe to use for all ages and during pregnancy. A single dose is administered each year, just prior to (or following) the start of the flu season. Contraindicated for persons with severe allergy to vaccine components, history of Guillain-Barré syndrome within 6 weeks of receipt of vaccine, or severe illness with fever (delay until illness is resolved). LAIV is a nasal spray and is contraindicated during pregnancy and during some flu seasons. See CDC guidelines.
Influenza: RIV (recombinant influenza vaccine)	All girls ≥ 9 years LAIV is not recommended for individuals < 18 years	All women	Contraindicated in pregnant women and in girls with asthma or underlying conditions that predispose them to influenza complications	All women	Recombinant influenza vaccine.
IPV (inactivated poliovirus)	Recommended only for girls < 18 years if not previously immunized	NA	NA	NA	Complete vaccination requires a three-dose series: second dose given 4 weeks following the first dose; third dose given 4 weeks after the second dose.

MMR (measles, mumps, and rubella)	Recommended only for girls not previously immunized	Regardless of birth year, women of childbearing age should have laboratory-confirmed rubella immunity Women who are not considered immune should receive the vaccine Vaccinate nonpregnant women during menses when nonpregnant status is certain	Contraindicated in pregnancy and for individuals who are immunocompromised Pregnant women without evidence of laboratory-confirmed immunity should be vaccinated immediately postpartum or following pregnancy termination	Women who are not considered immune	Complete vaccination requires administration of two doses separated by at least 4 weeks. Second dose is necessary only for women who are college students, women who work in a healthcare facility, and women who plan to travel internationally. Women who received inactivated measles vaccine or vaccine of unknown type from 1963 to 1967 should be revaccinated with two doses of MMR.
Meningococcal vaccines	Menactra or Menveo 11–12 years, with booster at age 16 years Catch-up vaccine given at 13–18 years if not previously immunized	MenACWY, MPSV4 Women who are first-year college students through age 21 years who live in residence halls and who were not previously vaccinated on or after their 16th birthday, HIV-positive, traveling to a country where the disease is prevalent or living in an area of outbreak, and military recruits	May be used if at risk for serogroup A, C, W, or Y	MenACWY, MPSV4 If not previously vaccinated or had disease	If first dose is received at $\geq$ 16 years, no booster is required. One-time single dose. Revaccination 5 years following first dose is necessary only for women who remain at increased risk for infection. There are several different meningococcal vaccines based on serotypes. Indications for each vaccine are different. See the CDC guidelines for additional information and scheduling recommendations for women with immunocompromising conditions and other risk factors for infection.
PCV (pneumococcal conjugate) or PPSV (pneumococcal polysaccharide)	NA	NA	Women $<$ 65 years only if they smoke, live in long-term care facilities, or have chronic lung disease, chronic cardiovascular disease, diabetes mellitus, chronic liver disease, alcoholism, cochlear implants, cerebrospinal fluid leaks, immunocompromising conditions, or functional anatomic asplenia (including sickle cell disease)	Women vaccinated before 65 years should be revaccinated at 65 years	Recommended vaccine types and interval spacing vary based on the indication for early vaccination. See the CDC guidelines for additional information and scheduling recommendations for women with immunocompromising and other serious medical conditions.

Tdap (tetanus diphtheria, and acellular pertussis)	All girls 11–12 years;18 years if not previously immunized	Single dose recommended for all pregnant women at 27–36 weeks' gestation during each pregnancy (regardless of number of years since last Td or Tdap vaccination)	Recommended for all adults. Substitute Tdap for Td once, then every 10 years	Recommended for all adults. Substitute Tdap for Td once, then every 10 years	Administer one dose with each pregnancy. A three-dose series is recommended for all adults with unknown or incomplete vaccination history: first two doses administered at least 4 weeks apart; third dose administered 6–12 months after the second dose.
VAR (varicella)	Only girls without evidence of immunity[d]	Contraindicated during pregnancy Pregnant women should be evaluated for laboratory-confirmed varicella immunity. Those without evidence of immunity should begin the two-dose vaccination series *immediately postpartum* or following pregnancy termination	Women without evidence of immunity only	Women without evidence of immunity only	Complete vaccination requires administration of two doses separated by at least 4 weeks for girls ≥ 13 years. For girls 7–12 years, the doses should be separated by 3 months. Evidence of immunity includes documentation of previous vaccination with two doses at least 4 weeks apart, history of herpes zoster diagnosed by a healthcare provider, history of varicella diagnosed by a healthcare provider, or laboratory-confirmation of immunity. Laboratory confirmation should be obtained for women who report having had a mild or atypical case of varicella.

Abbreviations: CDC, Centers for Disease Control and Prevention; HAV, hepatitis A virus; HIV, human immunodeficiency virus; LAIV, live attenuated influenza vaccine; NA, not applicable; Td, tetanus and diphtheria toxoids.

[a] See CDC guidelines for current immunization schedule.
[b] Women for whom hepatitis A vaccination is recommended include those who (1) use illicit drugs, (2) work with HAV-infected primates or HAV in laboratory settings, (3) have chronic liver disease, (4) receive clotting factor concentrates, (5) travel or work in countries with high or intermediate rates of hepatitis A, or (6) anticipate close contact with children from countries with high or intermediate rates of hepatitis A during the first 60 days after arrival in the United States (childcare providers or adoptive parents).
[c] HPV vaccination recommendations apply to male and female adolescents.
[d] Evidence of immunity = documented previous immunization or laboratory-confirmed evidence of immunity.

Based on Centers for Disease Control and Prevention. Recommended immunization schedules for adults aged 19 years or older—United States, 2017. 2017. Available at: https://www.cdc.gov/vaccines/schedules/hcp/adult.html. Accessed June 23, 2017.[70]

Health Maintenance: Secondary Prevention

In addition to primary preventive services to promote health and prevent the onset of disease, midwives provide secondary preventive services aimed at maintaining health through early detection and treatment of several disease states across the lifespan.

Routine Screening Across the Lifespan

After completing the health history and physical examination, additional screening information is obtained with the collection of laboratory tests. Recommendations for these tests are based on the existence of identified individual risk factors such as gender and age. Screening tests for asymptomatic women for which there is sufficient evidence to recommend are summarized in **Table 5-10**.[72] Screening test recommendations for pregnant women are presented in the *Prenatal Care* chapter.[72] These recommendations are reviewed regularly and are revised based on the findings of new evidence; readers are therefore encouraged to become familiar with and regularly access updated recommendations at the USPSTF website. Links to resources for preventive health services, including an electronic preventive services selector tool (ePSS), are found in the Resources section at the end of this chapter.

Table 5-10 **Recommended Routine Screening Tests**[a]

Disorder	Adolescents Age < 18 Years	Women Age ≥ 18 Years	Test Used	Screening Recommendation
Abnormal glucose and type 2 diabetes mellitus	Not indicated unless risk evident on history or physical examination	Women age 40–70 years who are overweight or obese	HbA1c *or* fasting plasma glucose *or* oral glucose tolerance test	Screening frequency is unknown, but every 3 years is suggested. Refer women with abnormal blood glucose for intensive counseling interventions to promote a healthy diet and increased physical activity.
Alcohol misuse	Insufficient evidence	Age ≥ 18 years	AUDIT, AUDIT-C, or single question screen	No evidence for screening frequency.
Breast cancer	NA	≥ 50 years, and screening at age 40 years made on an individual basis	Screening mammogram	Every 2 years until age 75 years. Insufficient evidence to recommend for or against screening for women age ≥ 75 years. Women with a family history of breast or ovarian cancer should be referred for genetic counseling and possible BRCA testing (see counseling recommendations described earlier in this chapter).
Cervical cancer	USPSTF recommends against screening women < 21 years unless at high risk	≥ 21 years	Cytology (Pap test)	Every 3 years until age 65 years. HPV testing combined with cytology (co-testing) every 5 years is acceptable for women age ≥ 30–64 years who prefer less frequent screening. Screening not recommended for women ≥ 65 years if adequately screened and not at high risk, or with history of hysterectomy without history of precancer or cervical cancer.

Colorectal cancer	NA	50 years Earlier if history of first-degree relative with early-onset colorectal disease	Fecal occult blood or DNA testing, sigmoidoscopy, CT colonography, or colonoscopy	Recommendations for appropriate screening intervals are controversial and should be made based on clinical findings. The decision to screen women age 76–85 years is made on an individual basis. Colonoscopy has greatest sensitivity and specificity for colorectal cancer screening. USPSTF found no studies that demonstrated greater effectiveness of any screening tool.
Chlamydia	All sexually active adolescents Recommended frequency depends on new or persistent risk factors since the last negative screen	All sexually active women ≤ 24 years Older women who are at increased risk for infection	Nucleic acid amplification tests	Nucleic acid amplification tests can be used with urine and vaginal swabs if a pelvic examination is not performed.
Depression	All adolescents age 12–18 years	18 years	PHQ-A or BDI-PC for adolescents Any reliable screening tool for adults	USPSTF also recommends ensuring that systems are in place to ensure accurate diagnosis, treatment, and follow-up. See the *Mental Health Conditions* chapter for a review of reliable screening tools, screening frequencies, and management of a positive screen.
Gonorrhea	All sexually active adolescents	All sexually active women ≤ 24 years and older women who are at increased risk for infection	Nucleic acid amplification tests	Recommended frequency depends on new or persistent risk factors since the last negative screen. Nucleic acid amplification tests can be used with urine and vaginal swabs if a pelvic examination is not performed.
Hepatitis B	Screen all adolescents and young adults at increased risk for infection, including those who have been vaccinated	All women at risk for infection	HBsAG	Anti-HBs and anti-HBc tests are done as part of a panel to distinguish between immunity and infection.
Hepatitis C	Screen all women at risk for infection	Offer screening to all women born between 1945 and 1965 Rescreen women at continued risk periodically	Anti-HCV antibody test; confirm with polymerase chain reaction testing	The most significant risk factor is past or current illicit IV drug use. Receiving a blood transfusion before 1992, long-term hemodialysis, incarceration, intranasal drug use, being born to a woman infected with hepatitis C, and getting a tattoo from an unregulated vendor are also risk factors. Women born between 1945 and 1965 have higher rates of hepatitis C than other women.

Human immunodeficiency virus (HIV)	All adolescents age ≥ 15 years	Unknown; suggested one-time screen for all women age 15–65 years, with repeat screens based on risk factors	Repeatedly reactive immunoassay and confirmation with Western blot	See the *Reproductive Tract and Sexually Transmitted Infections* chapter for review of risk factors and management of a positive screen.
Hypertension	Not indicated for routine screening	Yearly for women ≥ 40 years and for younger women with risk factors for hypertension Every 3–5 years for women age 18–39 years without a history of or risk factors for high blood pressure	Blood pressure measured with sphygmomanometer	USPSTF has found insufficient evidence to recommend for or against routine blood pressure measurements in children. Blood pressures should be taken with the woman seated at least 5 minutes after entering the examination room. Ensure use of a proper-size cuff and placement of the woman's arm at the level of the heart. Use the mean of two measurements taken at a single visit; confirm diagnosis with measurements take outside of the clinical setting.
Intimate-partner violence (IPV)	All adolescents 14–17 years	All women of childbearing age	Any reliable screening tool	Screening tools with high sensitivity and specificity for IPV identification include: Hurt, Insult, Threaten, Scream (HITS); Ongoing Abuse Screen/Ongoing Violence Assessment Tool (OAS/OVAT); Slapped, Threatened and Throw (STaT); Humiliation, Afraid, Rape, Kick (HARK); Modified Childhood Trauma Questionnaire—Short Form (CTQ-SF); and Woman Abuse Screen Tool (WAST). All women are at risk for IPV, and women age 14–46 years are at greatest risk. There is insufficient evidence to recommend for or against screening of younger girls and older women. Therefore, USPSTF makes no recommendation for or against screening girls < 14 years or women > age 46 years.
Latent tuberculosis infection	Yearly if at risk	Unknown; one-time screen may be sufficient with yearly screening for those at continued risk	Mantoux tuberculin skin tests or interferon-gamma release assays	Women at increased risk for latent tuberculosis infection include those who were born or lived in countries with high prevalence rates and those who live in high-risk settings (such as homeless shelters and correctional facilities). Consult local health departments to determine populations at risk in their community.

Lipid disorders	Insufficient evidence to recommend for or against screening for individuals < 40 years	40–75 years Frequency is every 5 years via expert opinion	Nonfasting or fasting total cholesterol, LDL-C, and HDL-C	These recommendations for cholesterol testing are part of a recommendation for the use of statins in adults with increased risk for CVD. Determining CVD risk requires assessment of cholesterol levels as recommended here.
Lung cancer	NA	55 years, then annually for women with a history of smoking	Low-dose computed tomography	Screen all women age 55–80 years who have a 30-pack-year smoking history and currently smoke or quit smoking in the past 15 years. Screening may be discontinued 15 years after the woman quits smoking.
Obesity	All	All	BMI	Those who are obese or overweight should be referred for intensive behavioral interventions to improve weight status. See the counseling interventions in this chapter.
Osteoporosis	NA	65 years	DXA scan of the hip and lumbar spine	Earlier screening is recommended for women with fracture risk ≥ that of white women age 65 years without risk factors. Rescreening should not occur sooner than 2 years following initial screening. Risk factors include Caucasian race, smoking, daily alcohol use, BMI < 21, and parental history of fracture.
Syphilis	Only those at increased risk for infection	Adults at increased risk for infection	VDRL or RPR—confirmed with FTA-ABS or TP-PA	Women at risk include those with HIV, commercial sex workers, certain racial and ethnic groups, and those who live in high-prevalence areas.
Tobacco use	All	All	Health history	Screen all adolescents. See the screening and counseling recommendations in this chapter.

Abbreviations: Anti-HBc, hepatitis B core total antibodies; anti-HBs, hepatitis B surface antibody; AUDIT, alcohol use disorders identification test; AUDIT-C, alcohol use disorders identification test—consumption; BDI-PC, Beck Depression Inventory for Primary Care; BMI, body mass index; CT, computed tomography; CVD, cardiovascular disease; DXA, dual-energy X-ray absorptiometry; FTA-ABS, fluorescent treponemal antibody-absorption test; HbA1c, hemoglobin A1C; HBsAG, hepatitis B surface antigen; HCV, hepatitis C virus; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; NA, not applicable; PHQ-A, Patient Health Questionnaire for Adolescents; RPR, rapid plasma reagin; TP-PA, treponemal pallidum particle agglutination assay; USPSTF, U.S. Preventive Services Task Force; VDRL, Venereal Disease Research Laboratory test.

[a] Unless otherwise indicated, these are the screening recommendations for healthy adolescents and women who are not at increased risk for infection or disease. Adolescents and adults with risk factors may require more frequent screening or screening with the use of additional tests and/or procedures.

Based on U.S. Preventive Services Task Force. Published recommendations. 2017. Available at: https://www.uspreventiveservicestaskforce.org/BrowseRec/Index/browse-recommendations. Accessed July 4, 2017.[72]

In addition to the recommendations made by the USPSTF, other groups such as the American College of Obstetricians and Gynecologists and the American Cancer Society have made

recommendations for screening that are often slightly different than the USPSTF recommendations. Healthcare facilities may adopt guidelines from different sources when establishing their institutional policies and procedures. Individual communities may also have "community standards" based on these recommendations. Therefore, it is essential that midwives remain aware of the recommendations of other groups and recommendations made based on community standards.

Reproductive Life Planning and Preconception Care

Reproductive life planning refers to a woman designing a set of personal reproductive goals and a plan for how to achieve those goals. Preconception care is a set of interventions that aim to identify and modify biomedical, behavioral, and social risks to the woman's health and her subsequent pregnancy outcomes through prevention and management. Preconception care is addressed separately from the rest of health promotion across the lifespan for two reasons: (1) Some health promotion interventions can directly influence perinatal outcomes, and (2) preconception care includes both primary and secondary health promotion activities.

Reproductive Life Planning

The CDC and other public health organizations have recommended reproductive life planning and preconception care,[73,74] though the evidence supporting preconception care as a package of services is sparse. Although strong evidence supports the value of specific healthcare services such as folic acid supplementation,[75] only a small number of women currently receive preconception care; in addition, health outcomes following preconception care as a package of services have not yet been well validated in research studies. The lack of positive evidence for preconception care may be at least partly related to women's lack of access to the multidisciplinary support and continuum of services that are the most effective interventions.

One of the critiques of preconception care is that this set of primary and secondary prevention strategies has value for women across the lifespan, not just during the time prior to childbearing. One of the dangers of viewing health care for women in such a way that prioritizes healthcare delivery based on plans for a future pregnancy rather than the general wellbeing of women is the presumption that every woman will attempt or want a pregnancy in the future. Moreover, given that approximately 45% of all pregnancies are not planned,[76] some experts in this field argue that a broader focus on optimal health behaviors for all women of reproductive age will be of greater benefit. The ACNM takes the position that "the health of a woman is a primary goal, independent of her reproductive life plan."[77]

Putting aside the controversy over these issues, it is clear that some adverse perinatal outcomes can be prevented via preconception identification of risks and use of risk-reduction strategies. Chronic diseases that are associated with adverse pregnancy outcomes are common among women of childbearing age.[76] In addition, reproductive life planning for women who do not want to become pregnant could help these women identify acceptable methods of contraception and improve spacing of births.

This section reviews the components of health promotion that relate specifically to pregnancy outcomes. The content included in a preconception care visit is presented in Appendix 5A. A woman-centered approach to initiating a discussion about reproductive goals is presented in **Table 5-11**.[78]

Table 5-11	Screening Strategies That Facilitate Reproductive Goals Counseling
Strategy	**Question(s) Included In Strategy**

One Key Question	Would you like to become pregnant in the next year?
Pregnancy **A**ttitudes, **T**iming, and **H**ow important is prevention (PATH)	1. Do you think you might like to have (more) children at some point? 2. If you are considering becoming a parent in the future, when do you think that might be? 3. How important is it to you to prevent pregnancy (until then)?

Based on Callegari LS, Aiken AR, Dehlendorf C, Cason P, Borrero S. Addressing potential pitfalls of reproductive life planning with patient-centered counseling. *Am J Obstet Gynecol.* 2017;216(2):129-134.[78]

The initial *one key question* will help direct the conversation. If a woman indicates that she is not planning a pregnancy in the near future, the next step is to determine whether she is at risk for becoming pregnant (i.e., engaging in heterosexual vaginal sex and possibly fertile), and if so, her desire to avoid pregnancy. A thorough review of contraceptive options and contraceptive counseling can be found in the *Family Planning* chapter. If a woman wants to become pregnant in the near future, the midwife continues with preconception care evaluation as presented in Appendix 5A.

Conclusion

Health promotion is an essential component of care provided by midwives, which is accomplished through the delivery of primary and secondary preventive services. Primary preventive services, which include counseling/education and vaccination, are provided to healthy individuals as a means to prevent the onset of disease. Secondary preventive services, which include evidence-based screening and early intervention, are provided to limit morbidity and mortality related to disease. As primary care providers who often see clients at regular intervals across the lifespan, midwives are well positioned to provide the preventive services described in this chapter and to offer individuals the tools necessary to become active participants in their own health maintenance and to achieve optimal levels of wellness.

Resources

Organization	Description	Webpage
Centers for Disease Control and Prevention (CDC)	Information about child safety seats for clinicians and women	https://www.cdc.gov/Motorvehiclesafety/Child_Passenger_Safety/index.html
	Vaccine Schedules App	https://www.cdc.gov/vaccines/schedules/hcp/schedule-app.html
	Vaccine Schedules webpage	https://www.cdc.gov/vaccines/schedules/hcp/adult.html
	Health insurance marketplace: list of clinical preventive services covered under the Affordable Care Act	https://www.cdc.gov/aca/marketplace/clinical-preventive-services.html
	Manual for clinicians planning to implement a brief intervention for risky alcohol use	https://www.cdc.gov/ncbddd/fasd/documents/alcoholsbiimplementationguide.pdf
	Physical activity guidelines for individuals across the lifespan: resources for clinicians and women	https://www.cdc.gov/physicalactivity/basics/index.htm
March of Dimes	Comprehensive interactive resources for women considering a pregnancy	http://www.marchofdimes.org/pregnancy/before-pregnancy.aspx
Georgia Breast cancer genomic consortium	B-RST screening tool to detect risks for hereditary breast and ovarian cancer	https://www.breastcancergenescreen.org
National Institutes of Health (NIH), National Human Genome Research Institute (NHGRI)	Genetic counseling services and resources for clinicians and women	https://www.genome.gov/11510370/

U.S. Department of Health and Human Services (DHHS)	*Smoke-Free Women*: smoking-cessation resources for women	https://women.smokefree.gov
National Highway Traffic Safety Administration (NHTSA)	Seat-belt recommendations during pregnancy	https://www.nhtsa.gov/sites/nhtsa.dot.gov/files/documents/pregnant-seat-belt-use.pdf
U.S. Food and Drug Administration (FDA)	Food safety resources for clinicians and women	https://www.fda.gov/food/foodborneillnesscontaminants/peopleatrisk/ucm081785.h
U.S. Preventive Services Task Force (USPSTF)	Preventive health services information for health professionals	https://www.uspreventiveservicestaskforce.org/Page/Name/tools-and-resources-f better-preventive-care
	Widget: electronic preventive services selector (ePSS)	http://epss.ahrq.gov/PDA/widget.jsp



References

1. International Confederation of Midwives. ICM international definition of the midwife. 2011. Available at: http://internationalmidwives.org/knowledge-area/icm-publications/icm-core-documents.html. Accessed June 3, 2017.
2. American College of Nurse-Midwives. Definition of midwifery and scope of practice of certified nurse-midwives and certified midwives. 2012. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000266/Definition%20of%20Mi Accessed June 3, 2017.
3. American College of Nurse-Midwives. Core competencies for basic midwifery practice. 2012. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000050/Core%20Comptencies% Accessed June 3, 2017.
4. International Confederation of Midwives. Essential competencies for basic midwifery practice. 2013. Available at: http://internationalmidwives.org/what-we-do/education-coredocuments/essential-competencies-basic-midwifery-practice/. Accessed June 3, 2017.
5. World Health Organization. Frequently asked questions. Available at: http://www.who.int/suggestions/faq/en/. Accessed June 3, 2017.
6. Centers for Disease Control and Prevention. Physical activity: glossary of terms. 2015. Available at: https://www.cdc.gov/physicalactivity/basics/glossary/index.htm. Accessed June 3, 2017.
7. Agency for Healthcare Research and Quality. Putting prevention into practice. 2014. Available at: https://www.ahrq.gov/professionals/quality-patient-safety/quality-resources/tools/ppip/ppipshort.html. Accessed June 3, 2017.
8. Organisation for Economic Co-operation and Development. Health spending. 2017. Available at: https://data.oecd.org/healthres/health-spending.htm. Accessed June 4, 2017.
9. Centers for Medicare and Medicaid Services. National health expenditure data: NHE fact sheet. 2017. Available at: https://www.cms.gov/Research-Statistics-Data-and-Systems/Statistics-Trends-and-Reports/NationalHealthExpendData/NHE-Fact-Sheet.html. Accessed June 4, 2017.
10. Agency for Healthcare Research and Quality. Trends in healthcare costs and the concentration of medical expenditures. 2012. Available at: https://www.ahrq.gov/news/events/nac/2012-07-nac/cohenmeyers/cohenmeyers.html. Accessed June 4, 2017.
11. Centers for Disease Control and Prevention. Chronic disease overview. 2016. Available at: https://www.cdc.gov/chronicdisease/overview/index.htm. Accessed June 4, 2017.
12. Centers for Disease Control and Prevention. Leading causes of death in females, 2014. 2017. Available at: https://www.cdc.gov/women/lcod/index.htm. Accessed June 4, 2017.
13. Curry SJ, Grossman DC, Whitlock EP, Cantu A. Behavioral counseling research and evidence-based practice recommendations: U.S. Preventive Services Task Force perspectives. *Ann Intern Med*. 2014;160(6):407-414.
14. Institute of Medicine. *Health Literacy: A Prescription to End Confusion*. Washington, DC: National Academies Press; 2004. Available at: https://www.nap.edu/catalog/10883/health-literacy-a-prescription-to-end-confusion. Accessed June 5, 2017.
15. Knowles MS, Holton EF, Swanson RA. *The Adult Learner: The Definitive Classic in Adult Education and Human Resource Development*. 8th ed. New York, NY: Routledge; 2015.
16. Institute of Medicine. *Health Professions Education: A Bridge to Quality*. Washington, DC: National Academies Press; 2003.
17. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS. Evidence based medicine: what it is and what it isn't. *Br Med J*. 1996;312(7023):71-72.
18. American College of Nurse-Midwives. Position statement: creating a culture of safety in midwifery care. 2016. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000059/Creating-a-culture-of-safety-in-midwifery-care-MAR2016.pdf. Accessed June 5, 2017.
19. U.S. Preventive Services Task Force. USPSTF: who we are and how we work. 2017. Available at: https://www.uspreventiveservicestaskforce.org/Page/Name/task-force-resources. Accessed June 5, 2017.
20. U.S. Preventive Services Task Force. Archived final recommendation statement: breast cancer screening. 2013. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/breast-cancer-screening. Accessed June 5, 2017.
21. U.S. Preventive Services Task Force. Grade definitions. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Name/grade-definitions. Accessed June 5, 2017.
22. Centers for Disease Control and Prevention. The four domains of chronic disease prevention. 2015. Available at: https://www.cdc.gov/chronicdisease/resources/publications/four-domains.htm. Accessed June 5, 2017.
23. U.S. Preventive Services Task Force. Screening and behavioral counseling interventions in primary care to reduce alcohol misuse: U.S. Preventive Services Task Force recommendation statement. 2013. Available at:

https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/alcohol-misuse-screening-and-behavioral-counseling-interventions-in-primary-care. Accessed June 5, 2017.
24. Centers for Disease Control and Prevention. Alcohol use and binge drinking among women of childbearing age—United States, 2011–2013. *MMWR*. 2015;64(37):1042-1046. Available at: https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6437a3.htm. Accessed June 5, 2017.
25. National Institute on Alcohol Abuse and Alcoholism. Drinking levels defined. Available at: https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking. Accessed June 5, 2017.
26. National Institute on Alcohol Abuse and Alcoholism. Helping patients who drink too much: a clinician's guide, updated 2005 edition. 2016. Available at: https://pubs.niaaa.nih.gov/publications/Practitioner/CliniciansGuide2005/clinicians_guide.htm. Accessed June 5, 2017.
27. Centers for Disease Control and Prevention. Planning and implementing screening and brief intervention for risky alcohol use: a step-by-step guide for primary care practices. 2014. Available at: https://www.cdc.gov/ncbddd/fasd/documents/alcoholsbiimplementationguide.pdf. Accessed June 8, 2017.
28. Substance Abuse and Mental Health Services Administration, HRSA Center for Integrated Health Solutions. The Alcohol Use Disorders Identification Test (AUDIT). Available at: https://www.integration.samhsa.gov/clinical-practice/screening-tools#drugs. Accessed June 8, 2017.
29. Substance Abuse and Mental Health Services Administration, HRSA Center for Integrated Health Solutions. AUDIT-C overview. 2014. Available at: https://www.integration.samhsa.gov/clinical-practice/screening-tools#drugs. Accessed June 8, 2017.
30. Whitlock EP, Orleans CT, Pender N, Allan J. Evaluating primary care behavioral counseling interventions: an evidence-based approach. *Am J Prev Med*. 2002;22(4):267-284.
31. Centers for Disease Control and Prevention. Breastfeeding report cards. 2016. Available at: https://www.cdc.gov/breastfeeding/data/reportcard.htm. Accessed June 10, 2017.
32. U.S. Department of Health and Human Services. *Healthy people 2020: maternal, infant, and child health*. 2017. Available at: https://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives. Accessed June 10, 2017.
33. U.S. Preventive Services Task Force. Breastfeeding: primary care interventions. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/breastfeeding-primary-care-interventions. Accessed June 10, 2017.
34. Woolf SH, Jonas S, Lawrence RS. *Health Promotion and Disease Prevention in Clinical Practice*. Baltimore, MD: Williams & Wilkins; 1996.
35. U.S. Preventive Services Task Force. Dental and periodontal disease: counseling. 2017. Available at: https://www.uspreventiveservicestaskforce.org/BrowseRec/InactiveTopic/200. Accessed June 10, 2017.
36. Centers for Disease Control and Prevention. What can adults do to maintain good oral health. 2016. Available at: https://www.cdc.gov/oralhealth/basics/adult-oral-health/tips.html. Accessed June 10, 2017.
37. Centers for Disease Control and Prevention. Defining adult overweight and obesity. 2016. Available at: https://www.cdc.gov/obesity/adult/defining.html. Accessed June 10, 2017.
38. U.S. Preventive Services Task Force. Healthful diet and physical activity for cardiovascular disease prevention in adults with cardiovascular risk factors: behavioral counseling. 2014. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/healthy-diet-and-physical-activity-counseling-adults-with-high-risk-of-cvd. Accessed June 10, 2017.
39. U.S. Preventive Services Task Force. Obesity in adults: screening and management. 2012. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/obesity-in-adults-screening-and-management. Accessed June 10, 2017.
40. U.S. Preventive Services Task Force. Healthful diet and physical activity for cardiovascular disease prevention in adults without known risk factors: behavioral counseling. 2017. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/healthful-diet-and-physical-activity-for-cardiovascular-disease-prevention-in-adults-without-known-risk-factors-behavioral-counseling. Accessed July 13, 2017.
41. Centers for Disease Control and Prevention. Breast cancer. 2016. Available at: https://www.cdc.gov/cancer/breast/index.htm. Accessed June 12, 2017.
42. U.S. Preventive Services Task Force. *BRCA*-related cancer: risk assessment, genetic counseling, and genetic testing. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/brca-related-cancer-risk-assessment-genetic-counseling-and-genetic-testing. Accessed June 12, 2017.
43. Centers for Disease Control and Prevention. 2016. Motor vehicle crash deaths. Available at: https://www.cdc.gov/vitalsigns/motor-vehicle-safety/index.html. Accessed June 10, 2017.
44. Sauber-Schatz EK, Ederer DJ, Dellinger AM, Baldwin GT. Vital signs: motor vehicle injury prevention—United States and 19 comparison countries. *MMWR*. 2016. Available at: https://www.cdc.gov/mmwr/volumes/65/wr/mm6526e1.htm?

s_cid=mm6526e1_w#suggestedcitation. Accessed June 10, 2017.
45. U.S. Preventive Services Task Force. The guide to clinical preventive services, 2014. 2014. Available at: https://www.ahrq.gov/professionals/clinicians-providers/guidelines-recommendations/guide/index.html. Accessed June 10, 2017.
46. National Highway Traffic Safety Administration. Proper seatbelt use. Available at: www.nhtsa.gov/staticfiles/nti/teen-drivers/pdf/seatbeltuse.pdf. Accessed June 10, 2017.
47. National Highway Traffic Safety Administration. If you're pregnant: seat belt recommendations for drivers and passengers. 2015. Available at: www.safercar.gov/parents/SeatBelts/seatbelts-images/Pregnant-Seat-Belt-Use.pdf. Accessed June 10, 2017.
48. Centers for Disease Control and Prevention. Distracted driving. 2017. Available at: https://www.cdc.gov/motorvehiclesafety/distracted_driving/index.html. Accessed June 10, 2017.
49. Bergen G, Stevens MR, Burns ER. Falls and fall injuries among adults aged ≥ 65 years—United States, 2014. *MMWR*. 2016. Available at: https://www.cdc.gov/mmwr/volumes/65/wr/mm6537a2.htm?s_cid=mm6537a2_w. Accessed June 10, 2017.
50. U.S. Preventive Services Task Force. Falls prevention in older adults: counseling and preventive medication. 2012. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/falls-prevention-in-older-adults-counseling-and-preventive-medication. Accessed June 10, 2017.
51. Centers for Disease Control and Prevention. STEADI: older adult fall prevention. 2017. Available at: https://www.cdc.gov/steadi/. Accessed June 10, 2017.
52. Centers for Disease Control and Prevention. Physical activity basics. 2015. Available at: https://www.cdc.gov/physicalactivity/basics/index.htm. Accessed July 11, 2017.
53. Centers for Disease Control and Prevention. 2015. Measuring physical activity intensity. Available at: https://www.cdc.gov/physicalactivity/basics/measuring/index.html. Accessed July 13, 2017.
54. U.S. Preventive Services Task Force. Sexually transmitted infections: behavioral counseling. 2014. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/sexually-transmitted-infections-behavioral-counseling1. Accessed June 10, 2017.
55. Centers for Disease Control and Prevention. Reproductive health: unintended pregnancy prevention. 2016. Available at: https://www.cdc.gov/reproductivehealth/contraception/unintendedpregnancy/index.htm. Accessed on June 10, 2017.
56. U.S. Department of Health and Human Services. *Healthy people 2020: family planning—objectives*. 2012. Available at: https://www.healthypeople.gov/2020/topics-objectives/topic/family-planning/objectives. Accessed June 10, 2017.
57. Centers for Disease Control and Prevention. Skin cancer: what is skin cancer? 2017. Available at: https://www.cdc.gov/cancer/skin/basic_info/what-is-skin-cancer.htm. Accessed June 11, 2017.
58. Centers for Disease Control and Prevention. Skin cancer statistics. 2016. Available at: https://www.cdc.gov/cancer/skin/statistics/index.htm. Accessed June 11, 2017.
59. U.S. Preventive Services Task Force. Skin cancer: counseling. 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/skin-cancer-counseling. Accessed June 11, 2017.
60. Centers for Disease Control and Prevention. Skin cancer: what can I do to reduce my risk of skin cancer? 2017. Available at: https://www.cdc.gov/cancer/skin/basic_info/prevention.htm. Accessed June 11, 2017.
61. Centers for Disease Control and Prevention. Smoking and tobacco use: data and statistics. 2017. Available at: https://www.cdc.gov/tobacco/data_statistics/index.htm. Accessed June 11, 2017.
62. Baraona LK, Lovelace D, Daniels JL, McDaniel L. Tobacco harms, nicotine pharmacology, and pharmacologic tobacco cessation interventions for women. *J Midwifery Womens Health*. 2017;62(3):253-268.
63. Blood-Siegfried J, Rende EK. The long-term effects of perinatal nicotine exposure on neurologic development. *J Midwifery Womens Health*. 2010;55(2):143-152.
64. U.S. Preventive Services Task Force. Tobacco smoking cessation in adults, including pregnant women: behavioral and pharmacotherapy interventions. 2015. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/tobacco-use-in-adults-and-pregnant-women-counseling-and-interventions1. Accessed June 11, 2017.
65. Centers for Disease Control and Prevention. 2017. Smoking and tobacco use: secondhand smoke. 2017. Available at: https://www.cdc.gov/tobacco/basic_information/secondhand_smoke/index.htm. Accessed June 11, 2017.
66. Agency for Healthcare Research and Quality. Five major steps to intervention (the "5 A's). 2017. Available at: https://www.ahrq.gov/professionals/clinicians-providers/guidelines-recommendations/tobacco/5steps.html. Accessed June 11, 2017.
67. Price JH, Mohamed I, Jeffrey JD. Tobacco intervention training in American College of Nurse-Midwives accredited education programs. *J Midwifery Womens Health*. 2008;53(1):68-74.
68. Abatemarco DJ, Steinberg MB, Delnevo CD. Midwives' knowledge, perceptions, beliefs, and practice supports regarding tobacco dependence treatment. *J Midwifery Womens Health*. 2007;52(5):451-457.

69. Chertok IR, Archer SH. Evaluation of a midwife- and nurse-delivered 5A's prenatal smoking cessation program. *J Midwifery Womens Health*. 2015;60(2):175-181.
70. Centers for Disease Control and Prevention. Recommended immunization schedule for adults aged 19 years or older—United States, 2017. 2017. Available at: https://www.cdc.gov/vaccines/schedules/hcp/adult.html. Accessed June 23, 2017.
71. Hayes C. Creating a conversation about immunizations. *J Midwifery Womens Health*. 2014;59(5):481-482.
72. U.S. Preventive Services Task Force. Published recommendations. 2017. Available at: https://www.uspreventiveservicestaskforce.org/BrowseRec/Index. Accessed July 4, 2017.
73. Johnson K, Posner SF, Biermann J, et al. Preconception Care Work Group; Select Panel on Preconception Care. Recommendations to improve preconception health and health care—United States. A report of the CDC/ATSDR Preconception Care Work Group and the Select Panel on Preconception Care. *MMWR*. 2006;55(RR06):1-23.
74. Files JA, Frey KA, Davis PS, Hunt KS, Noble BN, Mayer AP. Developing a reproductive life plan. *J Midwifery Womens Health*. 2011;56:468-474.
75. Wolff T, Witkop CT, Miller T. Syed SB. *Folic Acid Supplementation for the Prevention of Neural Tube Defects: An Update of the Evidence for the U.S. Preventive Services Task Force*. Evidence Synthesis No. 70. AHRQ Publication No. 09-051132-EF-1. Rockville, MD: Agency for Healthcare Research and Quality; May 2009.
76. Finer LB, Zolna MR. Declines in unintended pregnancy in the United States, 2008–2011. *N Engl J Med*. 2016;374(9):843-852.
77. American College of Nurse-Midwives. Position statement: the role of the certified nurse-midwife/certified midwife in preconception health and health care. 2013. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000081/Preconception%20Healt Accessed July 17, 2017.
78. Callegari LS, Aiken AR, Dehlendorf C, Cason P, Borrero S. Addressing potential pitfalls of reproductive life planning with patient-centered counseling. *Am J Obstet Gynecol*. 2017;216(2):129-134.

APPENDIX

5A

Preconception Care Visit

MARY C. BRUCKER AND MARY ANN FAUCHER

The editors acknowledge Cecilia M. Jevitt for contributions to this appendix.

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Introduction

Preconception care is founded on the basic principle that healthy women tend to have healthy offspring. The period of time before a woman is pregnant can be critical to promote and maintain her health during pregnancy. Targeted care during the preconception period can lead to identification and modification of risks that can influence not only a woman's health, but the health of her future child and society as a whole.[1] **Table 5A-1** defines terms commonly encountered in this area.[2-6]

Table 5A-1 Definitions of Terms

Term	Definition
Preconception care	Multiple definitions exist, but in all cases the goal is to provide care to reproductive-age women that promotes a woman's health and identifies and modifies her risks to promote a healthy pregnancy if or when it ensues.
Preconception period	The time before or between pregnancies. Some publications focusing on care of the pregnant woman have also called this span the *prepregnancy period* or *interconception period.*
One Key Question	A screening strategy using one question to promote efficient identification of an individual's intentions regarding pregnancy.
Reproductive life plan (RLP)	An individual's plan that reflects future intentions for reproduction. It should not be assumed that an RLP means that a pregnancy is planned; the RLP should be accepted at face value without judgment.

Based on American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Prepregnancy care. In: *Guidelines for Perinatal Care.* 8th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2017:131-148[2]; Burgess CK, Henning PA, Norman WV, Manze MG, Jones HE. A systematic review of the effect of reproductive intention screening in primary care settings on reproductive health outcomes. *Fam Pract.* September 8, 2017. [Epub ahead of print]. doi:10.1093/fampra/cmx086[3]; Files JA, Frey KA, David PS, Hunt KS, Noble BN,

Mayer AP. Developing a reproductive life plan. *J Midwifery Womens Health.* 2011;56(5):468-474[4]; Lang AY, Boyle JA, Fitzgerald G, et al. Optimising preconception health in women of reproductive age. *Minerva Ginecol.* 2018;70(1):99-119[5]; Nypaver C, Arbour M, Niederegger E. Preconception care: improving the health of women and families. *J Midwifery Womens Health*. 2016;61(3):356-364.[6]

The clarion call to action for preconception care was first sounded more than 30 years ago.[7] Since then, various controversies have arisen, predominantly focused on the strategies surrounding preconception care. For example, some groups have advocated an *Every Woman, Every Visit* approach, in which nonpregnant women are screened at every clinical encounter. Unfortunately, this strategy can be interpreted as a requirement to provide intensive health counseling and rigorous education about pregnancy for all women, raising concerns that such an approach could be viewed as pronatalism because it ignores the fact that some women may never desire to be pregnant.[8] Other healthcare professionals have expressed concerns that time spent with women not intending to become pregnant within the near future may be too narrowly focused and has a vocabulary that may disengage women.[9] None of these concerns questions the utility of preconception care, and a new approach to the concept addresses these issues.

Preconception Care

Today's approach to preconception care is to ask any individual who is of reproductive age *One Key Question*. This question is most commonly phrased as follows: *Would you like to become pregnant in the next year*? The key question is a critical part of a *reproductive life plan (RLP)*.[4] The answer should be documented in the woman's health record and the question regularly revisited because the woman may change the answer as their circumstances change. The key question is neutrally phrased and enables the professional to target health education, and thus, time during the healthcare visit, to the needs of the individual. **Figure 5A-1** provides an algorithm for use in providing care to reproductive-age women.

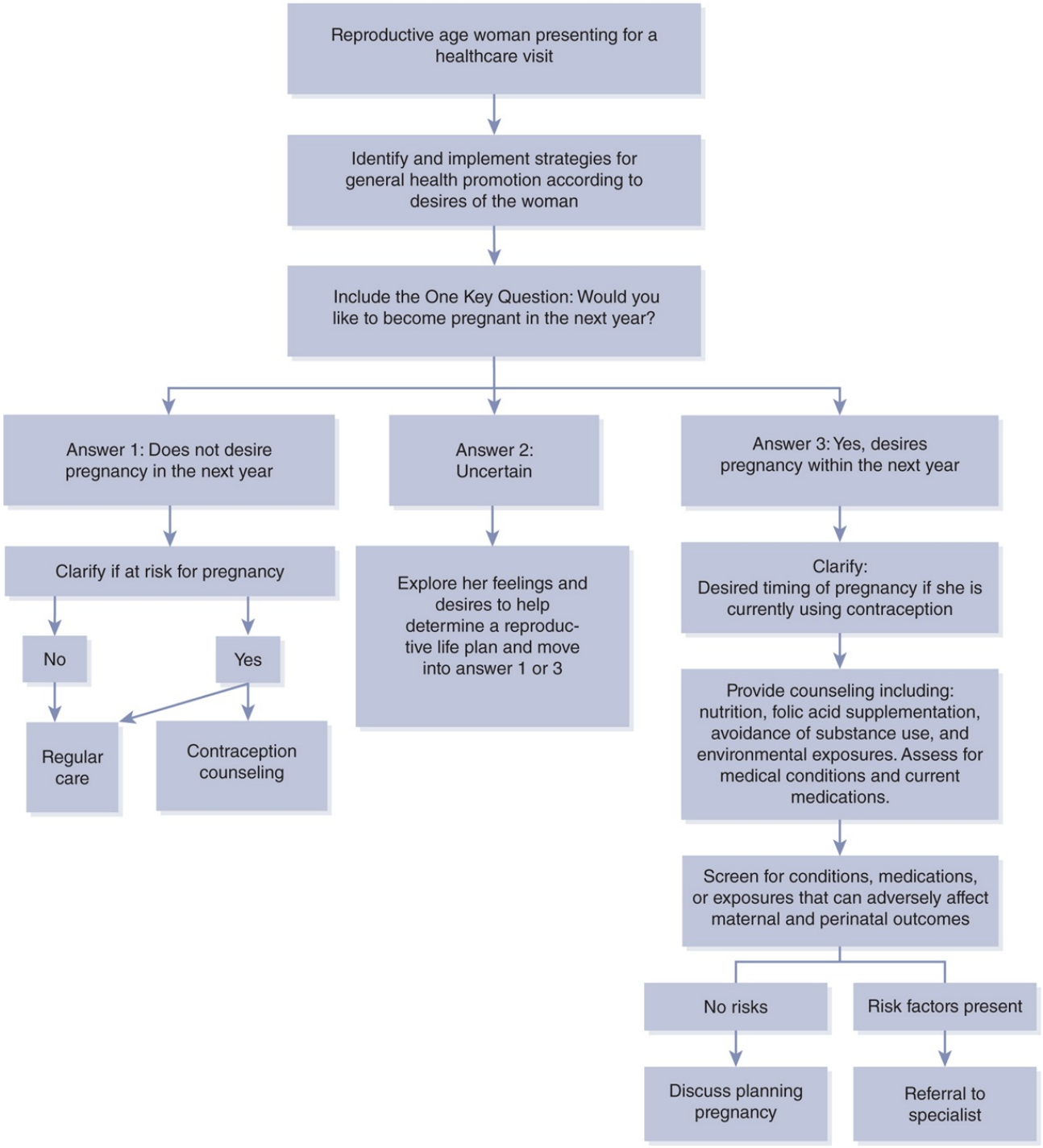


Figure 5A-1 Algorithm illustrating the use of One Key Question.

A midwife is most likely to meet women who provide one of three answers to the key question. For all of these women, health promotion—as discussed in the *Health Promotion Across the Lifespan* chapter—is an essential component of care.

For women who answer "1" (does not plan to get pregnant in the next year) who are at risk for pregnancy, time with the midwife can be best focused on identifying a reliable contraceptive method that she desires and can use consistently and correctly, as described in

the *Family Planning* chapter.

Individuals who answer the key question with "2" (uncertain about whether or not they are planning a pregnancy in the next year) can be helped to reflect and develop a reproductive life plan. Although ultimately a woman will determine that she does or does not plan a pregnancy in the coming year, many women will choose contraception while they ponder how to design their reproductive life plan.

Women who respond that they wish to become pregnant in the next year (answer 3 to the key question) are the women for whom this appendix is written. These women are planning a pregnancy within the next 6 months or 1 year. More information about use of the key question and details about preconception care can be obtained from the National Preconception Health and Health Care Initiative (PCHHC), a public/private partnership of more than 70 organizations. Its website, Before, Between & Beyond, is an extensive and credible source of information about individuals in the preconception period and is included in the Resources table at the end of this appendix.[10]

General Health Promotion and Preconception Care

All women can profit from health promotion, as discussed in the *Health Promotion Across the Lifespan* chapter. However, women who are planning pregnancy within the near future merit a review of some topics that can impact the course of pregnancy.

Substance Use

Counseling that is particularly important during a preconception visit includes recommendations regarding substance use. Women who use alcohol during pregnancy run the risk that their child will develop a fetal alcohol spectrum disorder, including fetal alcohol syndrome, the most severe form. Smoking or other tobacco use remains the leading preventable cause of premature death for women in the United States, despite the declining rate of use over the last few years. Tobacco is fetotoxic and is associated with an increased incidence of low birth weight in newborns who were exposed in utero. Particular attention also should be paid to the increasing number of women dealing with opioid use: Opioid use is harmful to the woman and also associated with neonatal abstinence syndrome. The preconception period represents an opportunity for women to seek help in changing their patterns of use and misuse of these substances, particularly since it usually takes time and sometimes several attempts to quit successfully.[2]

Several myths exist regarding oral health in pregnancy. Some women may have heard that loss of teeth is inherently associated with pregnancy and, therefore, may not seek dental care while pregnant. Other women may avoid such care due to fear of risk of X rays. In any case, research has demonstrated an increased risk of preterm birth when women have significant periodontal disease during pregnancy.[11] This increase in the incidence of preterm birth in conjunction with periodontal disease is likely linked to the inflammatory process. Ideally, dental work necessary to correct periodontal disease or dental caries should be completed

prior to pregnancy.

Immunizations

Evidence exists that several vaccines are effective interventions to promote healthy pregnancies when administered prior to pregnancy in women without immunity.[12] All women of reproductive age should have their immunization status for tetanus–diphtheria–acellular pertussis toxoid (Tdap); measles, mumps, and rubella (MMR); hepatitis B; and varicella reviewed annually. If immunity is lacking, these immunizations should be offered. Current recommendations are to give one dose of Tdap to every pregnant woman during every pregnancy (27–36 weeks) regardless of the duration of time since the last Tdap vaccination. This is true for all pregnant women, even those vaccinated preconceptionally. Women at risk for acquiring hepatitis B or who have not received the vaccine should be offered the immunization series prior to pregnancy.

Rubella is a known teratogen, causing congenital rubella syndrome when nonimmune women are infected during the first 16 weeks of pregnancy. Women who receive the rubella immunization should be counseled to avoid pregnancy for 1 month, based on a theoretical risk of passing rubella to a developing embryo. However, there are no reports of teratogenicity secondary to receiving rubella vaccine in early pregnancy; thus, pregnancy termination is not recommended for women who are inadvertently vaccinated for rubella within 4 weeks of becoming pregnant or when they are pregnant.[13]

Nutrition: Dietary Counseling and Folic Acid Supplementation

All women should be counseled to avoid vitamins or multivitamin preparations that exceed the current recommended daily allowances because of the potential adverse effects associated with higher doses. For example, vitamin A is associated with fetal malformations if very high doses of supplements are consumed in the first trimester. In addition, some nutritional supplements can adversely interact with absorption of foods or medications, so it is important to review and educate women about all medication use when taking multivitamins.

Most women in the United States do not consume the recommended amounts of folate from diet alone. All professional organizations recommend that women planning a pregnancy or capable of becoming pregnant take a supplement that has 400 to 800 micrograms of folic acid per day to reduce the risk of having a fetus with a neural tube defect.[15] Most over-the-counter multivitamins contain 400 micrograms (0.4 milligrams) of folic acid, and generic preparations are appropriate.

Vitamin D deficiency is widespread, yet data is still emerging with regard to the value of vitamin D supplementation. At present, regular screening of pregnant women for vitamin D deficiency and supplementation of vitamin D during pregnancy (beyond the amount included in a multivitamin) are not recommended. However, clinicians may consider screening women at increased risk of vitamin D deficiency, such as those who live in cold climates and along northern latitudes, those who wear sunscreen regularly, and those who belong to a racial/ethnic group with dark skin.[16] When a deficiency is identified, ingestion of 1000-4000 IU/day is

considered to be reasonable and safe by most experts.[16]

Decreasing Environmental Risks

The most common environmental toxins in the United States and their known health effects are listed in **Table 5A-2**. The adverse effects of environmental toxins is the subject of a great deal of research and new knowledge is rapidly emerging.[17-24] No woman can totally avoid exposure to toxins. Naturally occurring substances, such as lead and cadmium, are not toxic at low levels; however, human activity has resulted in concentrated levels and increased exposure to these substances. Even though lead has been banned from fuel mixes in the United States for several decades, lead from automobile exhaust continues to contaminate soil and water, especially in the land near busy highways. Lead-based paint continues to present exposure risks in older homes and playgrounds that were constructed before these paints were banned.

Table 5A-2 Environmental Toxins with General and Reproductive Effects

	General Effects					Reproductive Effects									
Agent/Source of Exposure	**Cancer**	**Increased BMI**	**Neurologic Impairment**	**Thyroid Dysfunction**	**Genomic Methylation Alternations**	**Spontaneous Abortion**	**Preterm Birth**	**Stillbirth**	**Decreased Birth Weight**	**Impaired Fetal Growth**	**Impaired Neurologic Development[a]**	**Congenital Malformations**	**Reduced Sperm Quality**	**Post-Term Birth**	**Breastfeeding Effects When Known**
Lead: Battery manufacture, smelting, soldering, firearm manufacture, lead-based paints, lead pipes, ceramics, jewelry, folk medicines, hair dyes, contaminated soil			X		X						X				Most common heavy metal found in breast milk
Mercury: Coal-fired plants, contaminated fish			X								X				Found in breast milk
Arsenic: Wood preservative, mining, manufacturing	X														Found in breast milk
Cadmium: Mining, industrial pollution	X														Least common heavy metal found in breast milk
Pesticides, organophosphates, organochlorines, organohalogens: Agriculture, landscaping, pest control	X		X							X	X		X	X	Levels decreasing in breast milk samples during 2000s
Solvents (benzene, toluene, xylene, styrene, bromopropanes, perchloroethylene): Plastics, synthetic fibers, dyes, detergents, lubricants, printing, insulation, leather tanning, cleaning products, fiberglass, carpet backing						X			X			X			
Phthalates: Medical devices, building materials, cosmetics, pharmaceuticals, food processing, toys							X				X				
Polychlorinated biphenyls: Industrial insulators and lubricants	X	X							X		X		X		May be present in breast milk

Perchlorate: Rocket fuel, fireworks, bleach, fertilizers	X										May be present in breast milk
Perfluorochemicals, Perfluorooctane sulfonate (PFOS): Nonstick cookware, water-resistant packaging, food containers, manufacturing		X				X			X		Present in breast milk samples of women from many countries; effect unknown
Polybrominated diphenyl ethers: Flame retardant, furniture, textiles, carpeting, electronics				X	X	X		X			Levels increasing in breast milk samples during 2000s
Bisphenol A: Plastics, resins, packaging			X					X			
Formaldehyde: Wood adhesives, abrasive agents, clinical laboratory preservative, embalming			X			X					
Antineoplastic drugs: Chemotherapy			X			X					
Anesthetic gases: Human and veterinary surgery			X						X		
Ethylene oxide: Sterilizing gas for heat-sensitive medical items and surgical instruments			X	X							
Ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, lead, particulate matter: Air pollution						X		X	X		
Cigarette smoke			X	X		X				X	

[a] Impaired neurologic development includes abnormal brain development, mental disability, lowered IQ, aggressive behavior, attention-deficit disorder, hyperactivity, and executive function deficits.

Based on American College of Obstetricians and Gynecologists Committee on Health Care for Underserved Women, American Society for Reproductive Medicine Practice, Committee, The University of California, San Francisco, Program on Reproductive Health and the Environment. *Exposure to Toxic Environmental Agents*. Washington, DC: American College of Obstetricians and Gynecologists; 2013[17]; Di Renzo GC, Conry JA, Blake J, et al. International Federation of Gynecology and Obstetrics opinion on reproductive health impacts of exposure to toxic environmental chemicals. *Int J Gynaecol Obstet.* 2015;131(3):219-225[18]; Furst P. Dioxins, polychlorinated biphenyls and other organohalogen compounds in human milk: levels, correlations, trends and exposure through breastfeeding. *Mol Nutr Food Res.* 2006;50(10):922-933[19]; Gao Y, Chen H, Xiao X, et al. Perfluorooctanesulfonate (PFOS)-induced Sertoli cell injury through a disruption of F-actin and microtubule organization is mediated by Akt1/2. *Sci Rep.* 2017;7(1):1110[20]; Project TENDR. Project TENDR: Targeting Environmental Neuro-Developmental Risk. The TENDR consensus statement. *Environ Health Perspect.* 2016;124(7):A118-A122[21]; Rebelo FM, Caldas ED. Arsenic, lead, mercury and cadmium: toxicity, levels in breast milk and the risks for breastfed infants. *Environ Res.* 2016;151:671-688[22]; Solomon GM, Weiss PM. Chemical contaminants in breast milk: time trends and regional variability. *Environ Health Perspect.* 2002;110(6):A339-A347[23]; Woodruff TJ, Zota AR, Schwartz JM. Environmental chemicals in pregnant women in the United States: NHANES 2003–20004. *Environ Health Perspect.* 2011;119(6):878-885.[24]

The most prevalent environmental toxins are manufactured chemicals, including pesticides, polychlorinated biphenyls, and perfluorochemicals. Most of these chemicals degrade slowly and are absorbed by humans through ingestion of contaminated food and water or inhalation of particles. Environmental toxins have broad deleterious effects on reproductive health, including decreased sperm quality, congenital anomalies, increased spontaneous miscarriage, stillbirth, low birth weight, and preterm labor. Many environmental toxins also cross the placenta and have been found in cord blood and fetal tissue as well as breast milk.

An important first step in decreasing exposure to environmental toxins is risk identification through the collection of a thorough history of occupational, recreational, residential, or dietary exposures. Counseling should include sources of environmental toxins and approaches used to limit exposure. To conduct this kind of counseling, midwives need a working knowledge of local and regional environmental pollutants, including soil contaminated by past and present industry and agricultural pesticides. Midwives can also provide guidance to reduce exposure to toxins for all women, including recommendations to rinse fruits and vegetables to remove pesticide residue and to avoid consuming contaminated foods.

Special Considerations

A complete history may reveal reproductive problems including extremes of age, genetic conditions of the woman or family, and chronic illnesses and treatments. In these situations, preconception care can be invaluable.

Risk for Genetic Disorders

Knowledge about genetic inheritance, screening, and counseling has exploded during this century. Collection of a three-generation history is needed to identify genetic conditions. If a specific risk factor is identified or if the future parents have concerns, referral to a genetic specialist is required.[2] Population genetics suggest that some screening can be focused by ethnic or racial backgrounds. For example, Ashkenazi Jews have a higher risk of Tay–Sachs disease than other groups. Sickle cell disease is more common among African Americans, while cystic fibrosis has a higher incidence among Northern Europeans.

When a woman has been identified as a carrier for a condition, her reproductive partner should also be offered screening. In the event that a referral for genetic counseling is necessary, midwives must remain sensitive to the costs and insurance coverage of genetic counseling services and provide women with affordable options. A detailed discussion of this entire subject is found in the *Genetics* chapter.

Reproductive History

Previous adverse pregnancy outcomes suggest the need for additional attention in the preconception period. For example, women who have had a previous pregnancy complicated by complications such as preterm birth or fetal growth restriction are at risk for similar outcomes in a subsequent pregnancy. If the etiology of a previous adverse perinatal outcome is known, further assessment and therapies may be instituted preconceptionally to mitigate recurrence risks.[25,26] Health education about birth spacing, nutrition, and healthy habits often are useful in improving outcomes.

Effect of Maternal Age on Reproductive Outcomes

Declines in fertility and adverse reproductive outcomes are more likely as women become older—specifically, after age 35 years.[2] In addition, as women age, their risks for diabetes, hypertension, and other chronic diseases increase. These chronic conditions independently increase the risk for poor perinatal outcomes. Thus, it is often difficult to distinguish the risks that are secondary to age from those that are secondary to a combination of age and a chronic condition.

The effects of age on reproductive outcomes form a continuum. Although 35 years is often used as a cut-off for identifying women at increased risk, most of these risks increase incrementally over time. Based on a woman's reproductive life plan discussion with women approaching this age may consider a variety of options related to fertility and pregnancy, including egg banking and other aspects of reproductive artificial technology—topics beyond the scope of this appendix.[27]

Medical Conditions

Women who have medical conditions that can adversely affect pregnancy (e.g., diabetes, hypertension, epilepsy, autoimmune disorders) should be referred for care by a specialist prior to attempting pregnancy. **Table 5A-3** lists selected chronic health conditions that can adversely affect the course of pregnancy, though it is not all-inclusive.[25,28-34] Several of these conditions have risks associated with the disease; others require treatments that may be teratogenic or fetotoxic.

Table 5A-3 Preconception Care Interventions for Chronic Medical Conditions

Medical Condition	Evidence-Based Interventions
Asthma	Inhaled corticosteroid therapy for asthma control may be associated with low birth weight, gastroschisis, and increased risk of preeclampsia. Women with asthma should be evaluated, the condition optimally stabilized, and medications carefully chosen before a woman becomes pregnant.
Autoimmune conditions (e.g., rheumatoid arthritis, systemic lupus erythematosus)	Some medications commonly used to treat autoimmune conditions, such as methotrexate (Trexall), are teratogens. Use of nonsteroidal anti-inflammatory drugs (NSAIDs) in the first trimester does not appear to be associated with fetal anomalies; however, NSAIDs are contraindicated later in pregnancy due to their effect in keeping the ductus ateriosus open.
Cardiovascular disease	Perinatal outcomes are closely associated with the severity of the heart disease (e.g., minimal risks exist associated with mitral valve prolapse; pulmonary hypertension is potentially life threatening).
	Warfarin (Coumadin) use in pregnancy is associated with embryopathy.
Diabetes	Good glycemic control (i.e., HbA1c < 6.0%) at the time of conception lowers the risk of miscarriage and congenital anomalies that are associated with hyperglycemia.
Hypertension	Use of ACE inhibitors and ARBs is associated with increased risks of fetal malformation, oligohydramnios, fetal growth restriction, and fetal death.
	Women with long-standing hypertension may have silent ventricular hypertrophy, retinopathy, and renal disease, requiring evaluation by a specialist prior to pregnancy.
IPV	IPV has been associated with preterm birth, low birth weight, postpartum depression, and even maternal mortality. Interventions before pregnancy can focus on helping the woman find a safe environment.
Mental health conditions	Although debate continues regarding the potential teratogenic effects of some psychotropic agents, there is a general consensus that the benefits associated with treatment generally outweigh the risks because mental health disorders in pregnancy are associated with poor obstetric outcomes, higher risk of postpartum psychiatric illnesses, increased rates of substance abuse, lower participation in prenatal care, and adverse infant outcomes.
Obesity	Considered the most common of the chronic diseases, obesity during pregnancy increases maternal risks of hypertensive disorders and gestational diabetes as well as preterm birth, birth defects, and perinatal death.
	Education and counseling during the preconception period can assist a woman in losing weight in a healthy manner before attempting pregnancy. Weight-loss drugs should be avoided, as they may be associated with increased risk of congenital anomalies when used in pregnancy.
Renal disease	Because women who have severe renal disease are likely to experience a worsening of the condition during pregnancy, including severe associated conditions such as hypertension, these women should be carefully counseled by a specialist and their medications evaluated prior to

	becoming pregnant.
Seizure disorders	Many anticonvulsants are known teratogens but based on the woman's seizure pattern may be prescribed because their risks can be outweighed by their benefits.
	Preconception counseling for women affected by seizure disorders has been shown to increase the likelihood of successful withdrawal to monotherapy and to positively impact birth outcomes.
Thyroid disorders	Medications for hyperthyroidism may be associated with congenital anomalies. The general recommendation is to avoid pregnancy for 6 months after receiving radioactive treatment.
	Overt hypothyroidism during a woman's first trimester of pregnancy is associated with dwarfism and intellectual impairment. Other pregnancy complications associated with hypothyroidism include miscarriage, preterm birth, preeclampsia, placental abnormalities, and low birth weight.

Medications

Medications used by the woman, including herbal preparations, botanicals, and over-the-counter agents, should be evaluated for potential teratogenic effects, and the continuing need for the medication should be assessed. Women should not be automatically counseled to discontinue medications because they are considering pregnancy, as this may negatively affect their medical or mental health. Ideally, a plan should be in place for use of any specific medication in the preconception period and during the early stages of organogenesis. When a woman reports taking a medication that has identified risks during pregnancy, the midwife should discuss the implications with the woman and recommend safer alternative medications as available.

Preparing for Pregnancy

The decision to seek pregnancy is unique to each woman. A preconception visit enables her to optimize her health. Health is more than the mere absence of disease, and exploration about a women's level of stress, socioeconomic stability, family dynamics, support networks, and other social determinants of health can also be explored during this time.[35] Short interpregnancy spacing, as defined as 6 months or less, has been found to be an independent risk factor for adverse perinatal outcomes, and that information may influence a woman's timing of pregnancy.[36]

The preconception period is also an ideal time for women and/or couples to consider their access to and availability of health care. An essential question to consider at a preconception visit is whether the woman wishes to see a midwife, obstetrician-gynecologist, family practice physician, or maternal–fetal medicine specialist when pregnant. Place of birth and provider options may be controlled by the woman's insurance carrier and subject to certain limitations. If couples investigate these options early, they may be able to make the arrangements for the birth provider and the environment they need and prefer.

While most midwives provide little care for men, male partners of a woman planning a pregnancy should also be encouraged to develop a reproductive life plan. Should they have a history of personal or family health conditions, they should also obtain an evaluation of their health and potentially genetic counseling.

Although many providers recommend using a barrier method for a set period of time before discontinuing contraception completely, this recommendation is based on the convenience of establishing a normal menstrual period that can be used to determine an estimated date for birth rather than evidence that pregnancy outcomes are improved if conception is delayed. After using a hormonal method of contraception, the first menses may be anovulatory or ovulation may occur quickly. When ovulation does occur quickly, a woman might conceive prior to having a menstrual period, which makes gestational dating by last menstrual period less reliable. Women should be reassured that conceiving prior to the first menstrual period is not associated with an increased risk of spontaneous abortion or congenital anomalies.

As a general guideline, a woman who is seeking pregnancy, engaging in regular intercourse, and not using any contraceptive method will usually become pregnant within a year. If pregnancy does not ensue after 12 months, an infertility evaluation for the woman and her partner can be initiated. Alternatively, some women age 35 years or older may wish to seek infertility care after only 6 months of failing to become pregnant because fertility wanes with increasing age.

Conclusion

Preconception is an important topic for healthcare providers and women alike. A healthy woman is more likely to have a healthy newborn. Health care during the preconception period verifies that a woman is healthy; provides an opportunity to treat a woman who has a disorder; and promotes health education, counseling, and habits that are congruent with a healthy pregnancy, and birth. Preconception care does not need to be pronatalistic or cost unnecessary time in the clinical area. By using the One Key Question, care can be focused appropriately on the care of the woman during the clinical encounter.

Resources

Organization	Description	Webpage
Centers for Disease Control and Prevention (CDC)	U.S. government site with specific information regarding preconception care. Includes download of the free consumer-oriented app *Show Your Love* to help women maintain healthy habits, chart ovulation, and plan for pregnancy.	https://www.cdc.gov/preconception/index.html
National Preconception Health and Health Care Initiative Website	Partnership among more than 70 public and private organizations to promote preconception health care. Source of extensive materials and information.	https://beforeandbeyond.org

References

1. American College of Nurse-Midwives. Position statement: the role of the certified nurse-midwife/certified midwife in

preconception health and health care. 2013. Available at: http://midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000081/Preconception%20Health%20 Accessed October 9, 2017.
2. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Prepregnancy care. In: *Guidelines for Perinatal Care*. 8th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2017:131-148.
3. Burgess CK, Henning PA, Norman WV, Manze MG, Jones HE. A systematic review of the effect of reproductive intention screening in primary care settings on reproductive health outcomes. *Fam Pract*. September 8, 2017. [Epub ahead of print]. doi:10.1093/fampra/cmx086.
4. Files JA, Frey KA, David PS, Hunt KS, Noble BN, Mayer AP. Developing a reproductive life plan. *J Midwifery Womens Health*. 2011;56(5):468-474.
5. Lang AY, Boyle JA, Fitzgerald G, et al. Optimising preconception health in women of reproductive age. *Minerva Ginecol*. 2018;70(1):99-119.
6. Nypaver C, Arbour M, Niederegger E. Preconception care: improving the health of women and families. *J Midwifery Womens Health*. 2016;61(3):356-364.
7. Freda MC, Moos MK, Curtis M. The history of preconception care: evolving guidelines and standards. *Matern Child Health J*. 2006;10(5 suppl):S43-S52.
8. Waggoner MR. Motherhood Preconceived: the emergence of the Preconception Health and Health Care Initiative. *J Health Polit Policy Law*. 2013;38(2):345-371.
9. Moos MK. From concept to practice: reflections on the preconception health agenda. *J Womens Health (Larchmt)*. 2010;19(3):561-567. doi:10.1089/jwh.2009.1411.
10. National Preconception Health and Health Care Initiative. Before, between and beyond pregnancy. Available at: https://beforeandbeyond.org/. Accessed October 9, 2017.
11. Kessler JL. A literature review on women's oral health across the life span. *Nurs Womens Health*. 2017;21(2):108-121.
12. Coonrod DV, Jack BW, Boggess KA, et al. The clinical content of preconception care: immunizations as part of preconception care. *Am J Obstet Gynecol*. 2008;199(6 suppl 2):S290-S295.
13. Sukumaran L, McNeil MM, Moro PL, Lewis PW, Winiecki SK, Shimabukuro TT. Adverse events following measles, mumps, and rubella vaccine in adults reported to the Vaccine Adverse Event Reporting System (VAERS), 2003–2013. *Clin Infect Dis*. 2015;60(10):e58-e65.
14. Gardiner PM, Nelson L, Shellhaas CS, et al. The clinical content of preconception care: nutrition and dietary supplements. *Am J Obstet Gynecol*. 2008;199(6 suppl 2):S345-S356.
15. U.S. Preventive Services Task Force. Folic acid for the prevention of neural tube defects: preventive medication. 2017. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryFinal/folic-acid-for-the-prevention-of-neural-tube-defects-preventive-medication. Accessed September 1, 2017.
16. American College of Obstetricians and Gynecologists. Committee Opinion 495: vitamin D: screening and supplementation during pregnancy. *Obstet Gynecol*. 2011;118(1):197-198. [Reaffirmed 2017].
17. American College of Obstetricians and Gynecologists Committee on Health Care for Underserved Women, American Society for Reproductive Medicine Practice, Committee, The University of California, San Francisco, Program on Reproductive Health and the Environment. *Exposure to Toxic Environmental Agents*. Washington, DC: American College of Obstetricians and Gynecologists; 2013.
18. Di Renzo GC, Conry JA, Blake J, et al. International Federation of Gynecology and Obstetrics opinion on reproductive health impacts of exposure to toxic environmental chemicals. *Int J Gynaecol Obstet*. 2015;131(3):219-225.
19. Furst P. Dioxins, polychlorinated biphenyls and other organohalogen compounds in human milk: levels, correlations, trends and exposure through breastfeeding. *Mol Nutr Food Res*. 2006;50(10):922-933.
20. Gao Y, Chen H, Xiao X, et al. Perfluorooctanesulfonate (PFOS)-induced Sertoli cell injury through a disruption of F-actin and microtubule organization is mediated by Akt1/2. *Sci Rep*. 2017;7(1):1110.
21. Project TENDR. Project TENDR: Targeting Environmental Neuro-Developmental Risk. The TENDR consensus statement. *Environ Health Perspect*. 2016;124(7):A118-A122.
22. Rebelo FM, Caldas ED. Arsenic, lead, mercury and cadmium: toxicity, levels in breast milk and the risks for breastfed infants. *Environ Res*. 2016;151:671-688.
23. Solomon GM, Weiss PM. Chemical contaminants in breast milk: time trends and regional variability. *Environ Health Perspect*. 2002;110(6):A339-A347.
24. Woodruff TJ, Zota AR, Schwartz JM. Environmental chemicals in pregnant women in the United States: NHANES 2003–20004. *Environ Health Perspect*. 2011;119(6):878-885.
25. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Medical and obstetric complications. In: *Guidelines for Perinatal Care*. 8th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2017:301-346.
26. Zhang G, Feenstra B, Bacelis J, et al. Genetic associations with gestational duration and spontaneous preterm birth. *N Engl J Med*. 2017;377(12):1156-1167.

27. Stevenson EL, Hurt MJ, Trotter KJ. Oocyte cryopreservation for fertility preservation in healthy women. *Nurs Womens Health*. 2017;21(5):384-392.
28. Bramham K, Lightstone L. Pre-pregnancy counseling for women with chronic kidney disease. *J Nephrol*. 2012;25(4):450-459.
29. Centers for Disease Control and Prevention. Preconception health and healthcare: medical conditions. Available at: https://www.cdc.gov/preconception/careforwomen/conditions.html. Accessed October 9, 2017.
30. Lassi ZS, Imam AM, Dean SV, Bhutta ZA. Preconception care: screening and management of chronic disease and promoting psychological health. *Reprod Health*. 2014;11(suppl 3):S5.
31. Marchi J, Berg M, Dencker A, Olander EK, Begley C. Risks associated with obesity in pregnancy, for the mother and baby: a systematic review of reviews. *Obes Rev*. 2015;16(8):621-638.
32. Coonrod DV, Jack BW, Stubblefield PG, et al. The clinical content of preconception care: infectious diseases in preconception care. *Am J Obstet Gynecol*. 2008;199(6 suppl 2):S296-S309.
33. Dunlop AL, Gardiner PM, Shellhaas CS, Menard MK, McDiarmid MA. The clinical content of preconception care: the use of medications and supplements among women of reproductive age. *Am J Obstet Gynecol*. 2008;199(6 suppl 2):S367-S372.
34. Frieder A, Dunlop AL, Culpepper L, Bernstein PS. The clinical content of preconception care: women with psychiatric conditions. *Am J Obstet Gynecol*. 2008;199(6 suppl 2):S328-S332.
35. Brucker MC. Social determinants of health. *Nurs Womens Health*. 2017;21(1):7-8.
36. Hanley GE, Hutcheon JA, Kinniburgh BA, Lee L. Interpregnancy interval and adverse pregnancy outcomes: an analysis of successive pregnancies. *Obstet Gynecol*. 2017;129(3):408-415.

CHAPTER

6

Common Conditions in Primary Care

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Introduction

Historically, midwives have been healers in their communities. In addition to attending births, a midwife often was the herbalist, first responder, and health counselor. Thus, midwives were among the original primary care providers. Today, midwives continue to be primary care providers and are often the first point of contact for women in the healthcare system.[1] Midwives have the training and expertise to conduct screenings, counsel and monitor women in making healthy behavior changes, diagnose and manage minor illnesses and some chronic diseases, and determine to whom and how to refer to other experts in the field as more complicated situations arise. This chapter is an introduction to health problems commonly experienced by women. More in-depth information can be obtained in primary care texts that address these conditions.

Primary Care

No universally accepted definition of “primary care” exists. Variations exist among insurers, providers, professional organizations, and consumers. Perhaps the most widely accepted definition is the one issued in 1996 by the Institute of Medicine, which characterized primary care as:

> *The provision of integrated, accessible health care services by clinicians who are accountable for addressing a large majority of personal health care needs, developing a sustained partnership with patients, and practicing in the context of family and community.*[2]

For some individuals, the “large majority” has been defined as provision of 80% of the care needed by a person annually. However, this definition of primary care fails to highlight the importance of health maintenance, age-appropriate screening, and health education—all activities are that are discussed in depth in the *Health Promotion Across the Lifespan* chapter.

It is increasingly appreciated that social determinants of health can dramatically affect the health of a woman and her family. Organizations such as the Centers for Disease Control and Prevention (CDC), National Institutes of Health (NIH), and Health Resources and Services Administration (HRSA) have called for integration of population health into primary care with the goal of creating a more unified approach to care.[3] This chapter reviews care of women who are not pregnant, but who have disorders or conditions that can be characterized as in the realm of primary care rather than gynecologic care.

Hematologic Conditions

Anemia

Anemia is defined as a decrease in red blood cell mass or decrease in hemoglobin. Most often, anemia remains a “silent” condition unless it is acute or severe. Signs and symptoms of anemia are listed in **Table 6-1**.[4] Anemia can be caused by decreased red blood cell production, increased red blood cell destruction, or blood loss, and is diagnosed in nonpregnant women when the hemoglobin level is less than 12.0 g/dL.[5] This cut-off was established by the World Health Organization for use in international nutrition studies and does not apply to all populations. For example, African American women have, on average, hemoglobin levels approximately 1 g/dL lower than those in whites, regardless of socioeconomic level.[5] Women who smoke (because of competition for oxygen-binding sites on red blood cells) and women living at high altitudes (because of lower oxygen concentration in the atmosphere) have higher hemoglobin and hematocrit levels: Their bodies adapt to maintain adequate oxygenation and their hemoglobin levels increase as the altitude or number of cigarettes smoked per day increases.[5] Thus, the hemoglobin level that indicates anemia may vary based on an individual’s personal health profile. The U.S. Preventive Services Task Force does not address whether screening for anemia should be a routine part of health care for nonpregnant women.[6]

Table 6-1 **Signs and Symptoms of Severe Anemia**

Signs	Symptoms
Pallor	Fatigue, drowsiness
Jaundice	Weakness
Orthostatic hypotension	Dizziness
Peripheral edema	Headaches
Pale mucous membranes and nail beds	Malaise
Smooth, sore tongue	Pica
Splenomegaly	Poor appetite, changes in food preferences
Tachypnea, dyspnea on exertion	Changes in sleep habits
Tachycardia or flow murmur	Changes in mood

Differential Diagnosis of Anemia

Anemia can be acquired as occurs in iron deficiency, or hemorrhagic anemia, or it can be inherited as occurs in persons with hemoglobinopathies, such as thalassemia or sickle cell disease. Anemia can be subclassified different ways. Subclassification by etiology refers to anemia secondary to decreased red blood cell production or increased red blood cell destruction. Alternatively, anemia can be subclassified as normocytic, microcytic, or macrocytic depending on the amount and type of hemoglobin present in red blood cells as reflected in the mean corpuscular volume (MCV). The clinical evaluation and diagnosis of anemia is based on MCV, which is how this chapter reviews this condition.

Anemia is commonly categorized by the size of the red blood cells. *Microcytic anemias* include iron deficiency, the thalassemias, and anemia of inflammation. *Macrocytic anemias* include folate and vitamin B12 deficiency, as well as anemia associated with liver disease, increased reticulocyte production, and some medication effects. *Normocytic anemias* commonly reflect acute blood loss or conditions such as sickle cell disease, hemoglobin C disease, or glucose-6-phosphate dehydrogenase (G6PD) deficiency. Aplastic anemia, while normocytic, is characterized by pancytopenia, meaning a reduction in the number of red blood cells (RBCs), white blood cells (WBCs), and platelets. **Table 6-2** lists the laboratory values associated with some common causes of anemia.

Table 6-2 Laboratory Values Associated with Common Anemias

Laboratory Test	Iron Deficiency	Vitamin B_{12} Deficiency	Folate Deficiency	Thalassemia	Chronic Disease
Red blood cells (RBCs)	Low	Low	Low	Normal	Low
Hemoglobin (Hgb)	Low	Low	Low	Low	Low
Mean corpuscular volume (MCV)	Low	High	High	Low	Normal-low
Mean corpuscular hemoglobin (MCH)	Low	High	High	Low	Low
Reticulocytes	Low	Low	Low	High	Low
Ferritin	Low	High	High	Normal–high	Normal–high
Iron	Low	High	High	Normal–decreased	Low
Total iron-binding capacity (TIBC)	High	Normal	Normal	Variable	Low
Other		Low vitamin B_{12}	Low folate	Teardrop red cells	
		Elevated methylmalonate	Normal methylmalonate	Target cells	
		Elevated homocysteine	Elevated homocysteine	Abnormal hemoglobin electrophoresis	

A complete blood count (CBC) provides the first level of assessment and helps differentiate many of the underlying causes of anemia. If a woman has menstrual periods, the history and review of systems should include obtaining a history of how heavy her menstrual flow is, even though the accuracy of women's estimation of their flow has been demonstrated to vary. A prior diagnosis of anemia merits a description of the circumstances and helps guide the differential diagnosis. For women with a hemoglobin value of less than 12.0 g/dL, a laboratory panel including serum folate and ferritin measurements should be ordered, and a hemoglobin electrophoresis performed. The ferritin level is the most sensitive and specific predictor of iron stores and, therefore, true iron deficiency.

Iron-Deficiency Anemia

The most common anemia in the United States is due to iron deficiency, which usually is mild and easily reversible. The Recommended Daily Intake (RDI) of iron for reproductive-age women is 15 mg for girls from 14 to 18 years and 18 mg for women from 19 to 50 years.[7] Following menopause, the RDI drops to 8 mg. The diets of most women in the United States include approximately 13–14 mg of iron daily. Normal daily iron loss through excretion, sweat, and cellular shedding amounts to 1 mg; menses causes an additional monthly loss. Pregnancy-related demands increase the daily iron need among reproductive-age women. RDI for pregnant women set at 27 mg per day.

Occult blood loss, excessive menstrual loss, and inadequate nutritional intake which is more likely among vegetarians, are the most common causes of iron deficiency in adults. If nutritional deficiency is ruled out and in the absence of an identifiable source of bleeding such as heavy menses, inquiry about use of aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) as well as assessment for gastrointestinal bleeding is warranted. Nutritional deficiencies causing significant iron depletion include restrictive vegetarian diets as well as pica; therefore, a careful diet history is part of the work-up.

A ferritin level less than 100–150 ng/mL confirms the diagnosis of iron-deficiency anemia and renders serum iron and total iron-binding capacity measurements unnecessary. Based on the severity of the anemia and its cause, consultation or referral may be indicated; when the hemoglobin indicates severe anemia (< 9.0 g/dL), consultation is appropriate, even when the anemia is clearly caused by iron deficiency.

First-line treatment is to increase dietary intake of iron-rich foods, along with consumption of vitamin C–enriched products to enhance absorption of the heme iron. Nutritional counseling should stress the importance of including iron-rich foods in the diet—such as green leafy vegetables, collard greens, egg yolks, raisins, prunes, liver, oysters, and some fortified cereals—as well as the elimination of picas (e.g., eating ice or laundry starches). A detailed discussion of dietary sources of iron can be found in the *Nutrition* chapter.

Iron supplementation, either through the diet or with oral medications, should be recommended for a nonpregnant adult woman when the hemoglobin is less than 12.0 g/dL.[7] When uncomplicated iron deficiency is the cause of the anemia, oral medications are used empirically, including ferrous sulfate, ferrous fumarate, and ferrous gluconate. Each of these preparations is readily available and marketed under several brand names. The equivalent of 325 mg of ferrous sulfate taken three times a day is standard dosing. Generally taking iron preparations with meals will decrease absorption of this nutrient, but will also improve gastrointestinal side effects such as nausea and reflux. Certain drugs, such as antacids, decrease the absorption of iron supplements and should not be taken at the same time. After the hemoglobin level has returned to normal, continued supplementation for 3 months should adequately replenish iron stores in the body.

While no clear standard for follow-up exists, the CBC and reticulocyte count are generally repeated 1–3 months following treatment initiation, depending on the severity of the original deficiency and the age of the woman. Response to iron supplementation is often rapid in cases of uncomplicated iron-deficiency anemia, as evidenced by rebounding hemoglobin and

hematocrit levels and elevated reticulocyte counts. Premenopausal women who are unresponsive to iron therapy and all postmenopausal women with iron-deficiency anemia should be referred for a consultation with a gastrointestinal (GI) specialist. Women may also require referral to a hematologist for further evaluation and possible treatment with intravenous iron therapy if they are unable to tolerate or absorb oral iron therapy. In extreme cases, women may need to be referred urgently to the emergency room for immediate evaluation or blood transfusion if their anemia is severe or symptomatic.

Hemoglobinopathies: Thalassemias

Normal adult hemoglobin is composed of four polypeptide subunits: two alpha-globin chains and two beta-globin chains. A hemoglobinopathy occurs when one or two of the subunits are replaced by a variant type of globin chain. Alpha- or beta-thalassemia occurs when deficient amounts of alpha or beta globin chains are made. Common hemoglobinopathies and thalassemias are listed in **Table 6-3**.

Table 6-3 Disorders of Abnormal Hemoglobin

Name	Description	Clinical Significance
Hemoglobinopathies		
Sickle cell disease	Hb SS	Severe illness with sickle cell crisis
Sickle cell trait	Hb SA	Mild anemia; increased risk for urinary tract infections; sickle cell crisis may occur at high altitudes, if dehydrated, or during extreme physical activity
Sickle cell hemoglobin C disease	Hb SC	Mild to moderate anemia and sickle cell crisis occur but less frequently than in persons with Hb SS; increased risk for infection; risk for retinopathy and blindness in adulthood
Hemoglobin C disease	Hb CC	Usually asymptomatic, may have symptoms associated with splenomegaly. No therapy needed; normal life expectancy.
Hemoglobin C trait	HB CA	Usually asymptomatic
Thalassemias: Alpha-Thalassemias		
Alpha-thalassemia silent carrier	1 of 4 alpha genes deleted	Asymptomatic and difficult to detect
Alpha-thalassemia trait	2 of 4 alpha genes deleted; heterozygous form (i.e., aa/– –) or homozygous form (i.e., a–/a–)	Mild microcytic anemia; individual is asymptomatic
Hemoglobin H disease	3 of 4 alpha genes deleted (i.e., a–/– –), or 2 of 4 alpha genes deleted and the third mutated to the constant spring (cs) form (i.e., – –/aacs)	Enlarged spleen; bone abnormalities; severe illness
Hemoglobin	4 of 4 alpha genes deleted (i.e., – –/– –)	Incompatible with life; causes hydrops fetalis

Bart's hydrops fetalis		
Thalassemias: Beta-Thalassemias		
Beta-thalassemia (Cooley's anemia)	Beta-thalassemia major, homozygous Beta0/Beta0 or heterozygous Beta0/Beta+[a], leading to absence of beta-chain formation	Severe anemia; jaundice; splenomegaly; requires frequent blood transfusions; often experience iron overload. Classic facial features.
Thalassemia intermedia	Beta+/Beta+, resulting in decreased beta-chain formation	Significant anemia but does not need blood transfusions
Thalassemia minor	Normal beta-chain/Beta+ or Beta0	Mild microcytic anemia; individual is asymptomatic

Abbreviations: Beta0, absence of beta-globin formation; Beta+, reduced beta-globin formation.

[a] Depending on the authority, Beta0/Beta+ has been classified as either beta-thalassemia major or intermedia. It is generally less severe than the Beta0/Beta0 presentation because some beta-chain production is possible with Beta+ mutations.

Most thalassemias are autosomal recessive inherited disorders of the globin chains that form normal adult hemoglobin (hemoglobin A). Worldwide, approximately 1.67% of the population is heterozygous for either alpha-thalassemia (α-thalassemia) or beta-thalassemia (β-thalassemia), and approximately 0.044% of the global population is affected by homozygous or multiple heterozygous mutations.[8] However, due to increased immigration from parts of the world where the thalassemias are more prevalent (Southeast Asia, the Mediterranean, Africa, Middle East, and the Indian subcontinent), the prevalence of these conditions has increased by 7.5% in the United States over the last 50 years.[9]

Alpha-thalassemia is most common among individuals of Chinese and Southeast Asian descent. Two genes on chromosome 16 control for alpha-globin chain production. A single deletion will create an asymptomatic carrier state; two deletions will cause a smaller MCV without anemia; and the three-deletion state leads to increased hemoglobin H with effects including splenomegaly and severe hemolytic anemia. The absence of all four alpha chains and the resultant increase in hemoglobin B produces non-immune fetal hydrops, which results in fetal demise.[10]

Beta-thalassemia is most common among women of Mediterranean origin and, to a lesser degree, among Chinese, Asian, and African women. Estimates of its frequency range from 3% to 10%.[11] More than 200 different point mutations can be involved in the beta-globin changes associated with thalassemia, leading to a wide spectrum of disease. Asymptomatic individuals with a mutation affecting a single beta-globin gene may remain in a silent carrier state or may present with only mild anemia (beta-thalassemia minor). For these women, the diagnosis in most likely made based on routine screening or incidental findings. Women with more severe disease—that is, beta-thalassemia intermediate and beta-thalassemia major—have mutations affecting multiple genes. The two conditions are differentiated by the age at diagnosis and degree of anemia, and both require lifelong therapy.[10,11]

The diagnosis of thalassemia is often made in childhood in persons in whom more genes are affected. However, milder presentations may not be identified until later in life, and the

diagnosis is often made for the first time in pregnancy when screening for hemoglobinopathies is commonly performed. Identification of thalassemia in pregnancy is important because a woman with thalassemia, sickle trait, or sickle disease could give birth to a child with a severe hemoglobinopathy if the biological father also is a carrier for abnormal hemoglobin.

Several laboratory findings may suggest the presence of a thalassemia. Among persons with alpha-thalassemias, decreased hemoglobin alpha production causes abnormal proportions of hemoglobins A, A2, and F. The beta-thalassemias are associated with elevated levels of hemoglobin F and hemoglobin A2 (greater than 3.5%). In both cases, the trait will appear as a microcytic anemia in which the MCV is markedly low relative to the hemoglobin level, usually less than 75 femtoliters (fL). The Metzer index is a calculation derived by dividing the MCV by the number of RBCs. When the Metzer index is less than 13, a thalassemia is strongly suspected. Values more than 13 suggest iron-deficiency anemia. Even with values less than 13, a complete anemia panel is justified to rule out a combination of iron deficiency and hemoglobinopathy.

When the diagnosis of thalassemia is established, folic acid supplementation may be employed but iron therapy is inappropriate. The anemia associated with the thalassemias is due to a combination of low hemoglobin production and mild hemolysis, since the abnormal RBCs are more vulnerable to destruction and have a shorter lifespan. Consequently, women with thalassemia may not be iron deficient. Simple iron supplementation will not correct the anemia and can be dangerous if it contributes to iron overload.[10]

Hemoglobinopathies: Sickle Cell Disease

Sickle cell disease (homozygous SS disease) is an autosomal recessive inherited disorder in which hemoglobin S is produced instead of hemoglobin A. Sickle cell trait (Hb AS) is found most commonly among African Americans compared to individuals of other racial and/or ethnic backgrounds. The incidence of sickle cell trait in the United States is estimated to be 73.1 per 1000 black births, 6.9 cases per 1000 Hispanic births, 3.0 per 1000 white births, and 2.2 per 1000 Asian/Native Hawaiian/other Pacific Islander births.[12] Sickle cell trait is rarely associated with hematuria, bacteriuria, and splenic infarct. Although the trait itself is often asymptomatic and does not commonly cause severe health complications, identification of those persons carrying the sickle trait is important to enable appropriate genetic counseling and testing prior to pregnancy. The primary complication for the nonpregnant woman is an increase in the development of urinary tract infections. Interestingly, sickle cell trait may lessen the accuracy of hemoglobin A1C testing, making screening for diabetes in individuals with sickle trait more difficult.

The estimated incidence of sickle cell disease is 1 in 375 births for African Americans and 1 in 16,300 Hispanic births.[13] In sickle cell disease, the Hb S changes shape (polymerizes) when deoxygenated, which causes the RBCs to form a permanent crescent moon or “sickle” shape, such that the RBCs are less able to bend their shape as they traverse through smaller vessels. These sickle-shape RBCs are also more adhesive and clump together to block the microvasculature. Sickle cell crises involve acute episodes of severe pain from ischemia and infarction of tissue and organs downstream from the area of vascular blockage. The disease

has a multi-organ effect and is associated with a shortened lifespan as a consequence of renal damage, cardiac damage, infection, acute chest syndrome, and increased risk for infection. Early death is common. Among a sample of individuals with sickle cell disease living in California and Georgia, the average age of death was 43 years. Approximately 1 out of 6 deaths occurred among individuals younger than 25 years.[14]

Hemoglobin S may also be present in heterozygous form as Hb SC disease or sickle thalassemia (Hb S-B thalassemia), conditions that are associated with somewhat milder forms of sickle crisis. When hemoglobinopathies exist, genetic counseling for couples planning a child is recommended.

Glucose-6-Phosphate Dehydrogenase Deficiency

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an X-linked genetic disease found most frequently among individuals of Mediterranean descent and African Americans. Because it is an X-linked condition, G6PD deficiency is rarely symptomatic among women. Even among individuals who have the condition, the clinical presentation ranges from being asymptomatic to presenting with severe acute or chronic hemolytic anemia. Hemolysis occurs when the individual has an infection or receives oxidative drugs. Among the contraindicated medications for women with this disorder are sulfa drugs and sulfa derivatives, nitrofurantoin (Macrobid), nonsteroidal anti-inflammatory drugs (NSAIDs), toluidine blue, and methylene blue. Consumption of fava beans and some other legumes should be avoided as well, because these foods can produce hemolysis among individuals with the Mediterranean variant. Prompt diagnosis and treatment of infection will minimize the risk of hemolysis. Surgery can also precipitate an episode of hemolysis. Therefore, the midwife should either notify the woman's surgical team prior to any surgical procedure or counsel the woman to do so.

Von Willebrand's Disease

Von Willebrand's disease is an autosomal dominant mutation that causes defects in a protein necessary for platelet adhesion and affects clotting factor VII.[15] This condition is related to development of heavy menses approximately 20% of the time, particularly among adolescents.[15] Von Willebrand's disease or another bleeding disorder should be suspected if the woman reports heavy menstrual bleeding in association with prolonged bleeding after surgery or a family history of bleeding problems.[16] The initial assessment includes obtaining a platelet count, ferritin level, and bleeding time and coagulation studies. When platelets are normal, the ferritin level is low, and the bleeding time is prolonged, von Willebrand's disease is a strong possibility. Although the midwife may continue to provide general care, referral of the woman to a hematologist for evaluation is warranted. Women with this condition should avoid aspirin or other agents with anticoagulant properties.

Cardiovascular Conditions

Assessment of a woman's cardiovascular health begins with a history and review of systems that assess for hypertension, cardiac events, and vascular changes in the woman and her immediate family. Assessment of heart sounds and of the pulses during the physical examination is essential, as is an accurate measurement of blood pressure.

Cardiovascular disease is the leading cause of death among women and ranks within the top five causes of death for women of reproductive age.[17] Various forms of cardiovascular disease, including stroke, account for almost 30% of deaths among women. Risk factors for cardiovascular disease include hypertension, dyslipidemia, diabetes, obesity, physical inactivity, and smoking. Simple aging is noted as a risk because 20% of women age 65 years or older have some form of heart disease.

Hypertension

Hypertension—an arterial disease characterized by persistent high blood pressure—is the most frequent diagnosis associated with primary care visits in the United States.[18] Hypertension is a major risk factor for a number of conditions such as coronary artery disease, stroke, heart failure, and renal failure. It is more common among African Americans and among the elderly. More than 30% of all adults in the United States have high blood pressure and the risk increases with age.[19,20] Hypertension is categorized as primary or secondary. Primary hypertension, which is the cause of the majority of hypertension, is thought to be secondary to factors such as genetics, age, and obesity. Secondary hypertension is less common and occurs as the result of another disorder such as a renal or heart condition. A third variation of hypertension is white coat hypertension, which refers to the observation that a person's blood pressure is higher in the presence of a healthcare provider. The prevalence of white coat hypertension ranges from 13% to 35% and is therefore a condition that must be in the differential diagnosis.[20] A fourth variation called masked hypertension refers to individuals who have normal blood pressure during an office visit but hypertension at home. Masked hypertension may occur in 1 in 7 persons. Definitions for blood pressure are listed in **Table 6-4**.20 An estimated 80% of women diagnosed with high blood pressure take medication, although only slightly more than half of those have well-controlled blood pressure.[21] Lack of awareness of a diagnosis of high blood pressure and failure to be treated effectively have been associated with lack of access to care and lack of health insurance.[22]

Table 6-4 **Classification of Blood Pressure for Adults**

Blood Pressure Classification	Systolic (mm Hg)		Diastolic (mm Hg)	Corresponding Values
Normal	< 120	and	< 80	Office reading of 120/80 equals: Home: 120/80 Nighttime ABPM: 100/65
Elevated	120–129	or	< 80	Not defined
Stage 1 hypertension	130–139	or	80–89	Office reading of 130/80 equals: Home: 130/80 Nighttime ABPM: 110/65
Stage 2 hypertension	≥ 140	or	≥ 90	Office reading of 140/90 equals: Home: 140/90 Nighttime ABPM: 140/85 Office reading of 160/100 equals: Home: 140/95 Nighttime ABPM: 140/90

Abbreviation: ABPM, ambulatory blood pressure monitoring.

Based on Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension.* 2017. doi:10.1016/j.jacc.2017.11.006. [Epub ahead of print].[20]

Diagnosis of Hypertension

Several professional organizations in the United States have published guidelines for care of persons with hypertension, which has led to some confusion. The most widely accepted guidelines for the diagnosis and treatment of hypertension were originally established by the Joint National Committee (JNC).[23,24] In 2014, the Seventh Joint National Committee (JNC 7) guidelines were vigorously reviewed by the panel members of the Eighth Joint National Committee (JNC 8), however, the JNC 8 panel was discontinued by the National Heart Lung and Blood Institute. The panel members published their recommendations which focus on pharmacologic management of persons with hypertension.[23] In order to resolve confusion, in 2017, the American Heart Association in conjunction with multiple other professional organizations updated the JNC 7 and published new guidelines for detection, evaluation and management of high blood pressure in adults that are presented in **Table 6-4**.[20]

The initial steps in screening and diagnosis of hypertension are: (1) accurate assessment of blood pressure; (2) identification of signs that suggest primary versus secondary hypertension; (3) screening for other cardiovascular risk factors; and (4) assessment for possible white coat hypertension or masked hypertension.

Accurate Measurement of Blood Pressure. Many clinicians take action based on blood pressure results that are inaccurate because the technique of assessment is incorrect. Blood pressures should always be taken with the cuff at the level of the heart while using a properly

sized cuff. The woman should have had 10 minutes to sit quietly and should not have ingested tobacco or caffeine for at least 30 minutes prior to measuring the blood pressure. She should be seated with her back against a chair, her legs uncrossed, and her forearm resting comfortably on a table. The diagnosis should be made on an average of two or more readings taken on two or more occasions. Out of office or home blood pressure readings are recommended for the confirmation of this diagnosis. If hypertension is diagnosed, the woman should be referred to a physician to establish a plan for care and monitoring.

Signs That Suggest Primary Versus Secondary Hypertension. Signs that suggest primary hypertension include family history, slowly increasing blood pressure over time, and lifestyle factors such as obesity, smoking, low level of physical activity, and excessive use of alcohol. Signs that suggest hypertension may be secondary include liable blood pressure, other systemic symptoms such as dizziness, snoring, muscle cramps or weakness, weight loss, edema, fatigue, or weakness. Disorders that are associated with secondary hypertension include hyperthyroidism, kidney disease, Cushing's syndrome, and medications such as cocaine, amphetamines, and NSAIDs.[20]

Atherosclerotic Cardiovascular Disease Risk. Screening for cardiovascular risks is a critical component of this evaluation because the benefit of pharmacologic management of hypertension is directly related to the risk for atherosclerotic cardiovascular disease (ASCVD). Pharmacologic treatment is recommended for persons with a systolic blood pressure of 130 mm Hg or higher or an average diastolic blood pressure of 80 mm Hg or higher.[20] Cardiovascular risk calculators are listed in the Resource at the end of this chapter.

White Coat Hypertension and Masked Hypertension. The 2017 guidelines include recommendations for screening individuals for white coat hypertension. White coat hypertension is the diagnosis for persons who have hypertension in an office setting but no hypertension at home. There are no data about the benefits or risks associated with treating persons with white coat hypertension.

Masked hypertension is not common but should be included in the differential diagnosis if the person reports a history of occasional hypertension but is normotensive during a primary care visit. Home blood pressure monitoring or ambulatory blood pressure monitoring can be helpful in establishing this diagnosis.

Laboratory Evaluation. If hypertension is detected, laboratory evaluation includes tests that will assess for complications of hypertension and the presence of comorbidities. Recommended laboratory evaluations include fasting blood glucose, thyroid indices, serum creatinine, lipid profile, serum sodium, potassium, and calcium, which may be part of a metabolic panel, and electrocardiogram.

Treatment of Primary Hypertension

Table 6-5 outlines the general management of persons with hypertension after the initial evaluation.[20] Many cases of primary hypertension are related to lifestyle, rather than underlying disease. These causes are directly modifiable through lifestyle changes such as weight loss, nutritional counseling for a low-sodium diet, exercise, smoking cessation, and moderation in alcohol consumption.[22,24,25] A weight loss of as little as 10 pounds may have a salutary effect on blood pressure. **Table 6-6** demonstrates the effect of lifestyle modification on blood pressure as reported by the JNC 7 and supported by the 2017 guidelines.[20,24] Achieving more than one modification has an independent effect on blood pressure so a combination of modifications can achieve better results.[25]

Table 6-5 Overview of Treatment and Recommended Follow-Up Following Initial Diagnosis of Hypertension

Diagnosis	Treatment and Recommended Follow-Up
Normotensive	Promote optimal lifestyle and repeat blood pressure evaluation annually
Elevated blood pressure	Non-pharmacologic lifestyle modifications and repeat evaluation in 3–6 months
Stage 1 hypertension with 10-year estimated ASCVD risk < 10%	Non-pharmacologic lifestyle modifications and repeat evaluation in 3–6 months
Stage 1 hypertension with 10-year estimated ASCVD risk > 10%	Non-pharmacologic lifestyle modifications and anti-hypertensive medication. Repeat evaluation in 1 month
White coat hypertension	If blood pressure is ≥ 130 mm Hg but <160 mm Hg systolic or > 80 mm Hg but < 100 mm Hg, HBPM or ABPM can be used to establish the diagnosis; if white coat hypertension is present, life style modification and annual monitoring with HBPM or ABPM to detect sustained hypertension
Masked hypertension	If office blood pressure is 120–129/80 mm Hg but the individual has a history of hypertension, HBPM or ABPM can be used to establish the diagnosis; if daytime HBPM or ABPM are ≥ 130/80 mm Hg, institute lifestyle modification and start antihypertensive medication

Abbreviations: ABPM, ambulatory blood pressure monitoring; ASCVD, atherosclerotic cardiovascular disease; HBPM, home blood pressure monitoring.

Based on Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension.* 2017. [Epub ahead of print]. doi:10.1016/j.jacc.2017.11.006.[20]

Table 6-6 Lifestyle Modifications to Prevent and Manage Hypertension[a]

Modification	Recommendation	Approximate SBP Reduction (Range)[b]
Weight reduction	Maintain normal body weight (BMI: 18.5–24.9 kg/m^2)	5–20 mm Hg/10 kg

Adopt DASH eating plan	Consume a diet rich in fruits, vegetables, and low-fat dairy products with reduced saturated and total fat, and reduce dietary sodium to ≤ 100 mmol/day (2.4 g of sodium)	8–14 mm Hg
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)	4–9 mm Hg
Moderation of alcohol[c]	Men: No more than 1 oz of ethanol per day (2 drinks) Women: No more than 0.5 oz of ethanol per day (1 drink)	2–4 mm Hg

Abbreviations: BMI, body mass index; DASH, Dietary Approaches to Stop Hypertension; SBP, systolic blood pressure.

[a] For overall cardiovascular risk reduction, stop smoking.

[b] The effects of implementing these modifications are dose and time dependent, and could be greater for a specific individual.

[c] Ethanol in a standard drink: 1 oz of ethanol = 12 oz beer, 5 oz wine, or 1.5 oz distilled spirits.

Based on Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2017. [Epub ahead of print]. doi:10.1016/j.jacc.2017.11.006[20]; National High Blood Pressure Education Program. *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: Complete Report.* Bethesda, MD: National Heart, Lung, and Blood Institute; August 2004.[24]

In addition to lifestyle modifications, several categories of drugs can be prescribed to treat hypertension. Low-dose thiazide diuretics are an effective first-line choice; they have been demonstrated to reduce all-cause mortality, stroke, and cardiovascular disease.[26] Angiotensin-converting enzyme (ACE) inhibitors, calcium-channel blockers (CCBs), beta blockers, angiotensin II receptor blockers (ARB), and other drug categories are used either alone or in combination; ACE inhibitors have shown evidence of similar efficacy to thiazide diuretics, but are not superior and are more costly.[26] A meta-analysis of trials of antihypertensive drugs concluded that all classes of these medications had similar effectiveness in preventing coronary heart disease.[27] The choice of agent recommended for use may vary depending on the presence or absence of other risk factors or comorbidities. An algorithm for managing hypertension can be found in the JNC 7 and JNC 8 guidelines.[23,24]

Women who need pharmaceutical therapies are referred to a physician to initiate treatment. However, many midwives will provide care for women being treated or at risk for hypertension. Based on the practice setting, a midwife may consult with a specialist, refer the woman at the time of diagnosis to a specialist, or initiate an initial antihypertensive agent. If the initial pharmaceutical agent proves inadequate, as evidenced by the woman not meeting goal blood pressure in the algorithm, consultation and/or referral is warranted.

Dyslipidemia

Dyslipidemia—that is, elevated fat or cholesterol in the blood—is another major risk factor associated with cardiac disease, and often is preventable or modifiable. Low-density lipoprotein (LDL) has been used as a surrogate biomarker to assess for risk for cardiac

disease and is the major component of cholesterol, accounting for approximately 65% to 70% of total cholesterol. An elevation in LDL level is directly associated with the development of atherosclerotic plaques.

Specific risk factors for dyslipidemia include obesity, hypertension, diabetes, smoking, preexisting coronary heart disease, and a family history of early cardiovascular disease. Risk increases with age: For women, the clinically relevant increased risk is related to menopause, and age 55 years and older often is used as the point at which risk begins to spike upward. Conditions that increase the risk of dyslipidemia include diabetes; thyroid disease; kidney diseases including chronic renal failure and nephrotic syndrome; obstructive liver disease such as gallstones, hepatitis, and cirrhosis; and use of medications such as protease inhibitors, progestins, corticosteroids, and anabolic steroids. Data suggest that among women age 20 to 45 years, the majority have coronary heart disease, a coronary heart disease equivalent such as stroke, diabetes, or elevated fasting glucose level, or one or more coronary heart disease risk factors, although most do not receive screening.[28]

Screening for dyslipidemia in women of reproductive age is not standardized in the same manner as screening for hypertension. The American Academy of Family Physicians[29] and the American College of Obstetricians and Gynecologists (ACOG)[30] recommend screening low-risk women for dyslipidemia beginning at age 40 and 45 years, respectively, while the U.S. Preventive Services Task Force has no active recommendations governing whether or when to screen for dyslipidemia in adults. According to ACOG, earlier screening is warranted in women with risk factors such as recurrent preeclampsia and preterm birth before 37 weeks' gestation accompanied by preeclampsia.[30] Neither of the other authorities discusses high-risk factors that might warrant earlier screening, although in clinical practice screening is often performed if a woman has hypertension, obesity, diabetes, or other risk factors.[28]

Screening for dyslipidemia includes first conducting a global risk assessment of risk factors. For example, women with type 2 diabetes, type 1 diabetes for more than 15 years, two risk factors for cardiovascular disease, or metabolic syndrome should be considered to have a high or very high risk for atherosclerotic cardiovascular disease. The second step in screening is to obtain a complete lipid panel after an overnight fast. The panel will include blood values of LDL cholesterol, total cholesterol, and high-density lipoprotein (HDL) cholesterol. The third step is to calculate the 10-year risk for having a coronary event. Clinicians are advised to use an online calculator to predict 10-year risk for a first atherosclerotic cardiovascular disease (ASCVD) event.[31] This calculation incorporates medical history risks and the results of the lipid panel, then predicts 10-year risk of ASCVD and compares the individual's 10-year and lifetime risks to a hypothetical individual with optimal characteristics. Links to online calculators are included in the Resources section at the end of this chapter.

When high cholesterol levels are found, counseling includes making diet changes to decrease total and dietary fat to between 25% and 35% of total calories, adding dietary fiber, stopping smoking, and increasing exercise levels. Medication therapy can be deferred while a trial of healthy lifestyle changes is undertaken, as long as there is no family history suggestive of genetic predisposition to coronary artery disease and the LDL cholesterol level is less than 160 mg/dL for women without other cardiac risk factors. Determining which woman needs

intervention and how her progress should be followed may be an indication for consultation or referral to a specialist. Pharmacologic therapy is instituted based on LDL level and risk profile.

The most widely accepted guidelines for the management of dyslipidemia are those established by the American College of Cardiology and the American Heart Association.[32] The most recent guidelines, released in 2013, focus on an algorithm for appropriate statin use. Liberal initiation of statins is recommended as the primary mode of treatment for individuals with clinical indications of atherosclerotic cardiovascular disease, including LDL cholesterol levels of 190 mg/dL or higher or conditions such as diabetes. The guidelines recommend the use of other products as adjunctive therapy in rare cases such as statin resistance cases or in individuals unable to tolerate statins.[32] According to the American College of Cardiology/American Heart Association, the strongest evidence for the use of statins are for the following indications[32]:

- Primary prevention of atherosclerotic cardiovascular disease in individuals with LDL cholesterol ≥ 190 mg/dL
- Primary prevention of atherosclerotic cardiovascular disease in individuals with diabetes age 40–75 years with LDL cholesterol levels of 70–189 mg/dL
- Primary prevention of atherosclerotic cardiovascular disease in individuals without diabetes with an estimated 10-year ASCVD risk ≥ 7.5%, with LDL cholesterol levels of 70–189 mg/dL, and age 40–75 years
- Secondary prevention of additional morbidity and mortality in individuals with clinical atherosclerotic cardiovascular disease

Statins are categorized by how well they lower LDL cholesterol on average. **Table 6-7** categorizes the common statins as either high, moderate, or low intensity to be consistent with the terminology used in the current guidelines.[32]

Table 6-7 High-, Moderate-, and Low-Intensity Statin Therapies[a]

High Intensity (Daily Dosage Lowers LDL-C by Average of ≥ 50%)	Moderate Intensity (Daily Dosage Lowers LDL-C by Average of 30–50%)	Low Intensity (Daily Dosage Lowers LDL-C by Average of < 30%)
Statins and Doses Approved by the FDA and Tested in RCTs		
Atorvastatin (Lipitor), 40 mg[b] to 80 mg	Atorvastatin (Lipitor), 10 (20) mg	Lovastatin (Mevacor), 20 mg
Rosuvastatin (Crestor), 20 (40) mg	Rosuvastatin (Crestor), (5) 10 mg	Pravastatin (Pravachol), 10–20 mg
	Simvastatin (Zocor), 20–40 mg[c]	
	Pravastatin (Pravachol), 40 (80) mg	
	Lovastatin (Mevacor), 40 mg	
	Fluvastatin (Lescol), 40 mg 2 times daily	
Statins and Doses Approved by the FDA But Not Tested in RCTs		

	Fluvastatin XL (Lescol XL), 80 mg	Simvastatin (Zocor), 10 mg
	Pitavastatin (Livalo), 2–4 mg	Fluvastatin (Lescol), 20–40 mg
		Pitavastatin (Livalo), 1 mg

Abbreviations: FDA, U.S. Food and Drug Administration; LDL-C, low-density lipoprotein cholesterol; RCT, randomized controlled trial.

[a] Individual responses to statin therapy varied in the RCTs and should be expected to vary in clinical practice. There might be a biological basis for less than average response.

[b] Evidence from one RCT only. Down-titration was initiated if the participant was unable to tolerate atorvastatin 80 mg in the IDEAL (Incremental Decrease Through Aggressive Lipid Lowering) study.

[c] Although simvastatin 80 mg was evaluated in RCTs, initiation of simvastatin 80 mg or titration to 80 mg is not recommended by the FDA because of the increased risk of myopathy, including rhabdomyolysis.

Modified with permission from Stone NJ, Robinson JG, Lichtenstein AH, et al. ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014;63:2889-2934.[32]

Individuals who will benefit from clinical use of statin therapy, as well as the types of therapy, are found in **Figure 6-1**. Some midwives may consult or refer a woman with dyslipidemia to a specialist upon discovery of the condition, whereas others may initiate statin therapy but will consult or refer if the response is inadequate as measured by serum cholesterol levels.

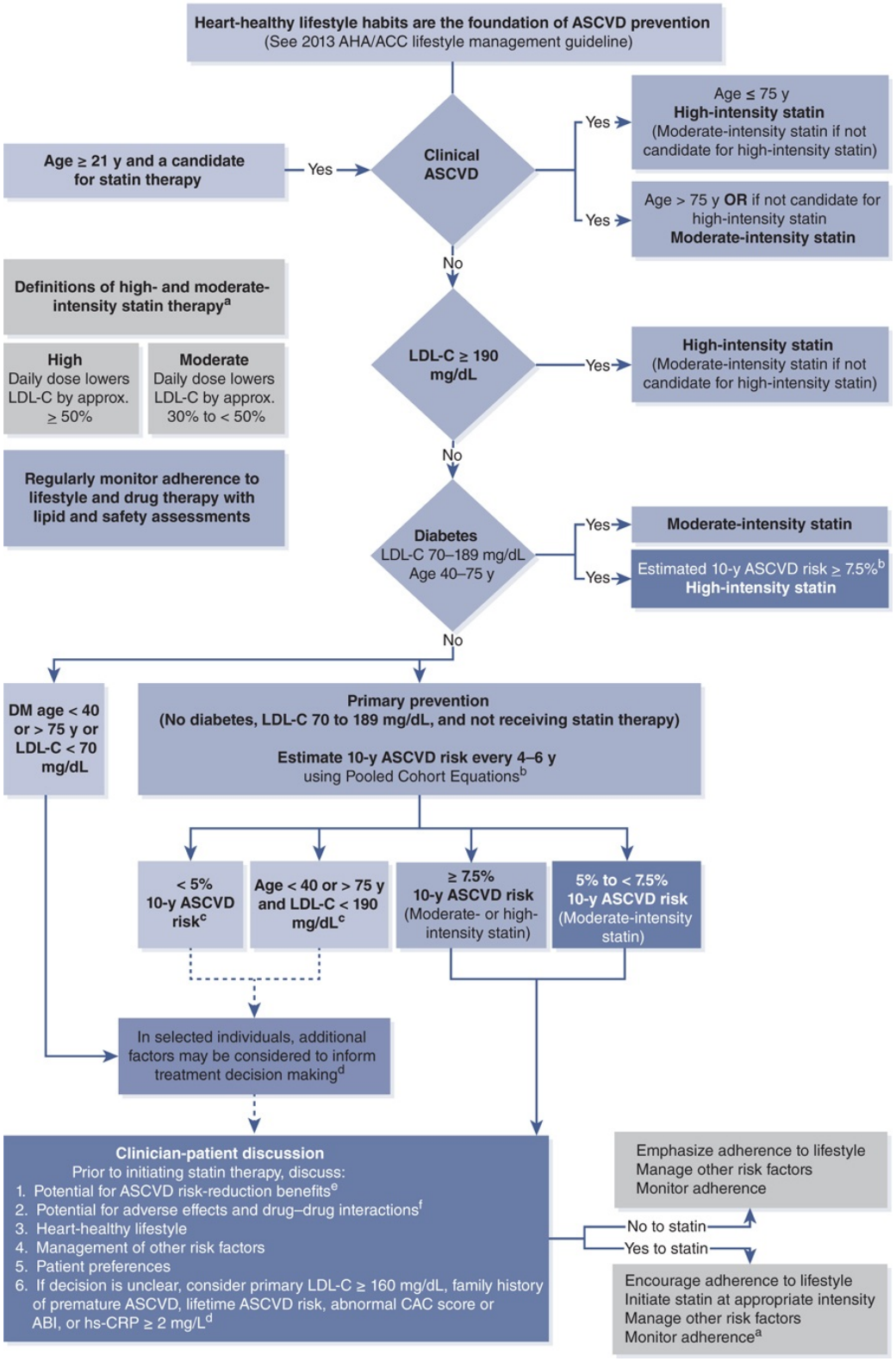


Figure 6-1 American College of Cardiology/American Heart Association blood cholesterol guideline.

Abbreviations: ABI, ankle-brachial index; ASCVD, atherosclerotic cardiovascular disease; CAC, coronary artery calcium; DM, diabetes mellitus; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; RCT, randomized controlled trial.

[a] Percent reduction in LDL-C can be used as an indication of response and adherence to therapy, but is not in itself a treatment goal.

[b] The Pooled Cohort Equations can be used to estimate 10-year ASCVD risk in individuals with and without diabetes. The estimator within this application should be used to inform decision making for primary prevention in patients not on a statin.

[c] Consider a moderate-intensity statin as more appropriate therapy in low-risk individuals.

[d] For those individuals in whom a risk assessment is uncertain, consider factors such as primary LDL-C ≥ 160 mg/dL or other evidence of genetic hyperlipidemias; family history of premature ASCVD with onset before 55 years of age in a first-degree male relative or before 65 years of age in a first-degree female relative; hs-CRP ≥> 2 mg/L; CAC score ≥ 300 Agatston units or ≥ 75th percentile for age, sex, and ethnicity; ABI < 0.9; or lifetime risk of ASCVD. Additional factors that may aid in individual risk assessment may be identified in the future.

[e] Potential ASCVD risk-reduction benefits. The absolute reduction in ASCVD events from moderate- or high-intensity statin therapy can be approximated by multiplying the estimated 10-year ASCVD risk by the anticipated relative-risk reduction from the intensity of statin initiated (approximately 30% for a moderate-intensity statin or approximately 45% for a high-intensity statin). The net ASCVD risk-reduction benefit is estimated from the number of potential ASCVD events prevented with a statin, compared to the number of potential excess adverse effects.

[f] Potential adverse effects. The excess risk of diabetes is the main consideration in approximately 0.1 excess case per 100 individuals treated with a moderate-intensity statin for 1 year and approximately 0.3 excess case per 100 individuals treated with a high-intensity statin for 1 year. In RCTs, both statin-treated and placebo-treated participants experienced the same rate of muscle symptoms. The actual rate of statin-related muscle symptoms in the clinical population is unclear. Muscle symptoms attributed to statin therapy should be evaluated.

Reproduced with permission from Stone NJ, Robinson JG, Lichtenstein AH, et al. ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014;63:2889-2934.[31]

Respiratory Conditions

Upper Respiratory Infections

Upper respiratory infections (URIs) include the common cold, rhinosinusitis, influenza, and pharyngitis. Commonly viral in nature, nonspecific URIs and influenza are a frequent reason for calls to healthcare providers, with individuals often asking for antibiotics that will not cure the problem. Although rates of inappropriate prescribing have decreased, rates of overtreatment remain high.[33] Both overtreatment and inappropriate treatment have led to a rapid increase in the prevalence of infections with drug-resistant *Streptococcus pneumoniae* and *Staphylococcus aureus*, which are the two most common bacterial respiratory pathogens. *S. pneumoniae* is increasingly resistant to penicillin, amoxicillin/clavulanate, and ceftriaxone, while more than 50% of *S. aureus* isolates have been found to be resistant to methicillin in antibiotic surveillance systems.[34]

Many URIs can be avoided through the use of simple hygiene techniques, most particularly the practice of hand washing. The use of antibacterial soaps and environmental sprays usually is not necessary in the home, but has not been linked to adverse ecological events. Hand sanitizers have become useful items in public areas, especially when respiratory infections are prevalent.

The diagnosis of nonspecific URI is made based on the presence of nasal congestion and a clear, white, or yellow/green discharge, as well as sore throat, muscle aches, headache, and cough. The symptoms of colds and influenza overlap, although high fever, profound malaise, and dry cough are more typical of the “flu” than of a cold. Symptomatic management may reduce the severity of symptoms, although many interventions commonly implemented lack evidence of effectiveness. Treatments include rest, increased fluids, saline gargle or spray, and a variety of medications. Pharmaceuticals are best targeted to specific symptoms, such as dextromethorphan (Delsym) for coughs; therefore, combination drugs are not advised. Antibiotics are not indicated for treatment of individuals with nonspecific URI or influenza.[35]

Ipratropium bromide (Atrovent), an anticholinergic nasal spray, can be used if needed to relieve rhinorrhea, sneezing, and congestion. Initial use consists of two sprays in each nostril, three to four times daily.[36] Antihistamines may provide limited relief in the first few days after the onset of a URI.[37] Intranasal corticosteroids have not shown any benefit for URIs, but are not harmful and may be continued if being used for another condition.[38] Regardless of pharmaceutical treatments, resolution of symptoms should be complete within a week.

A bacterial cause for URI should be suspected when the symptoms persist or worsen after 8 to 10 days; when maxillofacial pain is present (especially if only one side of the face is affected); when the symptoms over the first 3 to 4 days are severe; when symptoms are associated with a fever higher than 39°C (102.2°F); when purulent discharge is present; or when symptoms worsen after a viral infection had appeared to be resolving. If antibiotic therapy is needed, the narrowest-spectrum drug effective against *S. pneumoniae* and *Haemophilus influenzae* should be used.

Common Cold

The common cold is a self-limited syndrome that can be caused by many different viruses. This type of mild URI is the most frequent illness experienced by adults and children in industrialized nations. Symptoms include varying patterns of nasal discharge, headache, sore throat, sore muscles, cough, or sneezing. The common cold is usually spread via droplets and hard-surface contamination. The incubation period is 24 to 72 hours, and the period of infectivity starts approximately on the second day of illness and peaks on the third day of illness. Nevertheless, viruses that cause the common cold can be infective for several days. The initial differential diagnosis is allergic or bacterial rhinitis, bacterial pharyngitis, pertussis, and influenza. Complications associated with the common cold include rhinosinusitis, lower respiratory tract infections, exacerbation of asthma, and bronchitis.

Rhinosinusitis

When an acute URI has spread into the sinus cavities, the term *rhinosinusitis* usually is preferred to the older term *sinusitis*. A green or yellow nasal discharge commonly is found in addition to pain and pressure over the affected sinus. Other symptoms may include a toothache near the affected sinus, fever, and a cough that worsens when lying down. More than 95% of these infections are viral.[39] Acute rhinosinusitis lasts less than 4 weeks, and most such infections resolve spontaneously.[39] Although fewer cases of rhinosinusitis are bacterial in origin, overuse of antibiotics is common: One study found that these medications were prescribed in more than 80% of rhinosinusitis episodes.[39] Three clinical scenarios suggest a bacterial infection: (1) persistent symptoms for more than 10 days without improvement; (2) severe onset with a high fever of at least 39°C (102.2°F) and purulent nasal discharge for 3 or more days beginning with the onset of symptoms; or (3) “double-sickening,” in which viral symptoms begin to improve but then suddenly worsen near the end of the first week.[35]

Therapy for rhinosinusitis includes supportive management with over-the-counter (OTC) decongestants, analgesics, and antipyretics. As with the common cold, antihistamines should not be used unless the congestion has an allergic component. When symptoms of a bacterial infection or super-infection are present, then antibiotics should be initiated. Ampicillin–clavulanate (Augmentin) or doxycycline (Vibramycin) is the preferred initial regimen. Fluoroquinolones can be used as an alternative; however, azithromycin (Zithromax), trimethoprim–sulfamethoxazole (TMP-SMX; Bactrim), and cephalosporins are not recommended due to the increased risk of resistance to these drugs.[35] When symptoms persist for more than 12 weeks, referral to a specialist is warranted for evaluation of possible chronic rhinosinusitis or another underlying condition.[39]

Influenza

The onset of viral influenza is abrupt, marked by fever, rhinitis, cough, sore throat, headache, muscle pain, and general malaise. Most symptoms will resolve in a week or less, although malaise and tiredness may persist. Influenza should be suspected as the cause of URI during the flu season, which typically begins in October and lasts through April. Herd immunity achieved by widespread vaccination is the best protection against influenza outbreaks.

Vaccines for the seasonal flu types anticipated by the Centers for Disease Control and Prevention (CDC) become available in early fall and are recommended for all persons. Recommendations for vaccine formulations may change yearly depending on the prevalence and severity of the viruses in circulation and the availability and effectiveness of current vaccines. For example, the live attenuated influenza vaccine (LAIV), administered intranasally, was not recommended for use in the 2016–2017 flu season; the Advisory Committee on Immunization Practices has issued an interim recommendation that this vaccine not be used because it has been shown to have low effectiveness over the last two influenza seasons.[40] Detailed recommendations for flu vaccines are presented in the *Health Promotion Across the Lifespan* chapter. Individuals at greater risk of severe complications of influenza are listed in **Table 6-8**.[41]

Table 6-8	**Individuals at Increased Risk of Complications of Influenza**
Persons age ≥ 50 years	
Persons with chronic disease:	
Pulmonary (including asthma)	
Cardiovascular (except hypertension)	
Renal	
Hepatic	
Neurologic	
Hematologic	
Metabolic disorders (including diabetes mellitus)	
Persons who are immunosuppressed (caused by medications or HIV)	
Women who are or may be pregnant during the influenza season	
Children 6 months to 18 years receiving long-term aspirin therapy who might be at risk for Reye's syndrome after influenza virus infection	
Residents of nursing homes and other chronic care facilities	
American Indians/Alaska natives	
Persons with BMI > 40 kg/m^2	
Healthcare personnel	
Household contacts and caregivers of the following:	
Children age < 5 years	
Adults age ≥ 50 years	
Children age < 6 months	
Persons with conditions that increase risk for severe complications of influenza	

Abbreviations: BMI, body mass index; HIV, human immunodeficiency virus.

Reproduced with permission from Centers for Disease Control and Prevention. People at high risk of developing flu-related complications. 2016. Available at: https://www.cdc.gov/flu/about/disease/high_risk.htm. Accessed March 13, 2017.[41]

The CDC recommends the use of the neuraminidase inhibitors oseltamivir (Tamiflu) or zanamivir (Relenza) to treat influenza.[42] Treatment of infections suspected of being influenza within 48 hours of the onset of symptoms may shorten the duration of infection by

approximately one day. Without pharmacologic treatment, in healthy adults, influenza is a self-limiting disease and may be best treated with rest and supportive therapy.

Pharyngitis

Approximately 5% to 15% of cases of sore throat are caused by group A beta-hemolytic *Streptococcus* (GAS); the remainder are primarily viral in origin and require only supportive care while symptoms resolve. Testing for GAS is not necessary when symptoms—cough, runny nose, oral sores, and hoarseness—clearly indicate a viral infection. In contrast, tonsillar exudate, tender anterior cervical lymph nodes, fever, and lack of a cough suggest a bacterial infection. Either a rapid antigen detection test or culture can be performed if symptoms do not exclude GAS. Follow-up culture after a negative rapid antigen detection test is no longer recommended in adults.[43]

Penicillin and amoxicillin regimens lasting 10 days are the most appropriate first-line treatments for GAS. First-generation cephalosporins, clindamycin (Cleocin), clarithromycin (Biaxin), or azithromycin (Zithromax), are all acceptable alternatives when penicillin allergy exists. An analgesic or antipyretic can be used for supportive therapy; corticosteroids are not recommended. It is not necessary to treat household contacts.[43]

Bronchitis

Infections of the lower respiratory tract limited to the trachea and bronchi are termed *bronchitis*; they can appear as an inflammatory response to an otherwise uncomplicated URI. In healthy women of reproductive age, acute bronchitis is typically a viral syndrome of low-grade fever, malaise, fatigue, sore throat, chest pain, and cough. The cough may be productive or nonproductive. The cough of bronchitis can be differentiated from the cough of the common cold when it persists more than 5 days. Smoking, exposure to irritating fumes in the environment, and gastric reflux that irritates the bronchi can also precipitate acute bronchitis. Long-term exposure to smoking or environmental pollutants can lead to chronic bronchitis. On auscultation, areas of the lung other than over the bronchi should not have abnormal findings; a chest X ray, if performed, should not show infiltrates. Worsening chest pain with shortness of breath or pain on inspiration suggests pneumonia.

In most cases, the infection and the cough will resolve within 1 to 2 weeks with supportive therapy. If cough is persistent after the primary infection has resolved and wheezing is noted, the use of an albuterol inhaler may provide relief.[44] If an inhaler is prescribed, the directions should be for two puffs every 4 to 6 hours as needed to relieve symptoms. Asthma should be considered as an alternative diagnosis.

Antibiotics are not useful in the case of viral bronchitis, but under certain circumstances suspected bacterial infections of the bronchi may require antibiotic therapy. Antibiotics should be avoided in healthy adults with moderate symptoms; their potential benefit is outweighed in most cases by the potential for increased resistance and the expense of therapy.[45,46]

If the diagnosis is unclear, or if symptoms persist and worsen, both pneumonia and pertussis should be ruled out. Signs and symptoms that indicate the diagnosis is pneumonia include

temperature greater than or equal to 38°C (100.4°F), tachypnea at more than 24 breaths per minute, tachycardia at more than 100 beats per minute, rales or decreased breath sounds. and symptoms that develop quickly.[47] Women with symptoms suggestive of pneumonia warrant referral to a specialist for evaluation.

Community-Acquired Pneumonia

Together, influenza and pneumonia are the leading infectious cause of death in the United States and the eighth most frequent cause of death overall.[17] Predisposing factors for pneumonia include damage to the cilia of the respiratory tract from chronic cough, viral infections, or smoking. The common infecting organisms are *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Staphylococcus aureus*, and a variety of viruses, most commonly influenza. Because bacterial causes of community-acquired pneumonia predominate, antibiotic therapy should be instituted promptly when a diagnosis is made. Risk factors for community-acquired pneumonia include age older than 65 years, underweight or BMI less than 19 mm/kg^2, smoking, excessive alcohol intake, decreased ciliary activity, prior episodes of pneumonia or chronic bronchitis, chronic obstructive pulmonary disease, use of antihistamines, immunosuppression, and chronic illnesses.[48,49]

The onset of symptoms with community-acquired pneumonia is usually abrupt, with fatigue, cough, difficulty breathing, chills/sweats, anorexia, headache, and chest pain being the most common. Fever of 38°C (100.4°F) or higher, tachycardia, tachypnea, rales, and lung consolidation can be found on examination. When the cause is bacterial, high fever and a productive cough are more likely, whereas viral causes produce a more generalized malaise. Chest X ray is indicated to confirm the diagnosis and to identify underlying complications. Microbiologic testing to confirm the pathogen is not routinely recommended before empiric therapy is initiated,[48] but at least one expert has recently argued that this practice runs counter to the need for better targeting of antibiotic therapy to reduce risk of resistance in common pathogens.[50,51]

Outpatient versus inpatient treatment of women with community-acquired pneumonia is a decision with significant consequences in terms of treatment modalities, testing schemes, and costs. While uncomplicated pneumonia should be treated promptly with antibiotics and can safely be managed on an outpatient basis in healthy adults, consultation with and likely referral to a specialist are necessary for women with suspected pneumonia. Recommendations for pneumococcal vaccines can be found in the *Health Promotion Across the Lifespan* chapter.

Pertussis

Bordetella pertussis is an increasingly important respiratory pathogen; 20,752 cases of this infection were diagnosed in the United States in 2015 after a peak of 32,971 cases the previous year, and the CDC suggests that many cases go unreported.[52] Pertussis affects the ability to clear the respiratory tract by paralyzing the cilia lining the respiratory tract and causing inflammation as well as by directly invading the alveoli.[53] The incubation period is approximately 7 to 10 days, but symptoms can first occur as distant in time as 6 weeks

following exposure. Clinically, the first symptoms resemble a mild URI, with little temperature elevation. Although pertussis is milder in adults than in infants or young children, it can persist with an increasingly violent cough over several weeks. **Table 6-9** provides a description of the stages of the disease.[53]

Table 6-9 Stages of Pertussis (Whooping Cough)

Stage	Duration	Symptoms
Catarrhal	1–2 weeks	Cough, runny nose, low-grade fever, sneezing
Paroxysmal	1–6 weeks	Episodes of rapid coughing 15 or more times per day, ending with a long inspiratory gasp and high-pitched "whooping" sound, occurring more often at night, accompanied by possible vomiting, exhaustion
Recovery	Weeks to months	Slowly resolving symptoms: cough disappears within 2–3 weeks, may return with subsequent respiratory illnesses

Based on Hamborsky J, Kroger A, Wolfe S. Pertussis. In: Hamborsky J, Kroger A, Wolfe S, eds. *Epidemiology and Prevention of Vaccine-Preventable Diseases.* 13th ed. Washington, DC: Public Health Foundation; 2015. Available at: https://www.cdc.gov/vaccines/pubs/pinkbook/pert.html. Accessed May 13, 2017.[53]

Common complications of untreated pertussis in adults include rib fracture, pneumothorax, superimposed bacterial pneumonia, and urinary incontinence. Other risks that are less common include dehydration, epistaxis, hernia, and—rarely—encephalopathy, pneumothorax, rectal prolapse, subdural hematoma, and seizure.[53]

In addition to vaccinating infants and children, vaccination against pertussis with the tetanus/diphtheria/acellular pertussis (Tdap) combination vaccine among adults is an effective measure to control the spread of this disease. The action of vaccinating others around the child often is called "cocooning," and is accomplished by administering the Tdap vaccine to all adults who have close interaction with newborns. Recommendations regarding the pertussis vaccine for women of all ages are described in detail in the *Health Promotion Across the Lifespan* chapter.

Therapy for pertussis includes a macrolide antibiotic; the original recommendation for erythromycin was expanded to include azithromycin (Zithromax) and clarithromycin (Biaxin) as these macrolides are better tolerated. TMP-SMX (Bactrim) can also be used for therapy. Post-exposure prophylaxis with the same drugs is recommended for all close contacts of a pertussis case, for individuals at high risk of severe disease (newborns, women in the third trimester, and individuals with preexisting conditions such as moderate to severe asthma or immunosuppression that could be exacerbated by infection), and for persons who have close contact with those at risk of severe disease.[54] It is not known whether post-exposure vaccination has any benefit.[53]

Asthma

Asthma is a chronic inflammation of the airways associated with intermittent worsening of

symptoms; reversible obstruction from bronchospasm, edema, and mucus production; and hyper-responsiveness to stimuli. An individual's innate immunity, genetics, and environmental exposures all play a role in the development of this chronic disease. The prevalence of asthma in the United States is increasing, with this condition affecting almost 10% of all women.[55] Among the approximately 19 million women with asthma, those living in poverty have the highest risk.[55]

Signs and symptoms suggestive of asthma are listed in **Table 6-10**.[56] On examination, wheezing when breathing normally, prolonged expiratory phase, use of accessory muscles, increased nasal swelling, secretions or polyps, and evidence of atopy all increase the likelihood of an asthma diagnosis. Pulmonary function testing is required for diagnosis. The differential diagnosis in adults includes chronic obstructive pulmonary disease, vocal cord dysfunction, congestive heart failure, medication-related cough, pulmonary embolism, and gastroesophageal reflux disease, among others.[56] Midwives practicing in areas of high asthma frequency may keep flow meters in their offices either to assist in the presumptive diagnosis of asthma or to assess lung function when women with asthma present or are symptomatic. Four classes of asthma are distinguished based on severity of disease, as shown in **Table 6-11**.[57]

Table 6-10 Characteristic Signs and Symptoms of Asthma

The presence of one or more of these symptoms is an indication for evaluation:
Wheezing (not required for diagnosis)
History of:
Persistent cough (particularly at night)
Recurrent wheezing
Recurrent difficulty in breathing
Recurrent chest tightness
Symptoms occur or are worsened by:
Exercise
Viral infection
Inhalant allergens such as dust mites, mold, pollen, animals with fur
Irritants to lung tissue such as smoke or airborne chemicals
Changes in weather
Strong emotional expression (laughing or crying hard)
Stress
Menstrual cycles
Symptoms occur or worsen at night, awakening the individual

Modified with permission from National Asthma Education and Prevention Program. *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma (EPR-3).* Bethesda, MD: National Heart, Lung, and Blood Institute; 2007.[56]

Table 6-11 Classification of Asthma

Disease Severity	Daytime Symptoms	Nighttime Symptoms	PEF or FEV	Interference with Normal Activity	Use of Short-Acting Medication	Asthma Exacerbation frequency[a]
Mild intermittent	≤ 2 days/week	≤ 2 nights/ month	> 80%	None	≤ 2 days/week	0–1/year
Persistent mild	> 2 days/week but not daily	> 2 nights/ month	> 80%	Minor	> 2 days/week but not daily and not more than 1 time/day	≥ 2/year
Persistent moderate	Daily	> 1 night/week but not nightly	> 60 and < 80%	Some	Daily	≥ 2/year
Persistent severe	Continual	Frequent	≤ 60%	Extremely limited	Several times/ day	≥ 2/year

Abbreviations: FEV, forced expiratory volume; PEF, peak expiratory flow.

[a] Data are insufficient to link frequency of exacerbations to different levels of severity. Generally, more frequent and intense exacerbations are an indication of increasing severity. Persons with ≥ 2 exacerbations per year may be considered to have persistent asthma even in the absence of impairment levels consistent with persistent asthma.

Modified with permission from National Heart, Lung, and Blood Institute. Asthma care quick reference. 2012. Available at: https://www.nhlbi.nih.gov/files/docs/guidelines/asthma_qrg.pdf. Accessed March 13, 2017.[57]

Important components of the management of asthma include patient education, management of environmental factors and comorbidities that worsen symptoms, and use of medication. The key to control of asthma and prevention of worsening of this condition is inclusion of the woman in decisions about her care, awareness of environmental triggers and the need to minimize exposure when possible, and knowledge of the role that other health problems play in asthma management.[56]

Medication management is based on a stepwise approach.[57] Intermittent symptoms of asthma (occurring fewer than 2 days per week, disturbing nighttime rest fewer than 2 days per month, not interfering with activities of daily living, and with one or fewer exacerbations per year) can be managed with short-acting beta-agonists such as albuterol sulfate (Proventil) on an as-needed basis. When a rescue inhaler is needed on more than 2 days per week, persistent asthma should be considered.

Anyone with persistent asthmatic symptoms needs to be on daily medication. Inadequate treatment limits physical activity, decreases pulmonary function, and increases the risk of recurrent attacks. The choice of medications is based on both the severity and the persistence of symptoms. The main classes of asthma drugs for long-term maintenance include inhaled corticosteroids, immunomodulators, leukotriene receptor antagonists, long-acting beta-agonists, 5-lipoxygenase inhibitors, mast cell mediators, methylxanthines, and combination drugs. The preferred agents for all patients with asthma, regardless of stage, are inhaled corticosteroids because they reduce inflammation and are theorized to minimize the risk of airway remodeling.

Women with newly diagnosed asthma should be evaluated in consultation with or by referral to a specialist experienced in respiratory care. Individuals with known mild intermittent, persistent mild, or persistent moderate asthma can be cared for by a midwife for primary care

and monitoring. However, consultation with an asthma specialist is warranted if a woman requires more than medium-dose inhaled corticosteroids to achieve control (Stage 4 or higher) or if she has unstable asthma.[57]

Tuberculosis

Although the rate of tuberculosis (TB) infection has been declining in the United States for the last 20 years, nearly 10,000 cases were reported in 2015.[58] Asians and Pacific Islander/Native Hawaiians are disproportionally affected by this infection.[58] Notably, tuberculosis among individuals born outside of the United States accounts for more than 66% of all diagnosed cases.[58] Poverty, lack of education, and positive status of human immunodeficiency virus (HIV) are associated with increased risk of acquiring the infection. Even though midwives may not provide first-line management for women with tuberculosis, populations for whom midwives often provide care may be at high risk for TB. Factors that confer a high risk for contracting TB infection are listed in **Table 6-12**.

Table 6-12 **Persons at Higher Risk for Exposure to and/or Infection with *M. tuberculosis***

Close contact with a person who has known or suspected active TB
Foreign-born persons from areas with high rates of TB (e.g., Africa, Asia, Eastern Europe, Latin America and Russia)
Persons who work or reside with people at high risk for TB (e.g., correctional facilities, long-term care facilities, homeless shelters)
Healthcare workers who serve individuals who are at increased risk for TB
Active substance use, especially intravenous drug use
Immune suppression: HIV/AIDS, diabetes mellitus, transplant recipients, chronic steroid treatment, renal disease, cancer of head, neck, and/or lung
Intestinal bypass or gastrectomy procedures

Abbreviations: HIV/AIDS, human immunodeficiency virus/acquired immunodeficiency syndrome; TB, tuberculosis.

Modified with permission from Centers for Disease Control and Prevention. *Core Curriculum on Tuberculosis: What the Clinician Should Know.* 6th ed. Atlanta, GA: Centers for Disease Control and Prevention; 2013. Available at: https://www.cdc.gov/tb/education/corecurr/pdf/corecurr_all.pdf. Accessed April 29, 2017.[59]

Transmission

The tuberculosis bacterium is spread by droplet formation or aerosolization, so casual contacts of individuals with active disease are at minimal risk of becoming infected. Conversely, close family members and those in close daily contact with the infected individual are most likely to have been exposed. Because *Mycobacterium tuberculosis* does not produce toxins, infection with this pathogen does not induce an immediate host response. Cellular immune response and a positive tuberculin skin test occur 2 to 12 weeks after acquisition of the bacterium. Symptoms may include generalized malaise and fatigue, night sweats, weight loss, fever, and a productive cough with or without hemoptysis. Pleuritic pain may develop, and on examination, crepitus and rales may be heard.[58]

Screening for Tuberculosis

The purpose of screening for tuberculosis is to detect individuals who have inactive disease as well as those who have recently been infected. The test most commonly used for this purpose is the intradermal tuberculin screening test, formerly known as PPD (purified protein derivative).[58] This test is read 48 to 72 hours after the tuberculin is injected in the subcuticular area of the forearm. Results are assessed by the degree of the diameter of a palpable swelling (induration), measured in millimeters, excluding erythema. The amount of induration required for a positive reading is affected by several factors, listed in **Table 6-13**.[59] A positive intradermal tuberculin screening test indicates the woman has disease that may or may not be currently active. Women with latent tuberculosis, who are not contagious and do not have TB disease, may have a positive TB screen.[59] The intradermal tuberculin screening test does not need to be repeated for anyone with a documented prior positive result.

Table 6-13 **Classification of the Tuberculin Skin Test Reaction**[a]

Induration ≥ 5 mm is considered positive in:	Induration ≥ 10 mm is considered positive in:	Induration ≥ 15 mm is considered positive in:
HIV-infected persons A recent contact of a person with infectious TB disease Persons with fibrotic changes on chest X ray Persons with organ transplants Persons who are immunosuppressed for other reasons	Persons recently in the United States (< 5 years) from high-prevalence countries (Asia, Africa, Eastern Europe, Latin America, Russia) Injection drug users Residents and employees of high-risk congregate settings Mycobacteriology laboratory personnel Persons with clinical conditions that place them at high risk (diabetes, silicosis, severe kidney disease, certain types of cancer, and some intestinal conditions) Children < 5 years of age Infants, children, and adolescents exposed to adults in high-risk categories	Any person with no known risk factors for TB

Abbreviations: HIV, human immunodeficiency virus; TB, tuberculosis.

[a] Targeted skin testing programs should be conducted only among high-risk groups.

Modified with permission from Centers for Disease Control and Prevention. *Core Curriculum on Tuberculosis: What the Clinician Should Know.* 6th ed. Atlanta, GA: Centers for Disease Control and Prevention; 2013. Available at: https://www.cdc.gov/tb/education/corecurr/pdf/corecurr_all.pdf. Accessed April 29, 2017.[59]

Alternatively, two blood tests that may be used to screen for TB in individuals who have received the bacillus Calmette-Guerin (BCG) vaccine or who are unable to return to have the intradermal tuberculin screening test results read, such as women screened in an emergency department. Many women born outside of the United States and some U.S.-born women may have received a BCG vaccine in childhood to prevent tuberculosis infection; this test may result in a permanent positive intradermal tuberculin screening test, necessitating other methods of screening.

When the intradermal tuberculin screening test or TB blood test is positive, the woman may

have active TB disease or latent tuberculosis. Women with latent tuberculosis are not contagious and do not have TB disease.[59] Chest X ray is used to rule out pulmonary tuberculosis for individuals with a positive intradermal tuberculin screening test or TB blood test, though sputum culture is required to confirm a diagnosis of active TB disease.[58,59]

Sputum cultures should be performed when there is clinical suspicion of active tuberculosis. A delay as long as 2 weeks may occur before results become available, but presumptive management should continue, even though the diagnosis is not final without the confirmation of cultures. A smear from the sample can be evaluated for the presence of mycobacteria by acid-fast or fluorescent stain. Even when the stain is negative, the sputum sample should be sent for culture.[58]

Any woman suspected of latent tuberculosis or active TB infection should be promptly referred to a specialist for ongoing care. In some settings, it may be appropriate for the midwife to order the chest X ray or other testing to expedite treatment while the woman is waiting for an appointment with a physician.

Treatment

To prevent progression to active TB disease, most women who have been diagnosed with latent tuberculosis should be treated. Several treatment regimens are considered acceptable for the treatment of latent TB that differ in the agent used and duration of treatment, which ranges from 3 to 9 months.[58,59] Active disease is treated with one of several multidrug combinations, which are administered over 6 to 9 months of uninterrupted therapy.[58,59]

Teaching includes the benefits and risks of long-term therapy, the importance of adherence, and side effects. At least monthly, the woman should be assessed for adherence to the medication regimen, signs and symptoms of active disease, and symptoms of hepatitis.[59] Even though other healthcare providers will be responsible for prescribing and monitoring tuberculosis treatment regimens, midwives need to be cognizant of appropriate management to promote completion of therapy and monitoring.

Gastrointestinal Disorders and Abdominal Pain

Stomach aches and pains, diarrhea and constipation, and bloating are all common symptoms reported by women.[60] As part of the history for any woman of childbearing age, last menstrual period and potential pregnancy risk are routinely assessed. Ensuring that women of childbearing age are not pregnant is an essential first step whenever abdominal symptoms are present because diagnosis and management can be influenced by this condition. This section focuses on general gastrointestinal health concerns common to all women. Complications that occur during pregnancy and produce abdominal symptoms are addressed in the *Pregnancy-Related Conditions* and *Medical Complications in Pregnancy* chapters. Similarly, gynecologic concerns are addressed in the *Menstrual Cycle Abnormalities* and *Gynecologic Disorders* chapters.

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) is the more severe and persistent form of gastroesophageal reflux, caused by involuntary relaxation of the sphincter muscle separating the esophagus and the stomach. This condition is responsible for more than 8 million office visits annually, and is the most common gastrointestinal diagnosis.[60] When gastric reflux occurs more than twice a week, GERD is diagnosed. GERD produces symptoms of heartburn that worsen with meals, bending over, and lying down. Other, less easily recognizable symptoms include an asthma-like wheeze, cough, laryngitis, and chest pain. Persistent severe disease may produce complications such as injury to the epithelium of the esophagus (Barrett's esophagus), stricture formation, and adenocarcinoma. Hiatal hernia—that is, separation of the diaphragm that allows the sphincter and upper portion of the stomach to penetrate into the chest cavity—is common in healthy older adults and can contribute to the symptoms of GERD. Obesity and smoking influence the development of GERD, as can pregnancy. *Helicobacter pylori* infection, the dominant cause of gastroduodenal ulcers, does not appear to have an effect on the occurrence of GERD.[61]

Therapy for GERD includes weight reduction, consumption of a low-fat diet, and avoidance of triggers such as caffeine, tobacco, and spicy or acidic foods. Eating small meals and remaining upright after meals may help prevent or ameliorate symptoms. If reflux occurs primarily at night, elevating the head while in bed will also help avoid discomfort. Certain medications, including beta-adrenergic agonists, tranquilizers, sedatives, progesterone, and calcium-channel blockers, may worsen symptoms. The use of antacids is likely to bring short-term relief of intermittent symptoms but may interfere with absorption of other medications. Step-up therapy for more severe symptoms begins with an 8-week course of a proton pump inhibitor, such as omeprazole (Prilosec), esomeprazole (Nexium), or lansoprazole (Prevacid). No specific proton pump inhibitor appears to be more effective than another. A proton pump inhibitor should be taken in the morning, before the first meal of the day. A second dose in the evening may be needed for nighttime symptoms. If only partial relief is achieved, adding a second dose or changing to a different proton pump inhibitor may be effective.[61] A woman whose symptoms are unresolved following an 8-week trial of a proton pump inhibitor should

be referred for consultation with a specialist to assess the need for endoscopy or possible fundoplication of the stomach.[61]

Ulcers

Peptic ulcers are open lesions of the stomach or duodenum, penetrating through the mucosa into muscle. An estimated 15 million people, representing almost 7% of non-institutionalized adults in the United States, have these lesions.[62] Common etiologies include *Helicobactor pylori* infection, a condition responsible for 70% of gastric ulcers and 95% of duodenal ulcers, and excessive use of NSAIDs, such as ibuprofen or aspirin.

Testing for *H. pylori* is indicated in adults with a peptic ulcer, history of peptic ulcers, or undiagnosed dyspepsia. The American College of Gastroenterology does not recommend testing unless treatment is planned. Noninvasive testing methods include antibody screens, urea breath testing, and fecal antigen testing.[63] Endoscopy is warranted in individuals older than 50 years and those with alarming symptoms such as bleeding, unexplained weight loss, change in bowel habits, or recurrent vomiting. Endoscopic testing offers excellent sensitivity and specificity, but is invasive and expensive.[61] Thus, for midwives, the decision to consult or refer for management of suspected *H. pylori* ulceration is made early in the evaluation process.

In addition to medication, counseling about avoiding use of aspirin and NSAIDs, stress reduction, and smoking cessation are all useful interventions for management of persons with ulcers. By themselves, these lifestyle interventions are not curative; instead, antibiotic therapy is necessary to eliminate *H. pylori* from the gastrointestinal tract and is essential for healing. Recommended initial therapies include a proton pump inhibitor and clarithromycin (Biaxin) plus either amoxicillin (Amoxil) or metronidazole (Flagyl) for 10 to 14 days. Common side effects of these regimens include headache, altered taste, diarrhea, and stomach upset, and women should be aware that they will have gastrointestinal symptoms during therapy as a means to decrease nonadherence.[63] The use of probiotics may offer some relief for side effects and assist in maintaining normal gastric flora.[64] Sucralfate (Carafate) is also often used to promote healing and reduce inflammation. Gastric ulcers that remain unresolved need to be evaluated to exclude cancerous lesions of the stomach.

Abdominal Pain

Abdominal pain has many causes, both gynecologic and non-gynecologic. **Table 6-14** lists some of these etiologies. The diversity of differential diagnoses for women with abdominal pain reinforces the idea that midwives confronted with acute abdominal pain should not hesitate to seek consultation when the diagnosis is unclear.

Table 6-14	Common Differential Diagnoses for Abdominal Pain[a]
Aortic dissection Appendicitis Bowel obstruction	

Cholecystitis
Constipation
Cystitis
Diarrhea
Diverticulitis
Ectopic pregnancy
Endometriosis
Gastroenteritis
Hernia
Inflammatory bowel disease
Lower lobe pneumonia
Mittelschmerz
Ovarian torsion
Ovarian cyst
Pancreatitis
Pelvic inflammatory disease
Pyelonephritis
Ulcer

[a] This list is not comprehensive.

When a woman presents with abdominal pain, assessment begins with a comprehensive history. The onset, description, duration, associated symptoms, and precipitating or relieving factors are determined, as well as identifying whether the pain is localized and, if so, to which quadrant.

Acute Abdomen

Acute abdomen refers to sudden severe abdominal pain and is a medical emergency. Characteristics that distinguish the acute abdomen include abdominal rigidity or distension, guarding, rebound pain, tachycardia, and decreased or absent bowel sounds. Fever may be present but is not essential to the diagnosis. Vomiting as well as urinary, bowel, or vaginal symptoms may be present. Immediate referral for prompt physician evaluation is needed.

Gallbladder Disease

Nearly 20% of the population may have asymptomatic gallstones; as many as 33% will become symptomatic in the future and require evaluation or treatment.[65] Cholecystitis is an inflammation of the gallbladder—the organ that collects, concentrates, and dispenses into the digestive tract the bile produced by the liver. When excess cholesterol triggers a crystallization process, gallstones are formed. If gallstones become symptomatic, they usually cause only mild biliary colic or a sharp pain after a fatty or large meal that may last a few hours and resolve. Choledocholithiasis is blockage of the common bile duct by gallstones.[65]

Risk factors for developing gallbladder disease include obesity followed by rapid weight loss, hypertriglyceridemia, female gender, pregnancy, older age, diabetes, and genetic

predisposition. When cholecystitis develops, sharp epigastric pain may last for several hours or days and is often associated with nausea and vomiting. Women with acute attacks present with severe, persistent right upper quadrant pain, often radiating to the right shoulder blade or the central back opposite the epigastrium. Murphy's sign (**Figure 6-2**) is positive if the woman stops inspiration with deep palpation of the right upper quadrant of the abdomen. Leukocytosis, elevated liver function tests, and elevated bilirubin are common laboratory findings. Ultrasound is the most effective diagnostic tool for identifying gallbladder disease.

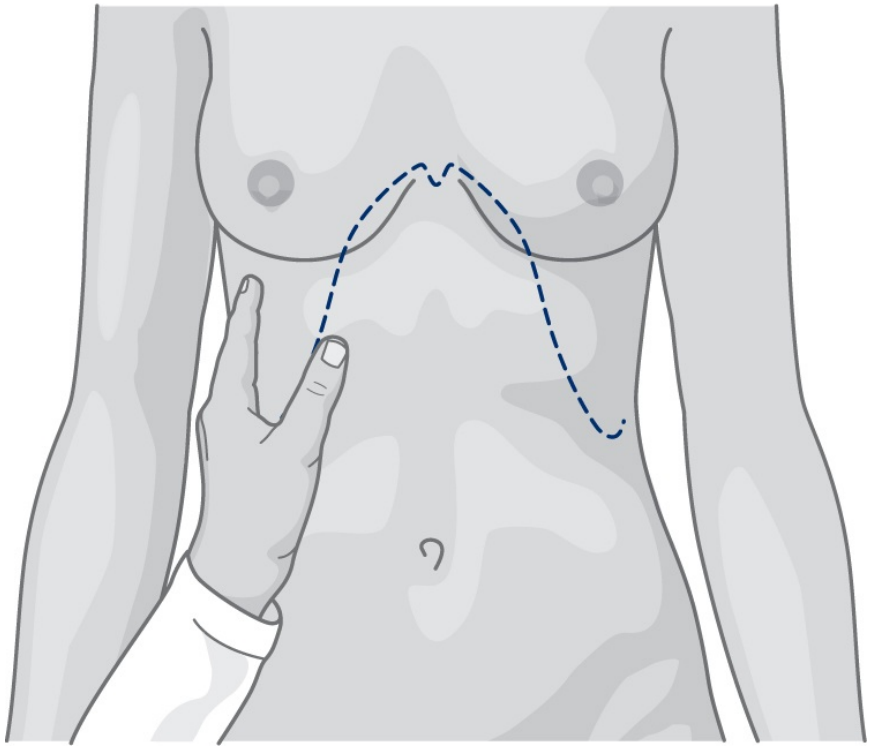


Figure 6-2 Murphy's sign.

Once a diagnosis of cholecystitis is considered, the midwife should refer the woman for physician care. Prompt surgical removal of the gallbladder is the usual treatment.[65] None of the nonsurgical approaches to treatment of gallstones has proved to be an effective alternative to surgery, but rarely nonsurgical options may be considered in individuals who are not good surgical candidates.[65]

Appendicitis

When the appendix becomes obstructed, enlarged, and unable to drain, the subsequent bacterial response leads to appendicitis. After decades of decreases in its incidence, the rate of acute appendicitis is increasing in the United States.[66] Although the most common age at which individuals develop appendicitis remains 10 to 20 years, this diagnosis is becoming more common among individuals older than 40 years.[66] One suggested source of the relative

increases in appendicitis incidence is improved diagnostic accuracy, particularly the accuracy of computed tomography, which decreases missed diagnoses attributed to other, usually gynecologic, pelvic symptoms.[66]

The classic descriptive symptoms for appendicitis are anorexia and generalized abdominal pain, resolving into acute right lower quadrant abdominal pain, vomiting, and fever. A complete blood count will show leukocytosis with an increased neutrophil ratio. Deep tenderness over McBurney's point in the lower right abdomen is an early sign of acute appendicitis. Rebound pain with release of pressure from the left lower abdomen is known as Rovsing's sign. **Figure 6-3** illustrates the locations of McBurney's point and Rovsing's sign.

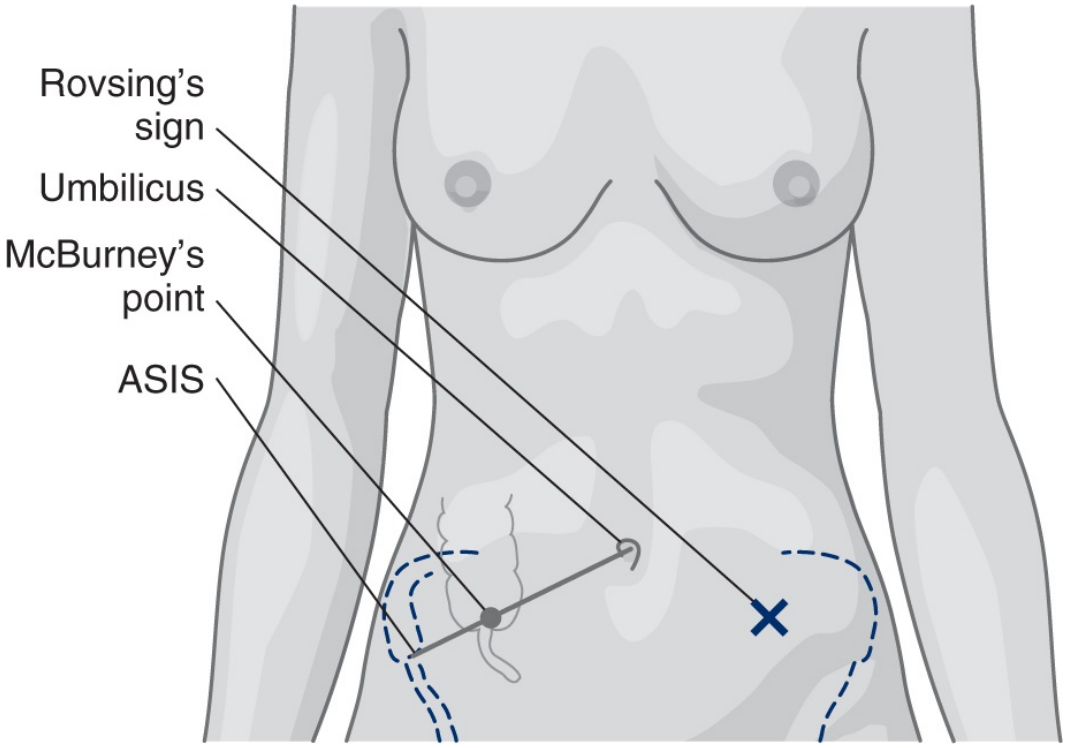


Figure 6-3 McBurney's point and Rovsing's sign. McBurney's point is on the abdominal wall that lies between the umbilicus and ASIS, and is usually two-thirds the distance from the umbilicus toward the ASIS. The most common placement is at the base of the appendix.

Abbreviation: ASIS, anterior superior iliac spine.

When appendicitis is suspected, the woman should be referred immediately for evaluation and possible surgery. Antibiotic therapy for nonsurgical management of appendicitis is being studied, but is not yet a standard practice.[67]

Gastroenteritis and Acute Diarrhea

Some women report diarrhea when they have any change in bowel habits that produces soft, frequent, or less-formed stools. True diarrhea is characterized by increased water content and may include mucus or bloody discharge. Women who report having diarrhea are asked about associated symptoms such as cramping, nausea or vomiting, or fever. Careful examination of

the abdomen, including auscultation of bowel sounds, palpation, and percussion, is necessary.

Acute diarrhea is predominantly infectious; chronic diarrhea, lasting for more than 2 consecutive weeks, can result from a variety of infections, medication use, chronic illness, malabsorption syndromes, stress, and irritable bowel syndrome. Recent travelers, the elderly, members of an identified community outbreak, and those recently prescribed antibiotics require closer attention to a specific diagnosis. Women with chronic watery diarrhea are referred for physician consultation for further testing.

Viral gastroenteritis is a generally self-limiting disease, in which oral exposure to a pathogenic virus such as Norovirus or rotavirus leads to explosive onset of nausea, vomiting, and/or diarrhea, fever, and malaise within 1 to 3 days of exposure. The abdomen is tender; no guarding is present. Bowel sounds are increased. Norovirus alone accounts for 58% of acute foodborne illnesses.[68] Other causes of diarrhea-linked infections include *Escherichia coli* species, *Salmonella*, and a host of other bacteria, most of which are foodborne pathogens.

The majority of cases of diarrhea in healthy adults will spontaneously regress within 1 to 4 days. Rest and oral fluids are the basic components of care. Medications such as diphenoxylate (Lomotil) or loperamide (Imodium) may be offered to decrease the frequency of stools. Intravenous rehydration with electrolytes is indicated for severe dehydration and for individuals at increased risk, such as the elderly and immunocompromised persons.

Investigation of acute diarrhea includes a stool sample to check for white blood cells or frank bleeding. Laboratory evaluation of electrolytes should be reserved for more severe or persistent cases when dehydration is suspected. If symptoms are not resolving or the woman becomes dehydrated, stool samples for culture or for ova and parasites (depending on the suspected pathogen) as well as a toxin assay for *Clostridium difficile* (with recent antimicrobial use) would be indicated.

Traveler's diarrhea (TD), as distinguished from Norovirus infections acquired in large travel groups such as cruises, is more likely to have a bacterial source—specifically, *E. coli* or another enterococcus. Prevention through hand washing and food selection may help reduce the risk of contracting TD, though in high-risk regions the risk can never be completely eliminated. High-risk regions include Asia, the Middle East, Africa, Mexico, and Central and South America.[69] When infection occurs, early rehydration is essential, and antimotility agents such as loperamide may provide symptom relief.[69] When antibiotic treatment does not resolve symptoms, parasitic pathogens should be considered based on the location of travel.

Constipation

Straining to produce hard stools, infrequent bowel movements (fewer than once in 3 days), and painful defecation are characteristic of constipation. Estimates of the frequency of constipation average almost 15% of the female U.S. population, with the elderly having a greater prevalence.[70] Initial questioning of women with these symptoms should include an assessment of their "usual" bowel function. Among healthy women, inadequate dietary fiber, decreased fluid intake, and iron therapy for anemia, whether prescribed or self-initiated, are common causes of constipation. Other medications, such as tricyclic antidepressants (e.g., amitriptyline [Elavil]), anticholinergics (e.g., ipratropium bromide [Atrovent]), and calcium-channel

blockers (e.g., diltiazem [Cardizem] and verapamil [Calan]), may slow peristalsis and increase stool transit time, leading to constipation. Misuse of laxatives leading to decreased natural stimulation of the bowel is also common, particularly among the elderly. Stress, anxiety, and depression may lead to changes in bowel habits, as may a history of sexual abuse. Other serious causes include hypothyroidism, cancer, strictures, and opioid use. A careful history and physical assessment of women with any chronic bowel changes is necessary to rule out serious underlying disease that would require a referral.

Management of chronic constipation in healthy women includes counseling about diet and exercise, as well as increased fluid intake. Avoidance of straining and recognizing the physical cues that indicate the need to defecate are included in teaching. Women should also be counseled to stop the overuse of laxatives, cathartics, and enemas. If any medication is necessary during early treatment, a bulk-forming OTC drug such as Metamucil should be used; however, adding fiber to the diet is a better strategy because a pattern of healthy eating will help maintain normal function. If symptom relief is not obtained with the preceding measures, a trial of laxatives such as docusate sodium (Colace) is in order before referring the woman to a physician.

Irritable Bowel Syndrome Versus Inflammatory Bowel Diseases

Irritable bowel syndrome (IBS) is a functional bowel disorder that consists of lower gastrointestinal tract symptoms of increased bloating, diarrhea, or constipation in the absence of any structural or biochemical cause. This condition is chronic in nature, it tends to wax and wane, and is one of the most common presentations in primary care practice. The current definition (Rome III) includes onset associated with changes in the frequency or composition of the stool and pain relief with defecation, lasting for more than 6 months.[71] To meet the diagnostic criteria, the symptoms need not be continuous. Intermittent symptoms totaling 12 weeks are considered significant.[71] Increased or decreased stool frequency, abnormal stool formation (either hard or watery), bloating, difficulty in the passage of stool (straining, urgency, or failure to completely empty the bowel), and mucus in the stool are further confirmation of the diagnosis. Because the symptoms are unstable, the terms “IBS with diarrhea” and “IBS with constipation” are commonly used.

The onset of IBS usually occurs during the young adult years. Because organic causes of bowel changes are more frequent with increasing age, a new diagnosis should be made with care in adults older than 40 years. Women are more often affected than men and are more likely to have alternating symptoms of diarrhea and constipation. Assessment includes evaluation for bowel obstruction, including malignancies, and for gastrointestinal bleeding. Irritable bowel syndrome should not be confused with inflammatory bowel diseases such as Crohn’s disease and ulcerative colitis, which are chronic, recurrent inflammations of the bowel. Symptom relief after having a bowel movement is strongly suggestive of IBS.

Although the symptoms of IBS are common among adults who do not seek care, those who do are more likely to be distressed by the condition. Thus, an important step in managing care for individuals with this disorder is building a trusting relationship. Education about IBS, reassurance about the course and management possibilities, and dietary modification are

important in helping women manage their symptoms effectively.[72] Some individuals can identify foods that trigger symptoms, but not enough evidence exists to recommend specific dietary changes.[72] Probiotics may relieve bloating and flatulence for some individuals. The American Gastroenterological Association has made conditional recommendations supporting the use of antispasmodic medications, tricyclic antidepressants, and various medications for symptomatic relief of constipation or diarrhea.[72]

Because of the strong likelihood of underlying psychological distress among women with IBS, a careful assessment for anxiety disorders and depression is warranted. Considerations in managing this aspect of the disease include the use of antidepressants, psychotherapy, and supportive behavioral therapy.

In contrast, inflammatory bowel diseases are autoimmune disorders that affect the small and large intestine. Examples include Crohn's disease and ulcerative colitis. Inflammatory bowel diseases often have a genetic component. Inflammatory bowel diseases have symptoms that overlap with those of irritable bowel disorder but some dissimilarity. For example, both disorders are associated with intestinal symptoms but irritable bowel disorder is more likely to be triggered by dietary factors whereas inflammatory bowel disease is more unpredictable. Persons with inflammatory bowel diseases need physician care and often require medications that can be complex to manage.

Hepatitis

Hepatitis is the generic term used to describe inflammation of the liver, regardless of etiology. The most common causes of hepatitis include the infections caused by pathogenic viruses identified by the letters A through E and linked to liver disease by the acute and chronic nature of the conditions. While hepatitis A and E are spread by the fecal–oral route, hepatitis B, C, and D are spread by contact with blood and body fluids and can be contracted sexually. Hepatitis A and B are the most common forms of hepatitis in the United States. Although there are fewer new cases per year of hepatitis C than hepatitis A and B, infection with the C variant becomes chronic in approximately 75% to 85% of persons who are infected with it. In contrast, hepatitis B develops as a chronic state in fewer than 6% of adult cases.[73] Together, the A, B, and C strains are responsible for most new viral hepatitis diagnoses in the United States.[73]

Hepatitis D occurs as a coinfection with hepatitis B or as a secondary infection, and requires the presence of hepatitis B for its transmission. Hepatitis D is uncommon in the general population of North America, although it may occur in intravenous drug users and persons with frequent exposure to blood products (e.g., persons with hemophilia) as well as their sexual contacts.[74]

Hepatitis E is an enterically transmitted virus, often associated with contaminated water sources; it is most commonly found in Asia, the Middle East, South America, and Latin America. Hepatitis E has been diagnosed in the United States primarily in individuals traveling from low-resource countries, although it can be associated with foodborne transmission. This disease is frequently associated with only mild symptoms, and does not result in a carrier state.[74]

Hepatitis can also result from generalized infection by other viruses, including cytomegalovirus, Epstein-Barr virus, herpes simplex virus, and measles virus. Nonviral causes of liver inflammation include autoimmune disease, bacterial sepsis, and syphilis. Hepatitis can also be chemically induced by chronic alcohol ingestion or by medications such as acetylsalicylic acid (aspirin), acetaminophen (Tylenol), phenytoin (Dilantin), isoniazid (Niazid), and rifampin (Rifadin).

Symptoms common to all types of hepatitis include jaundice, dark-colored urine, abdominal pain, clay-colored stools, fatigue, loss of appetite, and nausea or vomiting. Laboratory abnormalities include elevated liver function tests. Testing for antibodies to the various viral causes of hepatitis, followed by consultation or referral for management, is appropriate.

Hepatitis A

Hepatitis A virus (HAV) is an RNA virus transmitted through the fecal–oral route. Most cases of hepatitis A arise during community-wide outbreaks. In the United States, vaccination has been successful in limiting the rate of infection, although it still occurs occasionally. In adults, most cases are symptomatic.[75] Contaminated water and food (especially shellfish) are typical sources of infection, with most cases being associated with close personal contact with an infected person, although blood-borne transmission has been documented in both infants and

adults.[74]

Hepatitis A has an incubation period of 28 days (range: 15–50 days), with the virus being shed through the feces approximately 2 weeks prior to the emergence of clinical symptoms. The infection has a short acute phase of 10 to 15 days, with symptoms resolving within 2 months, although as many as 15% of symptomatic persons have prolonged or relapsing disease lasting up to 6 months.[74] This self-limited disease does not result in chronic infection and is managed with symptomatic treatment and monitoring for worsening liver disease.[74]

Serologic testing with a finding of immunoglobulin M (IgM) antibody is required to confirm infection. IgM anti-HAV usually becomes detectable 5 to 10 days before the onset of symptoms and can persist for up to 6 months after infection. Immunoglobulin G (IgG) anti-HAV appears early in the course of the disease and indicates lifelong protection against the disease.[76]

Since 1995, two licensed inactivated hepatitis A vaccines have been available in the United States. Recommendations for hepatitis A immunization are described in detail in the *Health Promotion Across the Lifespan* chapter.

Hepatitis C

Hepatitis C virus (HCV) is rarely identified at the time of exposure. The CDC estimated that more than 30,000 cases occurred in the United States in 2014, but only 2194 cases were reported.[75] Of the more than 3 million persons currently living with chronic hepatitis C, the majority were probably exposed during the 1970s and 1980s, when rates of this infection reached their highest point. Risk factors include birth between 1945 and 1965, ever use of injectable or intranasal drugs, long-term hemodialysis, known exposure (healthcare worker, organ or blood recipient from a positive donor), HIV infection, and abnormal liver enzyme test results. Healthcare workers in general, long-term partners of individuals who are hepatitis C positive, and those with a history of multiple partners or sexually transmitted diseases may be tested for HCV infection, although the utility of screening these individuals in unclear.[77,78]

Transfusion with unscreened blood is an uncommon risk today for contracting hepatitis C. In contrast, unsterile tattoos from unlicensed providers, whether in social settings or during incarceration, have been linked to an increased risk of hepatitis C.[79] All-cause mortality is increased by infection with hepatitis C; racial disparities in rates of infection and mortality from hepatitis C exist as indicated by higher rates among Mexican Americans, Hispanic Americans, and blacks compared to whites.[80]

The period of incubation for hepatitis C ranges from 2 weeks to 6 months. Approximately 30% of individuals newly infected with HCV are symptomatic.[75,77] Chronic disease develops slowly, and both excessive alcohol use and obesity have been associated with progression.

Targeted screening for the hepatitis C virus is based on risk factors and is described in detail in the *Health Promotion Across the Lifespan* chapter. Diagnosis of hepatitis C infection is made by serum enzyme immunoassay (EIA) or chemiluminescent immunoassay (CLIA) detection of antibody to the virus. Hepatitis C antibody testing is accurate 4 weeks to 6 months after exposure. If the test for antibody to hepatitis C is positive, qualitative and quantitative HCV-RNA polymerase chain reaction (PCR) testing should be performed. Because of the high

(70% to 85%) incidence of chronic disease, serum alanine aminotransferase (ALT) and quantification of HCV-RNA (viral load) should be performed.[77,80]

Currently, there is no vaccine that can prevent infection with hepatitis C although identification of treatments including curative therapies is rapidly evoloving.[78] Counseling, including use of barriers with sexual activity and avoiding sharing intravenous drug paraphernalia, is essential. Treatment for acute and chronic hepatitis C should be managed by a physician familiar with the currently available therapeutic regimens.

Conditions of the Genitourinary System

The close proximity of the urinary tract to the reproductive organs means that women frequently call a midwife for management of urinary tract symptoms. Outpatient evaluation and management of uncomplicated cystitis is an essential skill for midwives, as are prompt recognition and triage of more serious conditions. Definitions and diagnostic criteria for urinary tract infections are listed in **Table 6-15**.[81,82]

Table 6-15	Definitions and Diagnosis of Urinary Tract Infections
Asymptomatic bacteriuria	Urine culture demonstrating ≥ 10^5 colonies of no more than 2 different microorganisms from appropriately collected clean catch midstream urine specimen culture in a woman without fever (≤ 38°C), urgency, frequency, dysuria, or suprapubic tenderness and with no history of indwelling catheter use within prior 7 days
Cystitis	Infection limited to the lower urinary tract, typically with symptoms of dysuria, frequency, and urgency
Pyelonephritis	Infection ascending to renal system, accompanied by fever, flank pain with or without lower urinary tract symptoms but with bacteriuria
Uncomplicated UTI	Cystitis or pyelonephritis occurring in a woman with normal genitourinary tract and no complicating factors
Complicated UTI	Cystitis or pyelonephritis occurring in a woman with recurrent UTIs, functional or structural abnormalities, an indwelling catheter, or associated systemic comorbidities such as metabolic disorders
Recurrent UTI	Recurrence involves ≥ 2 UTIs within 1 year *Relapse or persistent infection* occurs when the infective organism is identical to the pretreatment organism and is typically related to persistence within the genitourinary tract and occurring within 2 weeks of completion of the original treatment *Reinfection i*mplied infection with a new organism after adequate treatment but can include a new bacterial strain or a previously isolated strain that has persisted in the colonizing flora of the gut or vagina; typically a reinfection occurs > 2 weeks after the original treatment or following a negative test of cure
Diagnostic criteria for acute UTI	In a woman with a normal urinary tract and no other recognized cause for signs and symptoms, and no urinary catheter in place within the previous 48 hours, the diagnosis of UTI is confirmed by the presence of urgency, frequency, dysuria, suprapubic tenderness, costovertebral angle pain or tenderness, or fever (> 38°C) if ≤ 65 years of age AND 1. A positive urine culture of ≥ 10^5 CFU/mL with no more than 2 species of microorganisms OR 2. A positive urine culture of ≥ 10^3 CFU/mL with no more than 2 species of microorganisms with urinalysis demonstrating: a. positive dipstick for leucocyte esterase and/or nitrite b. pyuria (urine specimen with ≥10 WBC/mm^3 or ≥ 3 WBC/high power field of unspun urine c. microorganisms seen on Gram stain of unspun urine

Abbreviations: CFU, colony-forming units; UTI, urinary tract infection; WBC, white blood cell.

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Acute Cystitis

Uncomplicated acute cystitis is defined variously as the presence of 1000 or more colony-forming units (CFU) per milliliter of urine in a culture from a catheter specimen for symptomatic women or, more traditionally, as 100,000 or more CFU per milliliter whether or not symptoms of a lower urinary tract infection (UTI) are present. However, lower CFU counts may be associated with infection and in a woman with symptoms, a CFU of less than 100,000 CFU per milliliter should be interpreted with caution.

"Asymptomatic bacteriuria" is the term applied when symptoms are absent and 100,000 CFU per milliliter or more of a single organism grow in a urine culture. The predominant organism implicated in uncomplicated lower urinary tract infection is *E. coli*, with *Staphylococcus saprophyticus*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Enterobacter*, and *Enterococcus* species making up the majority of the other pathogens that are associated with UTI.[81-83]

Lower urinary tract infections are among the most common infections that women experience. Once a woman has such an infection, she is at risk for recurrence, often within a few months. Risk factors for UTI include sexual activity, barrier contraceptive and/or spermicide use, vaginal infections, trauma/or manipulation, increasing age, and genetic susceptibility.[84] Symptoms of a lower UTI include pain on urination, increased voiding frequency, and persistent suprapubic or low back pain. Because the symptoms of UTI overlap with those of sexually transmitted infections such as chlamydia, sexually active young women should be screened as indicated[84] and described in the *Health Promotion Across the Lifespan* chapter.

Lower UTI is frequently diagnosed based on symptoms and urinalysis findings consistent with bacteriuria and pyuria, in the absence of vaginal pain or discharge.[85] When cystitis is suspected, a culture and sensitivity should be obtained while the woman is symptomatic to confirm that a UTI is the cause of symptoms and to ensure that an antibiotic is prescribed to which the pathogen is sensitive. Between 30% and 50% of women with one UTI will have a recurrence within 1 year.[84]

Recurrent lower UTI is diagnosed when the woman has three or more infections proven by culture within a year. Identifying characteristics of uncomplicated recurrences include frequent sexual activity, a prior history of pyelonephritis, and rapid symptom resolution after treatment. Nocturia, hematuria, dyspareunia, and persistent symptoms after antibiotic therapy are indicative of another source of irritation, such as interstitial cystitis.[86] Among postmenopausal women, recurrent lower UTI is associated with urinary incontinence, cystocele, and prolapse. The use of vaginal estrogen creams or rings help prevent recurrent lower UTI in this population by restoring integrity to the vaginal and urethral tissues affected by estrogen deprivation.[86]

Recommendations for treatment of women with lower UTI have become more complicated as resistance to various antibiotics has emerged.[82] For low-risk women, judicious use of hydration, frequent voiding, anti-inflammatory analgesics, and probiotics with Lactobacilleae may become the first-line therapy, to be supplemented with antibiotics if symptoms persist for three days.[87] Nitrofurantoin (Macrodantin) or the combination of nitrofurantoin monohydrate

with macrocrystals (Macrobid) is preferred if there is no suspicion of pyelonephritis, or TMP-SMX (Bactrim) if that regimen has not been used within 3 months. Amoxicillin or ampicillin should never be used as a sole agent; fluoroquinolones, amoxicillin–clavulanate (Augmentin), and beta-lactams should be reserved for more serious infections whenever possible or if the recommended drugs are contraindicated.[82]

Despite popular belief, cranberry products are not useful in resolving UTIs and may exacerbate symptoms given the acidity of cranberry is irritating to the bladder. Cranberry is not recommended for treatment of a UTI. Studies of use of cranberry products for prevention of UTIs have had conflicting findings and most of the studies conducted to date had methodologic problems that limit generalizing their findings.[88] Although there is little harm in the use of these products, cranberry should not be recommended as an effective prevention strategy at this time.

Interstitial Cystitis

Interstitial cystitis is a persistent inflammatory bladder condition that is associated with suprapubic or retropubic pain, dysuria, frequency and urgency, dyspareunia, and nocturia. The term "painful bladder system" is used as an alternative when symptoms do not meet all criteria for diagnosis but no other cause of inflammation and pain can be identified. Over time, the bladder wall can become irritated or scarred, leading to the development of submucosal bleeding. Diagnosis is often delayed because the symptoms are mistaken for lower urinary tract infection. No evidence can be found for a microbial pathogen; however, some women report an acute UTI prior to the onset of symptoms. Numerous potential contributors to causation have been proposed, and links to other chronic pain syndromes are recognized.[89] More than 3 million adult women are estimated to have pain and urinary symptoms of interstitial cystitis/painful bladder syndrome in the United States.[90]

Midwives can offer women with interstitial cystitis/painful bladder syndrome counseling on restricting alcohol and possible food triggers such as spicy or high-acid foods, caffeine, and chocolate; tobacco cessation; and bladder training. Referral for further evaluation, including a possible biopsy to rule out bladder cancer, and medication is often warranted.[90]

Pyelonephritis

Women with abdominal pain should be evaluated for acute pyelonephritis, an inflammation of the kidneys. The characteristic presentation of pyelonephritis is severe flank pain and fever with associated chills, nausea or vomiting, and painful urination. Assessment of costovertebral angle tenderness (CVAT) by striking beneath the 12th rib in the acute angle formed with the spine can help to assess the location of the pain (**Figure 6-4**). The risk factors and pathogens associated with pyelonephritis are the same as those for lower UTI. The diagnosis should be confirmed by a urinalysis, which will be positive for white blood cells or the presence of pyuria, and by urine culture. Additional laboratory tests include blood urea, creatinine, and CBC; an elevated leukocyte count is an indicator of severity.[84]

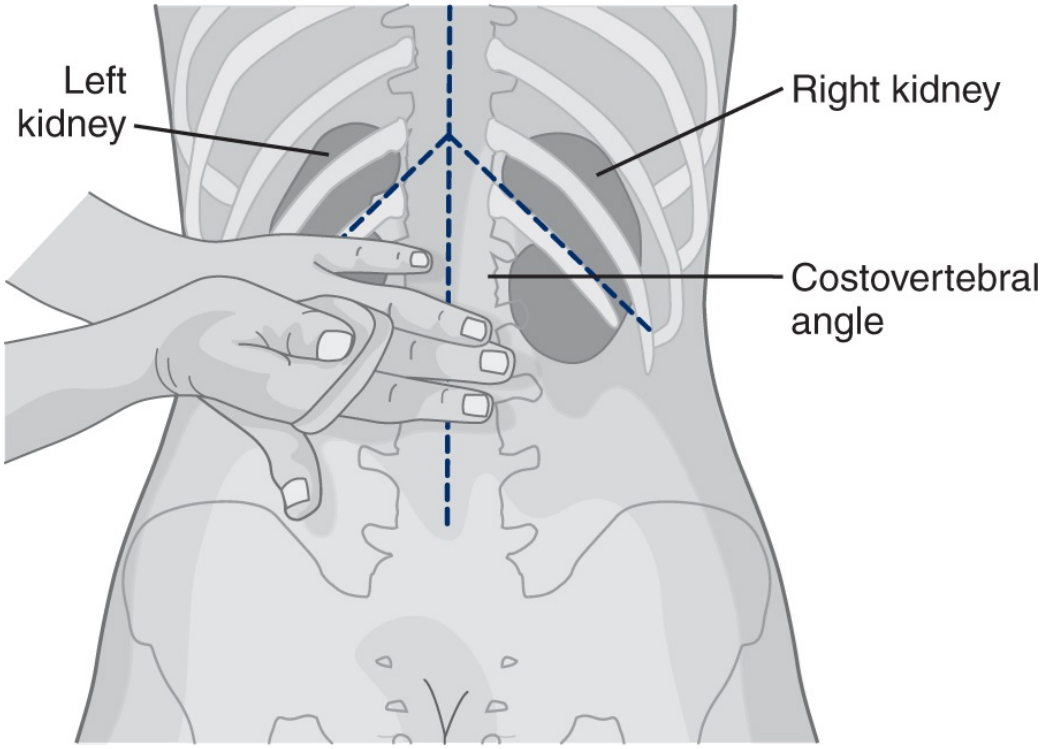


Figure 6-4 Assessing for costovertebral angle tenderness.

Note: Choose the technique that is most comfortable for both examiner and patient.

1. With the woman seated on the examination table, place the palm of one hand over her costovertebral angle on one side of the patient's back. Make a fist out of your other hand. Use the ulnar surface of your fist for striking. Strike the back of the hand that is over the costovertebral angle with the fist of your other hand.

OR

2. Put your hands around the woman's waist and locate by the palpation the costovertebral angles with the flat part of your index and middle fingers on each hand. Alternately strike each costovertebral angle with your fingers by a sudden, upward motion of your hand.

Indications for physician consultation or referral include evidence of severe upper urinary tract infection, frequent recurrence of a UTI, infection with resistant organisms or in women with multiple antibiotic allergies, comorbidities that may affect immune status, comorbidities that may affect the structure or function of the urinary tract, hematuria that persists after treatment, or observed anatomic abnormality.[82] Physician consultation is required when pyelonephritis is suspected; a pending urine culture should not delay consultation. Treatment varies depending on whether it is delivered on an inpatient basis or at an outpatient site. Commonly prescribed drugs include various combinations of ciprofloxacin (Cipro), cephalosporins, fluoroquinolones, aminoglycosides, and penicillins.[82]

Kidney Stones

The usual symptoms of nephrolithiasis (kidney stones) include severe renal colic caused by the

stone passing into the ureter. As the stone enters the bladder, it triggers symptoms resembling those of a urinary tract infection. Hematuria is common but not required for diagnosis. Helical computed tomography without contrast is the preferred confirmatory test.

When kidney stones are suspected, the midwife will need to refer the woman to a specialist for management. While arranging transfer, NSAIDs can be initiated for pain management and are as effective as opioids for this purpose. Small stones, less than 0.5 to 1 centimeter in diameter, can be allowed to pass without surgery, using effective pain management and increasing fluids.[91-93] Calcium-channel blockers and alpha-adrenergic agonists have been used to speed the passage of stones through the ureter, and are associated with decreased pain.[92] Surgical management of kidney stones that are large, obstructive, or associated with infection now relies on extracorporeal shock-wave lithotripsy, ureteroscopy, and percutaneous nephrolithotomy as means to avoid open procedures.

Women have a 7% lifetime risk of developing kidney stones.[94] Recurrence is common, and has been estimated to be as high as 50% within 5 years.[94]

Endocrine Conditions

Metabolic Syndrome

Obesity, especially central obesity, is associated with an increased risk of insulin resistance and elevated blood cholesterol. The National Cholesterol Education Program has defined metabolic syndrome as atherogenic dyslipidemia, elevated blood pressure, dysglycemia, a prothrombotic state, and a pro-inflammatory state.[30] **Table 6-16** provides one commonly accepted definition of metabolic syndrome in women.[95]

Table 6-16 **National Cholesterol Education Program Diagnostic Criteria for Metabolic Syndrome**

Positive diagnosis with any three of the following:
Waist circumference ≥ 88 cm in women
Serum triglycerides ≥ 150 mg/dL or treatment to reduce triglyceride levels
Serum HDL-C < 50 mg/dL or treatment for reduced HDL-C
Blood pressure ≥ 130/85 mm Hg or treatment for hypertension
Fasting plasma glucose ≥ 110 mg/dL or treatment for hyperglycemia

Abbreviation: HDL-C, high-density lipoprotein cholesterol.

Based on National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002 Dec 17;106(25):3143-3421.[95]

Women with one or more of these characteristics should be assessed with blood pressure measurement, waist circumference measurement, fasting glucose, and a full cholesterol panel. Counseling about lifestyle changes, including weight loss, increased exercise, and dietary changes, is essential. Women should be referred as appropriate for evaluation and pharmacologic treatment and/or management of hyperlipidemia, hypertension, or diabetes.

Diabetes Mellitus

The definition of diabetes mellitus is based on the underlying cause of hyperglycemia. “Prediabetes” is the term used to describe women with abnormal blood glucose values that do not meet the measurement that defines diabetes. **Table 6-17** distinguishes between four types of diabetes.[96] In addition to the four classifications listed in **Table 6-17**, a fifth term, pre-diabetes, is used to identify persons with abnormal plasma glucose values that do not reach criteria for diabetes. These individuals are at increased risk for developing diabetes and should be monitored closely.

Table 6-17 **Classification of Diabetes**

Classification	Description
Gestational	Diabetes diagnosed in the second or third trimester of pregnancy that is not clearly overt or

diabetes	preexisting to the pregnancy (type 1 or type 2) diabetes.
Type 1 diabetes	Diabetes resulting from beta-cell destruction, usually leading to absolute insulin deficiency
Type 2 diabetes	Diabetes resulting from progressive loss of pancreatic beta-cell insulin secretion in the face of increased insulin resistance
Other types of diabetes	Diabetes due to other causes such as cystic fibrosis, pancreatic disease, drug-induced or chemically induced

Based on American Diabetes Association. Standards of medical care in diabetes—2017. *Diab Care.* 2017;40(suppl 1):S1-S135.[96]

Type 1 diabetes is almost universally mediated by the immune system and causes pancreatic B-cell destruction. It is most commonly diagnosed in children or young adults (hence the older name of "juvenile" diabetes) and accounts for approximately 5% to 10% of all cases of diabetes.[96] *Type 2 diabetes*, which is the most common form of diabetes, is the result of resistance to insulin and the inability of the pancreas to increase insulin production to compensate for this resistance.

Together, diabetes types 1 and 2 affect more than 29 million persons in the United States, of whom approximately 8 million (27.8%) remain undiagnosed; 13.4 million of the people with diabetes are women.[97] Rates of diabetes increase as persons age, and in persons with obesity. A first-degree family member with diabetes, lack of physical activity, and smoking are other factors that increase risk.[96,98] A diet high in red meat, high-fat dairy, and processed carbohydrates and sweets increases the risk for developing diabetes regardless of other risk factors.[97] Ethnic groups with an increased risk of developing diabetes include non-Hispanic blacks, Mexican Americans and Puerto Ricans, Southwest American Indians, and Asian Americans. Although having a large-for-gestational-age infant is no longer considered a risk factor for diabetes, women with a history of gestational diabetes and/or polycystic ovary syndrome have demonstrated increased risk.[96]

Screening for diabetes mellitus should be performed for all women older than 45 years, and for anyone with a BMI of 25 kg/m^2 or greater (BMI $\geq$ 23 kg/m^2 among Asian Americans) and one or more risk factors for cardiac disease.[96] Screening tests include glycosylated hemoglobin A1c (HbA1c), fasting blood glucose, and the 2-hour glucose tolerance test.[96] HbA1c measures the average plasma glucose level over the last month; its results are expressed as the percentage of total hemoglobin. This test can be used to assess either the degree of elevated blood glucose over time in persons with a new diagnosis of diabetes or the degree of glycemic control in persons who are being treated for diabetes. Type 2 diabetes is diagnosed if the HbA1c result is equal to or greater than 6.5%.

Table 6-18 summarizes the criteria for the diagnosis of diabetes.[96,97] Any one of the three tests can be used but the diagnosis is not made until the same test is repeated twice. The repeat test requires a new blood sample and can be performed right away. Women newly diagnosed with diabetes should be referred to a healthcare provider skilled in treating women with diabetes.

Table 6-18 **Diagnosis of Diabetes Mellitus**

Test	Description	Glycemic Goals Following Treatment
Any one of the following three tests will confer the diagnosis of diabetes if repeated twice:		
Hemoglobin A1c[a] ≥ 6.5% or	Fasting not required.	< 7.0%
Fasting plasma glucose ≥ 126 mg/dL or	Fasting is defined as no caloric intake for at least 8 hours.	80–130 mg/dL
2-hour plasma glucose ≥ 200 mg/dL after a 75-g glucose load	This test will diagnose more individuals with diabetes than will hemoglobin A1c or a fasting plasma glucose level.	< 180 mg/dL 2-hour postmeal

Abbreviation: RBC, red blood cell.

[a] Hemoglobin A1c measures the percentage of glycosylated hemoglobin present, Glycosylated hemoglobin is hemoglobin that is coated in glucose. As plasma glucose levels rise, the amount of glycosylated hemoglobin increases. Glucose stays attached to an RBC for the life of the RBC and is therefore an average measure of plasma glucose levels for the prior 3 months.

Based on American Diabetes Association. Standards of medical care in diabetes—2017. *Diab Care.* 2017;40(suppl 1):S1-S135[96]; Inzucchi SE, Majumdar SK. Current therapies for the medical management of diabetes. *Obstet Gynecol.* 2016;127:780-794.[97]

Midwives can plan an important role in some components of care for women with diabetes including diet education, promotion of moderate exercise, counseling for weight loss, and smoking cessation. Women with diabetes need regular gynecologic care and family planning, and may have an increased risk for developing vaginal infections with candidiasis if glucose control is not optimal. All women with diabetes who desire a pregnancy should be encouraged to achieve glucose control prior to becoming pregnant.Hyperglycemia in early pregnancy is associated with miscarriage and congenital anomalies. Preconception counseling and care can have an important positive effect on pregnancy outcomes. Counseling guidelines for women with diabetes who are planning a pregnancy can be found in the *Health Promotion Across the Lifespan* chapter.

The American Diabetes Association recommends beginning lifestyle changes with the addition of the pharmaceutical agent metformin (Glucophage) as soon as type 2 diabetes is diagnosed.[96] Tight glucose control can reduce the incidence of microvascular complications and glycemic targets are based on long-term clinical studies that have identified plasma glucose levels associated with minimal progression of disease. However, this is a progressive disease and frequently second or third medications are added over time to help maintain euglycemia. Multiple new therapies have been developed in the last several years so that treatment can be individualized to minimize side effects and maximize glucose control. Most medication for diabetes work by improving insulin secretion or insulin sensitivity. Others suppress appetite or promote weight loss. Insulin therapy may also be recommended. If a midwife is participating in primary care services for a woman using medication to control diabetes, additional knowledge about the medications and their effects is needed because these medications have wide ranging physiologic effects.[97] The Resources section at the end of this chapter lists websites that have additional education and training about management of diabetes.

Thyroid Disease

Thyroid conditions are common among women of childbearing age, with an overall incidence of more than 5%.[99] These conditions are more common in women compared to men, and more common as age increases. To date, the U.S. Preventive Services Task Force has not made a recommendation for or against screening asymptomatic, nonpregnant adults for thyroid conditions.[100] Other experts recommend screening in adults, with the age of first testing ranging from 35 to 60 years.[101]

Thyroid-releasing hormones from the hypothalamus stimulate pituitary release of thyroid-stimulating hormone (TSH). In turn, TSH stimulates the production and release of triiodothyronine (T3) and thyroxine (T4) from the thyroid gland. Although T3 is the more active form of the thyroid hormones, little is produced in the thyroid itself. Instead, most T3 is produced from circulating T4 as it loses iodine.

When screening for or diagnosing thyroid disease, the first tests performed usually are measurements of TSH and free T4. Among the antibody tests, confirmation of thyroid peroxidase antibodies is associated with chronic autoimmune thyroiditis (Hashimoto's thyroiditis), the predominant cause of overt hypothyroidism. **Table 6-19** identifies the changes in screening laboratory values associated with common thyroid disorders.

Table 6-19 Thyroid Hormone Values in Thyroid Disorders

Condition	Thyroid-Stimulating Hormone	Free T_4	Serum T_3
Graves' disease (hyperthyroid)	Absent	Elevated	Elevated
Subclinical hyperthyroid	< 0.1 mU/L	Normal	Normal
Hypothyroid	High with primary hypothyroidism Low with secondary hypothyroidism	Low	Low/normal
Subclinical hypothyroid	Mildly elevated (< 10.0 mU/L)	Low/normal	

Hypothyroidism

Hypothyroid conditions result primarily from failure of the gland to produce adequate hormone and are more common than hyperthyroidism. The most common cause of hypothyroidism in the United States is the autoimmune disorder known as Hashimoto's thyroiditis. Other causes include treatment of women with Graves' disease, thyroidectomy, and medications. Secondary hypothyroidism, caused by pituitary or hypothalamic disorders, is uncommon. In primary hypothyroid states, the TSH level will be elevated; the pituitary gland secretes excess TSH in response to low levels of T3 and T4.

Uncomplicated primary hypothyroidism usually is treated with thyroid hormone supplementation. Management of hypothyroidism is complex; medication dosage is based on laboratory values and symptom relief. The midwife plays an important role in screening women who present with symptoms of hypothyroidism. When thyroid disease is suspected or confirmed with laboratory values, women should be referred to a healthcare provider

specialized in the treatment of thyroid disease.

Hyperthyroidism

The single most common cause of a hyperthyroid state (also referred to as thyrotoxicosis) is Graves' disease, an autoimmune disease that has a higher incidence among women than men and is the cause of more than 10% of all cases of thyrotoxicosis in young adults. The thyroid is typically enlarged and tender in hyperthyroidism. Many women will notice irritation of the conjunctiva, diplopia, or blurred vision, or present with proptosis (bulging of the eye or displacement forward of the eye in its orbit) and periorbital edema. **Table 6-20** lists the common signs and symptoms of thyroid disease.

Table 6-20 Common Clinical Features of Thyroid Disease

Hypothyroidism	Hyperthyroidism
Bradycardia	Anxiety, nervousness
Cold intolerance	Diarrhea
Constipation	Fatigue
Delay in deep tendon reflex relaxation	Goiter
Depression	Increased sweating
Decreased sweating	Heat intolerance
Dry skin, brittle nails, thinning hair	Hyperreflexia
Edema (nonpitting)	Irregular menses
Fatigue, sleepiness	Increased appetite with weight loss
Goiter	Lid lag, stare
Headaches	Ophthalmopathy
Hoarseness	Palpitations with angina
Lethargy	Tremor
Loss of appetite	Warm, moist skin
Muscle weakness	Weakness
Muscle cramping or pain	
Menorrhagia, amenorrhea (late)	
Pallor	
Slowed speech and body movements	
Weight gain (occasionally loss)	

Other causes of hyperthyroidism include toxic multinodular goiter, toxic adenomas, early stages of Hashimoto's thyroiditis, and postpartum thyroiditis. This disease has also been associated with very high levels of circulating human chorionic gonadotropin (hCG), such as those found in women who have a hydatidiform mole. Subclinical hyperthyroidism is most commonly associated with suppressive thyroid therapy, although mild Graves' disease is a possible cause.

The midwife should refer for physician consultation any woman in whom hyperthyroidism is diagnosed or suspected. Women who are being treated for thyroid disease will need frequent

laboratory assessment of circulating thyroid hormone levels.

Neurologic Conditions

Headaches

Headaches have many causes and differing degrees of risk, and often are divided into two types: primary and secondary. Primary headaches include tension headaches, migraine, and cluster headaches, whereas secondary headaches are rarer and are related to underlying disease such as a neoplasm. A complete health history should be collected for women with headaches, including a clear description of symptoms associated with onset and duration of headaches, frequency of severe headache, frequency of mild headache, level of interference with normal activities, and frequency of medication use to relieve symptoms. Additional questions can be asked about nausea, light sensitivity, and noise sensitivity, all of which suggest a migraine headache. A headache diary can be of help in diagnosis and treatment.[102]

Conditions that can provoke headache as a symptom, such as sinus infection or concussion, are not discussed here, nor are vascular emergencies such as aneurysm and arteriovenous (AV) malformation. Warning signs identified during the history taking and physical examination that require emergency attention are summarized in **Table 6-21**.

Table 6-21	Warning Signs of Headache Emergencies
Rapid onset of severe symptoms	
Alteration in consciousness or mental status	
High fever	
Severe vomiting, especially without nausea	
Neck stiffness	
Neurologic symptoms:	
Weakness	
Loss of balance	
Numbness	
Paralysis	
Vision changes	
Pattern of worsening headache	
Increasing medication use to control headache	
Direct association with exercise or position change	

Tension Headache

According to the International Headache Society (IHS), tension headaches are characterized by bilateral pain, with a pressing or tightening quality and mild to moderate intensity.[103] Such headaches are relatively common, with prevalence rates ranging from 30% to 78%. Sensitivity to light or sound may be present. While the duration of tension headaches may vary from minutes to multiple days, normal physical activity does not affect the sensation. Other than in the most severe cases, nausea does not occur.[103]

Tension-type headaches are classified based on their frequency: infrequent episodic (less than 1 day/month), frequent episodic (1–14 days/month), and chronic.[103] Most individuals with infrequent or moderate-frequency tension headaches do not seek treatment. Among persons with chronic tension headaches, it has been reported that most are able to continue working and managing daily activities, but they are more likely to have mood or anxiety disorders, decreased ability to function, disturbed sleep, and decreased emotional well-being.[104]

Caring for women with episodic tension headaches includes respecting the woman's report of symptoms, assessing for the presence of anxiety or depression, and promoting lifestyle changes that include relaxation and stress reduction. NSAIDs or acetaminophen can be used as initial therapy to relieve symptoms, especially with episodic tension headaches. Combination medications that include caffeine with aspirin (e.g., Excedrin), ibuprofen (Advil), or acetaminophen (Tylenol) are useful when a single medication is not adequate.[105] Both acupuncture[106] and cognitive-behavioral therapy[107] have shown promise in treating tension-type headaches. Prophylactic treatment is not effective or useful in managing episodic headache.[108]

Chronic tension headache is a more severe condition that often requires prophylactic medication. The tricyclic antidepressants—most commonly, amitriptyline—can be used for this purpose; biofeedback, stress reduction, and other nonpharmacologic interventions to interrupt the cycle of headaches can be used as additional modalities.[109,110] When women report chronic headache over several months while using pain relievers, a medication overuse headache should be suspected, and a plan to stop or taper the drugs in question should be implemented. Women with chronic headache should be referred to a neurologist for management.

Migraine Headache

Migraine headache is both common and frequently underdiagnosed in primary care settings, with an estimated 25% of cases being missed. This tendency suggests that a woman with episodic headaches should be assumed to have migraines unless otherwise diagnosed.[111] The prevalence of migraine among women is estimated to be more than 20%.[112] Among all races, women are more likely than men to be diagnosed with migraine; the condition is more common among Caucasians. Incidence of new-onset migraine in women is highest in the decade after menarche, with the median age at new onset being 25 years.[113] Prevalence rises through the fourth decade of life, but then declines thereafter. Migraine is relatively rare among postmenopausal women, and new-onset headaches in this age group should be evaluated carefully for other causes.

Both genetic predisposition and environmental triggers appear to be involved in the development of migraine headache. The underlying cause is an abnormally increased sensitization to neuronal stimuli, beginning in the cerebral cortex and affecting the trigeminovascular system.

The diagnosis of migraine is subdivided based on the presence or absence of an aura, complications, and associated conditions. The International Headache Society defines *migraine without aura* as a "recurrent headache disorder manifesting in attacks lasting 4–72

hours."[103] Typical characteristics of such headaches are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity, and association with nausea and/or photophobia and phonophobia.[103] Migraine without aura in women of childbearing age is often related to the menstrual cycle. *Migraine with aura* is characterized by "recurrent attacks, lasting minutes, of unilateral fully reversible visual, sensory or other central nervous system symptoms that usually develop gradually and are usually followed by headache and associated migraine symptoms."[103] Visual, sensory, and speech symptoms associated with aura are reversible.

Migraine is an intermittently recurring event, with well-defined stages: (1) the premonitory stage; (2) aura (if present); (3) headache; and (4) postdrome (**Table 6-22**).[114] The premonitory stage is noted in approximately 30% of those individuals with the condition and occurs up to 48 hours before onset of the actual headache; tiredness, fatigue, malaise, mood changes, and stomach upset are the most common symptoms.[114] Following the headache, variable symptoms may persist for hours or days. Over time, migraine may progress from an episodic to a persistent condition with almost daily symptoms. Chronic migraine (defined as occurring on 15 or more days per month) is accompanied in some individuals by both physiologic changes in pain perception and anatomic alterations within the brain.[103]

Table 6-22 **Symptoms of Migraine Headaches and Criteria for Diagnosis**

Migraine Phase	Symptoms
Prodrome	
Symptoms may or may not precede headache by several hours	Fatigue, depression, irritability, food cravings, yawning, muscle tenderness, neck stiffness, abnormal sensitivity to light, sound and smell
Migraine without Aura	
Symptoms present at least 5 times lasting 4-72 hours (untreated or unsuccessfully treated)	The following characteristics (≥ 2): Unilateral location Pulsating quality Moderate or severe pain intensity Aggravated by or causing avoidance of routine physical activity Symptoms during a headache (≥ 1): Nausea, vomiting or both Photophobia and phonophobia
Migraine with Aura	
Symptoms present at least 2 times	Symptoms listed above with fully reversible aura symptoms (≥ 1): Visual Sensory Speech and/or language Motor Brainstem Retinal The following characteristics (≥ 2):

	One aura symptom occurring gradually over > 5 minutes Individual aura symptom lasting 5–60 minutes One aura symptom is unilateral Headache accompanies or follows aura within 60 minutes
Postdrome	
Symptoms may or may not present after resolution of the headache	Feeling drained or exhausted, difficulty concentrating, stiff neck, mild residual head discomfort

Reproduced with permission from Deneris A, Allen PR, Hayes EH, Latendresse G. Migraines in women: current evidence for management of episodic and chronic migraines. *J Midwifery Womens Health* 2017;62(3):270-285.[114]
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A number of lifestyle changes may improve the frequency of migraine. These modifications include the avoidance of substances that the individual can identify as headache triggers (e.g., cheese, alcohol, chocolate), stress reduction, and stable patterns of eating and rest.

Pharmacologic therapy is used to treat acute attacks (abortive therapy) or reduce the severity and frequency of subsequent episodic attacks (preventive therapy).[114] Abortive treatments for women with mild, infrequent cases of migraine, include NSAIDs taken in maximal doses, or acetaminophen (Tylenol) 1000 mg. Either type of medication may also be combined with caffeine in OTC preparations. Historically, the most common abortive therapies were ergotamine preparations such as ergotamine tartrate/caffeine (Cafergot). Ergotamine is teratogenic, so women of childbearing age must use an effective and reliable contraceptive method when using it, and women planning a pregnancy require a change in therapeutic management. Ergotamines are less frequently used today as more effective medications have been identified. The triptans, such as sumatriptan (Imitrex) and naratriptan (Amerge) are serotonin receptor agonists and are quite effective as abortive therapy. Triptans are available as oral, intranasal, oral disintegrating tablets, or subcutaneous formulations. The U.S. Food and Drug Administration (FDA) has issued a caution with regard to use of triptans and selective serotonin reuptake inhibitors (SSRIs) because both drug classes increase serotonin, which can place the woman at risk for serotonin syndrome. If the triptan is used infrequently, the risk of serotonin syndrome is very small.

Preventative therapy for migraine includes agents from a number of drug classes, such as the tricyclic antidepressants, antiepileptics, beta-blockers, calcium-channel blockers, ACE inhibitors, and angiotensin receptor blockers. Women requiring long-term migraine management should be under the care of a neurologist or another practitioner skilled in the management of headache.

Menstrual-related migraines may benefit from long-cycle combined hormonal therapy or progesterone-only contraception, particularly the levonorgestrel intrauterine system (IUS). Decreasing the frequency of estrogen withdrawal minimizes the headache response.[115] When estrogen is included in treatment, the woman should be monitored for possible increases in the frequency of headache, which would mandate the use of progesterone-only methods. Combined hormonal contraception is contraindicated in women who have migraine with aura, due to increased risk of stroke (Medical Eligibility Criteria for Contraceptive Use [MEC] category 4); among women who experience migraine without aura, the CDC considers the use of

combined hormonal contraception acceptable but not optimal (MEC category 2).[115] Additional discussion can be found in the *Hormonal Contraception* chapter. Short-term use of a triptan in the days prior to the menses may also interrupt the headache cycle. Midwives can care for women with migraines but to do so, additional education regarding different presentations, danger signs, drug effects and prescribing considerations is needed.[114]

Epilepsy

Epileptic seizures are transient excessive aberrant or synchronous neuron activity that causes physical signs or symptoms.[116] Diagnosis requires two unprovoked or reflex seizures more than 24 hours apart; or one such seizure and a likelihood greater than 60% that further seizures will occur within 10 years; or diagnosis of a syndrome that includes epilepsy as one of its features.[116]

The classification of seizure disorders is based on whether the seizures are generalized or partial, with these categories being further subdivided by specific patterns of associated symptoms. Generalized seizures are caused by significant activity in both lobes of the brain, and almost always involve changes in consciousness. Tonic–clonic seizures (grand mal), absence (petit mal) seizures, and brief myoclonic seizures are all examples of generalized seizure activity. Partial seizures may or may not involve alteration in consciousness; if this symptom is present, they are referred to as complex partial seizures.

Seizure activity in women of childbearing age may peak around the times of ovulation and menstruation, or less commonly with an inadequate luteal phase—a condition known as catamenial epilepsy.[117] The midwife's role in caring for women diagnosed with a seizure disorder is primarily focused on the effect of epilepsy medications on reproductive health, the management of contraception, and assessment of risks associated with epilepsy or medications used to treat epilepsy. Menstrual disorders, polycystic ovary syndrome, decreased libido, and infertility are all associated with epilepsy and with some antiepileptic medications.[117] Decreased bone mineral density has also been linked to the use of antiepileptic drugs, although the seizure-associated falls' contribution to overall fracture rates is not clear.[118] Some anticonvulsants are associated with an increased risk for teratogenic effects, and pregnant women on these medications should be referred to a genetic specialist for counseling. A review of preconception care for women with epilepsy can be found in the *Health Promotion Across the Lifespan* chapter.

Contraceptive management for women with epilepsy is complicated by the interaction of estrogen-containing hormonal products with antiepileptic medications that affect the cytochrome P450 metabolic pathway—for example, barbiturates, carbamazepine (Tegretol), lamotrigine (Lamictal), phenytoin (Dilantin), and topiramate (Topamax; in larger doses).[119] These medications may interfere with the effectiveness of hormonal contraceptives and increase the risk of unintended pregnancy. The contraceptive implant etonogestrel (Nexplanon) has also been shown to have an increased risk of failure among women taking these types of antiepileptic agents.[119] Both depot medroxyprogesterone acetate (DMPA) and the levonorgestrel-containing IUS (Mirena) are safe for use in women taking antiepileptic

therapies, as is the copper intrauterine device (ParaGard); however, some authorities suggest DMPA may need to be given at 10-week rather than 12-week intervals.[119] Use of a barrier method as a secondary method of contraception can be recommended. More information about contraception, including evidence-based criteria for eligibility, can be found elsewhere in this text. All women with epilepsy should receive counseling regarding risks associated with pregnancy, and referral to a maternal–fetal medicine specialist should be considered.

Another important concern among women with epilepsy is the increased risk of depression and suicide. Both the psychological and physical stresses of living with the disease and the side effects of medication factor into these risks.[120] Screening for mood changes and suicidal ideation during gynecologic or well-woman visits is particularly appropriate when caring for women with this disorder.

Carpal Tunnel Syndrome

The carpal tunnel carries the median nerve through the wrist, where compression from edema, inflammation of the tissue, or anatomic distortion can produce the classic symptoms of carpal tunnel syndrome (CTS)—tingling, numbness, or altered sensation across the palmar surface of part of the thumb, the first two fingers, and part of the ring finger in the affected hand. Over time, CTS almost always becomes bilateral. The woman may report that pain and numbness are worse at night. **Figure 6-5** illustrates the area affected by carpal tunnel syndrome.

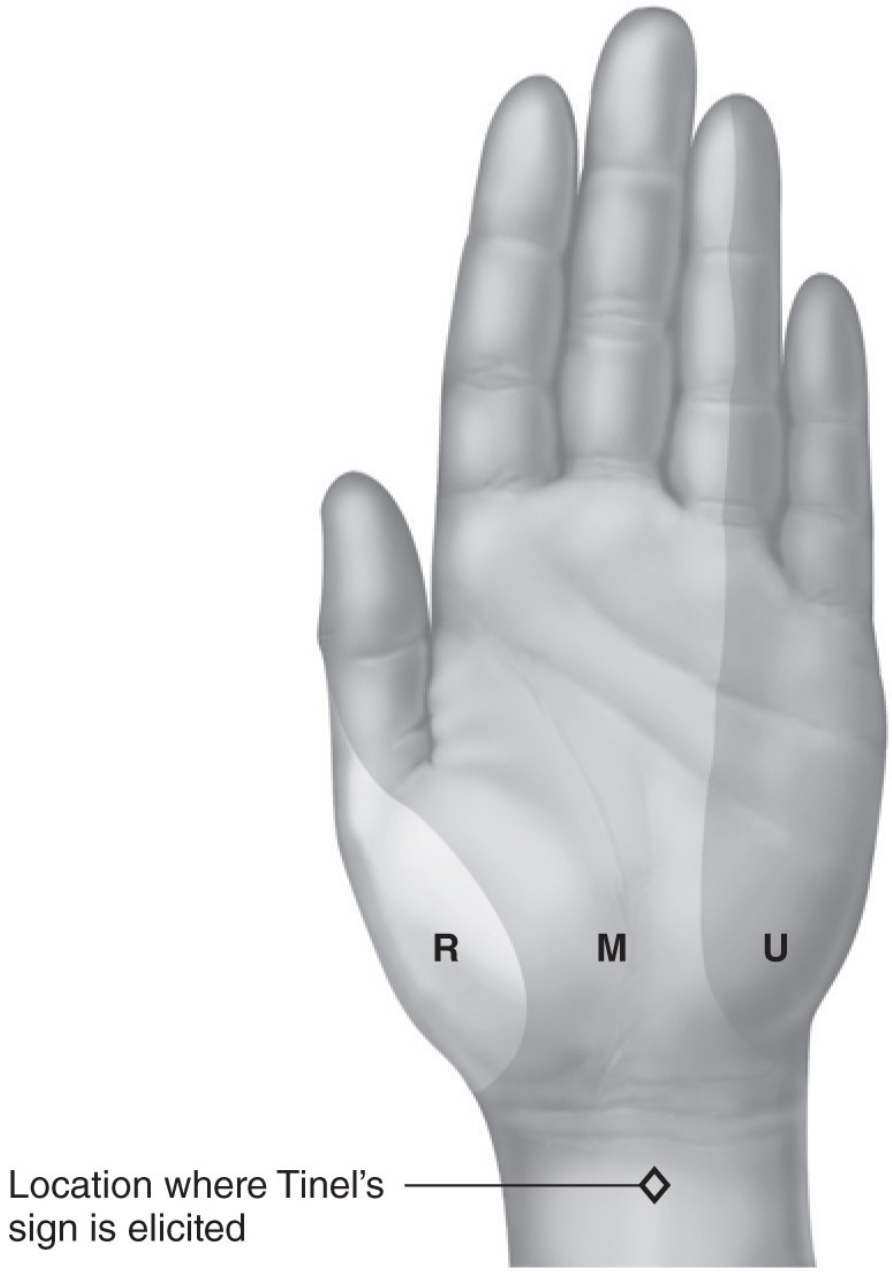


Figure 6-5 Distribution of sensation by the radial (R), median (M), and ulnar (U) nerves.

Female gender, pregnancy, increasing age, obesity, history of wrist injury or arthritis, diabetes, and hypothyroidism have all been associated with increased risk of developing CTS.[121] Recent research indicates that work-related activities play a significant role in the development of CTS.[122] When women engaged in a repetitive motion of the wrists report numbness or loss of sensation in this pattern, carpal tunnel syndrome is often assumed to be a causative factor. Other forms of nerve damage can also present with similar symptoms. If additional symptoms unrelated to the median nerve are present, prompt referral is warranted.

Two simple tests can help to confirm that the woman's report is consistent with carpal tunnel syndrome (**Figure 6-6**). First, tapping over the wrist crease in the midline should produce

tingling in the affected area; this is referred to as Tinel's sign (Figure 6-6B). Second, holding the wrist flexed for 45 to 60 seconds and releasing it should produce symptoms; this is referred to as the Phalen test (**Figure 6-6A**). Electromyography and nerve conduction studies will confirm the diagnosis of carpal tunnel syndrome.

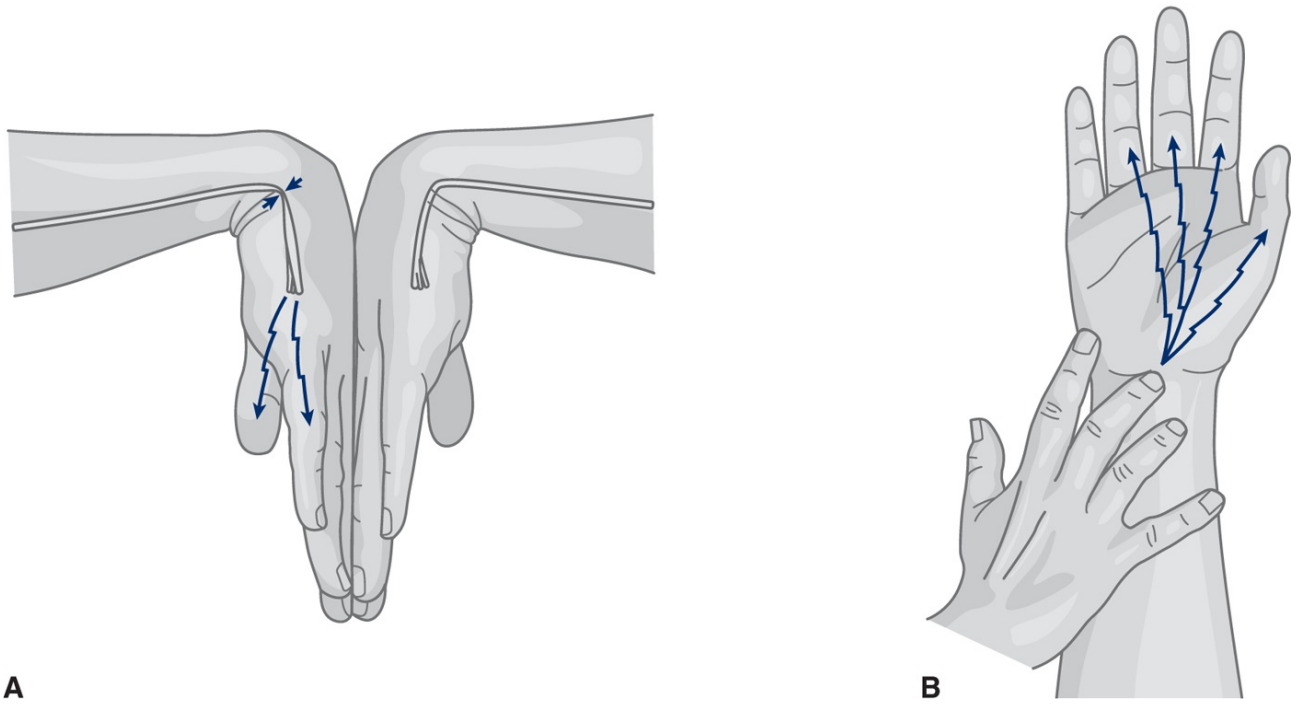


Figure 6-6 A. Phalen test. **B.** Tinel's sign.

Initial treatment is conservative, and may begin with splinting at night to place the wrist in a neutral position. Only limited evidence supports the effectiveness of this technique.[123] Other conservative therapies, ranging from nerve gliding techniques to wrist manipulation to yoga and aerobic exercise, have been recommended, but there is little evidence of their effectiveness.[124] If symptoms are severe, persist, or worsen with conservative management, the woman should be referred to an orthopedic surgeon or neurologist for further evaluation. Treatment may then include injections of steroids or surgical release of the nerve. If left untreated, the condition will worsen over time and eventually lead to permanent decrease in sensation.

Musculoskeletal Disorders

Sprains and Strains

Musculoskeletal complaints account for approximately 10% to 15% of primary care visits. A systemic approach to any complaint of musculoskeletal pain will help the midwife evaluate for the multiple differential diagnoses that are possible. When evaluating a woman for musculoskeletal pain, it can be helpful to have her point to the pain and describe any associated injury. The initial assessment includes determination of localized versus diffuse, acute versus chronic, and inflammatory versus non-inflammatory conditions. Sprains are stretched or torn ligaments; strains are stretched or torn muscles or tendons. **Table 6-23** compares common signs and symptoms of the two conditions.

Table 6-23 Differential Diagnosis for Sprain or Strain

Type of Injury	Symptoms
Sprain	Bruising over the joint
	Limited flexion/extension of joint
	Pain
	Swelling
Strain	Limited contraction/release of muscle
	Muscle spasm
	Pain
	Swelling

Sprains

The most commonly sprained joint is the ankle, with more than 600,000 ankle injuries occurring each year in the United States.[125] The knee, wrist, and thumb are other commonly injured sites. Twisting or overstretching a joint can cause the ligaments securing the bones to be stretched or torn. Once injured, a joint is prone to reinjury, possibly from persistent laxity in the ligament, or damage to the nerves and muscles associated with the joint. Approximately 20% of ankle sprains result in some degree of permanent instability.[126]

Sprains are graded based on degree of injury, as shown in **Table 6-24**.[127] Ankle stability can be evaluated using the talar tilt and anterior drawer tests (**Figure 6-7**). Normal inversion of the ankle is less than 35 degrees; normal plantar flexion is less than 50 degrees. Whenever evaluating the ankle, the opposite foot should also be assessed for comparison purposes, as there is significant individual variation in normal joint flexion. Diagnosis of an ankle sprain includes ruling out a fracture. The Ottawa rules used to determine the need for X ray of the ankle include the presence of pain near the affected ankle, and either tenderness on the posterior aspect or tip of the malleolus or inability to bear weight for at least four steps both immediately after the event and in the examining room.[127]

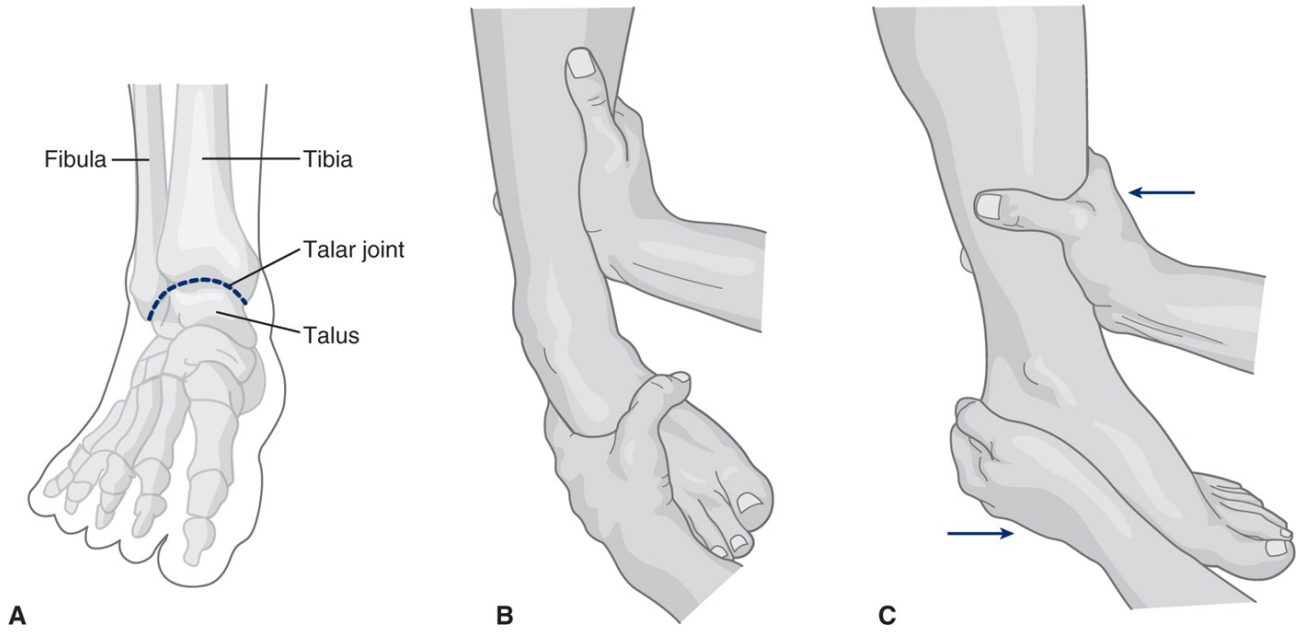


Figure 6-7 Talar tilt and anterior drawer tests to assess ankle sprain. **A.** Talar joint. **B.** Talar tilt. **C.** Anterior drawer test.

Table 6-24 **Sprain Grades**

Grade	Symptoms
Grade 1	Ligament is stretched; may have micro-tears Minimal swelling; full weight bearing possible No instability in the joint
Grade 2	Macro-tears but ligament remains attached Moderate swelling and bruising Difficulty with weight bearing Joint is unstable
Grade 3	Complete tear through the ligament Extensive swelling, bruising, and severe pain Unable to bear weight; significant joint instability

Based on Stiell IG, Greenberg GH, McKnight RD, et al. Decision rules for the use of radiography in acute ankle injuries: refinement and prospective validation. *JAMA.* 1993;269(9):1127-1132.[127]

The management of sprains is determined by the severity of the injury. NSAIDs and acetaminophen are generally sufficient pain medications for these conditions; use of opioids should be avoided. The immediate implementation of a strategy to limit bruising and swelling follows the mnemonic “PRICE”: protection, rest, ice, compression, and elevation (**Table 6-25**). Progressive rehabilitation of the joint is appropriate in all cases. Long-term immobilization is less useful than an approach that encourages exercises to strengthen the joint after 2 to 3 days.[128] Only the most severe sprains require surgical repair, following unsuccessful physical therapy.[126]

Table 6-25	PRICE: A Conservative Strategy for Management of Sprains and Strains
Strategy	**Management**
Protection	Use of an air splint, fixed-joint, or adjustable walking cast
Rest	Limitation of activity involving weight bearing
Ice	Applied intermittently for up to 20 minutes each time
Compression	Elastic bandages that limit swelling and extravasation into damaged tissue
Elevation	To the level of the heart

Strains

Strains are the result of over-stretching a muscle or tendon causing a tear; the degree of risk of an injury is related to the strength and conditioning of the individual, the level of activity, and the direction and intensity of the movement involved. The actual degree of muscle tearing, the resultant bleeding into the tissue, and inflammation all factor into determining the extent of injury. Clinical diagnosis is frequently all that is necessary in case of a strain; ultrasound or magnetic resonance imaging (MRI) may occasionally be required for further evaluation.

Similar to treatment of individuals with sprains, PRICE therapies are used for the first 3 to 5 days following strains, followed by gradual reintroduction of physical activity.[128] NSAIDs provide pain relief and decrease associated inflammation, and can be used as adjunctive treatment in the acute phase of tissue injury. Long-term use of NSAIDs may be associated with poorer tissue healing.[129]

Low Back Pain

Pain in the lower back, with or without sciatic nerve pain, is divided into acute pain lasting less than 7 weeks and chronic pain. The rate of low back pain in women has been reported as 30%.[130] Approximately 25% of all individuals who experience an episode of acute, nonspecific low back pain will have a recurrence within one year.[131] Risk factors include female gender, increasing age, obesity, smoking, psychological factors, and physically intensive work.[132] The differential diagnoses range from sprain/strain to spinal conditions that include herniated discs, compression fractures, spinal stenosis; systemic diseases including cancer and connective tissue disease; and referred pain from pelvic or abdominal disease.[133]

Assessment of the woman presenting with symptoms consistent with acute lower back pain includes history and related symptoms, her subjective evaluation of the pain, associated psychological factors, ability to function normally, environmental or work-related risks, prior episodes, and questioning regarding red-flag symptoms of more serious disorders. Red-flag signs and symptoms include history of trauma, parenteral drug use, unexplained weight loss, cancer history, long-term use of steroids, fever, incontinence, and intense localized pain on physical examination.

Clinical assessment does not require imaging for acute, episodic low back pain.[134] Instead,

MRI is required only for those individuals with progressive neurologic deficit. The assessment should allow for differentiation of pain into nonspecific causes, possible radiculopathy or stenosis, and other risks including progressive neurologic disease.[134]

Initial management of acute low back pain includes health education about exercise, encouragement to remain physically active, nonpharmacologic measures, and pharmacologic management. Heat may offer relief from strains. Bed rest or prolonged immobilization is of no additional benefit in caring for women with acute low back pain.[134,135]

Pharmacologic therapy for acute low back pain begins with NSAIDs or acetaminophen (Tylenol). Short-term use (fewer than 4 days) of a muscle relaxant can assist when acute pain is severe. No strong evidence exists to recommend one agent over the others, although cyclobenzaprine (Flexeril) is commonly recommended.[134,135] While opiate medications can be effective pain relievers, their benefit in treating low back pain is often outweighed by their potential for abuse. Prolonged use of opiates occurs frequently and is associated with higher rates of adverse outcomes, including slower recovery, addiction, overdose, and accidental death.[136] Among women with chronic low back pain, tricyclic antidepressants have been shown to offer some benefit.

Nonpharmacologic therapies have also shown some benefit in the treatment of individuals with chronic low back pain. Cognitive-behavioral therapy, spinal manipulation, acupuncture, massage, and yoga, as well as interdisciplinary approaches, have all demonstrated moderate to significant benefits.[134,135]

Dermatologic Conditions

When assessing a skin lesion or rash, a history specific to the current concern, including the pattern of recurrence, timing and progression, and potential environmental exposures, will facilitate identification of the dermatologic condition. The physical examination includes assessment of the appearance, shape, and texture of the lesion; the pattern of multiple lesions; and distribution of lesions across the body. Knowing the terminology for common skin changes standardizes descriptions and facilitates identification; such terms are identified in **Table 6-26**. Several of the more common or significant conditions found during the delivery of primary care to women are described in this section.

Table 6-26 Terms and Definitions for Common Skin Manifestations

Term	Definition
Acne	A dermatologic condition primarily of the sebaceous skin glands characterized by papules, pustules, or comedones. This condition is more properly termed acne vulgaris to differentiate it from other, less common types of acne.
Actinic keratosis (AK)	A premalignant skin condition with multiple crusty patches. Also termed solar keratosis.
Calcineurin inhibitors	Immunosuppressant agents that are theorized to act by selectively inhibiting inflammation through action on T-cell activation.
Carbuncle	A skin infection composed of a cluster of boils (furuncles). This infection is most frequently caused by *Staphylococcus aureus*.
Cellulitis	An acute spreading dermatologic bacterial infection characterized by significant edema, erythema, and pain.
Cosmeceutical	A word coined from the terms “cosmetic” and “pharmaceutical,” which is used to describe agents that both have therapeutic effects and promote attractiveness.
Dermatophytes	Parasites that may infect the skin. An example is athlete’s foot.
Eczema	Inflammatory dermatologic process that is characterized by pruritus, erythema, and lesions that may be encrusted and scaly.
Furuncle	Skin infection commonly involving a hair follicle. Also called a boil.
Humectant	Agent that absorbs water and promotes maintenance of moisture on the skin.
Intertrigo	Rash or inflammation of the body folds or intertriginous areas of the skin.
Keratinocyte	Epidermal cell that synthesizes keratin.
Langerhans cell	Dendritic skin cell that transports antigens to lymph nodes.
Melanin	Skin pigment produced by special cells (melanocytes).
Melanocyte	Cell located in the bottom layer (stratum basale) of the skin’s epidermis that produces melanin.
Merkel cell	Cell found in the middle layers of the skin around hair follicles. Cancer originating among these cells—Merkel cell carcinoma—tends to be highly aggressive.
Onychomycosis	Fungal infection of the nails on either the fingers or the toes.
Psoriasis	Skin condition caused by overgrowth of keratinocytes, resulting in patchy thickened skin.
Pyoderma	Skin condition characterized by purulent filled lesions.
Retinoids	Natural or synthetic derivative of vitamin A that is widely used in pharmacotherapeutics in dermatology.
Seborrheic	Wart-like, benign skin lesions.

keratoses	
Solar lentigo	Flat pigmented lesions on sun-exposed skin. Also known as senile lentigo.
Sun protection factor (SPF)	A measure of the degree to which a sunscreen provides protection from the sun's ultraviolet rays. A sunscreen with an SPF factor of 15 or greater is recommended by the American Academy of Dermatology.
Telangiectasia	Thread-like red lines on the skin, often seen in clusters and referred to as "spider veins." Thought to be caused primarily by exposure to the sun and extreme temperatures.
Xerosis	Dry skin.

Atopic Dermatitis (Eczema)

Initial signs of atopic dermatitis most often occur during infancy, and develop progressively throughout the years into adulthood. Much of the data on the prevalence of atopic dermatitis and eczema are based on pediatric data. However, a survey of adults found that more than 10% reported symptoms consistent with eczema and 6% met the more stringent definition of atopic dermatitis.[137] Living in urban areas, black race, and higher educational status among adults in the household all contribute to increased risk; higher family income is inversely associated with risk.[137,138]

Both genetic and environmental factors contribute to the development of symptomatic disease. Among the environmental triggers are aeroallergens such as pets and dust mites, and food sensitization, particularly to milk or eggs. Skin bacteria (such as *S. aureus*) and fungi (including *Malassezia* species) are also involved in development of atopic dermatitis.[139] Hypersensitivity to a variety of irritants, including chemicals, rough fabrics such as wool, humidity, or temperature change, helps account for the pruritus associated with atopic dermatitis. Finally, increased stress is associated with worsening symptoms of atopic dermatitis.

Management of atopic dermatitis consists of the application of emollient creams, possible use of moderate-strength topical corticosteroids, or topical calcineurin inhibitors (tacrolimus [Protopic], pimecrolimus [Elidel]). When secondary bacterial infections occur, topical antibiotic treatment for short periods may be recommended; long-term use of topical antibiotics may lead to resistance and should be avoided. In addition to medication, education about maintenance use of emollients, avoidance of triggers, and stress reduction are important components of care. Referral to a dermatologist for development of a long-term plan of care often is indicated.

Psoriasis

Psoriasis is a chronic skin disorder caused by an underlying immune defect that produces thick pruritic, scaly plaques on the skin. Commonly the head and neck, elbow and knee joints, and lower back are affected. Although lesions can occur anywhere on the body, the extremities are more often affected than the trunk. The prevalence of psoriasis in the United States is reported to be approximately 2% to 3% of the population, with the incidence of new cases peaking among young adults.[140] Among the various expressions of psoriasis, plaque psoriasis accounts

for almost 90% of all cases. In this form, well-defined areas of thickened, erythematous skin with silvery, scaly patches are seen, usually with symmetric distribution.

Women in whom psoriasis is suspected should be referred to a dermatologist for evaluation and plan of care. For women whose plaques cover less than 5% of the body surface, topical therapy may be sufficient, although this treatment rarely is associated with complete remission. Initial topical treatments include corticosteroids, salicylic acid (to soften keratinized skin), and moisturizers. Ultraviolet light therapy, retinoids, methotrexate (Trexall), cyclosporine-A (Neoral, Sandimmune), and biologic agents are used both for more severe cases and for more complete healing.[141]

Psoriasis is a complex condition that may remit, but rarely resolves. It has been associated with a number of comorbidities, including metabolic syndrome, cardiovascular disease, and autoimmune disorders. Associated psychological conditions include depression and suicidal ideation.[141] Ongoing assessment of depression status and stressors is an important part of providing care for women who have psoriasis. Counseling can include use of stress reduction techniques and comfort measures such as oatmeal baths.

Skin Cancers

Unprotected exposure to ultraviolet radiation is directly related to development of malignant melanomas and other skin cancers, as well as precursor lesions such as actinic keratosis, squamous cell carcinoma, and basal cell carcinoma. Exposure may occur via natural sun or a tanning facility. All of the skin cancers share the risk factors of fair skin with a tendency to freckle or burn, blond/red hair, blue or green eyes, history of severe sunburns, immunosuppression, and family history. Each year, more than 3.3 million persons in the United States are estimated to be diagnosed with a non-melanotic skin cancer.[142] Prevention of and screening for skin cancers is addressed in detail in the *Health Promotion Across the Lifespan* chapter.

Several types of skin cancer are discussed here to provide midwives with an understanding of abnormal findings on examination of the skin. The diagnosis and management of skin cancers is beyond the scope of practice for midwives. All women with suspicious skin lesions should be referred to a dermatologist for further evaluation and treatment.

Actinic Keratosis

Actinic keratoses are small, dry, rough textured papules or plaques caused by excess exposure to ultraviolet (UV) radiation. In the United States, as much as 25% of the population may develop actinic keratoses over a lifetime, with those older than 40 years being at increased risk. Early actinic keratosis lesions are usually smaller than 2 cm; they may remain the color of the underlying skin, or be gray or pink. Over time, actinic keratoses may progress to a harder, warty texture. There is disagreement about whether actinic keratosis is itself a precancerous lesion or a squamous carcinoma in situ.[143]

Basal Cell Carcinoma/Squamous Cell Carcinoma

Basal cell carcinomas have a waxy or translucent appearance with a raised edge and central erosion. Risk factors for basal cell carcinoma include skin injuries such as old burns or scars, inflammation, radiation therapy, arsenic exposure, and skin ulcers.[142] Like actinic keratoses, squamous cell carcinoma is scaly, but the lesions will be thickened, poorly defined, occasionally hard (horn-like), on an erythematous base. In addition to the specific risk factors for basal cell carcinoma cited earlier, cutaneous human papillomavirus (HPV) infection and a history of actinic keratoses are factors that increase the risk of squamous cell carcinoma.

Melanoma

The incidence of melanoma has been steadily increasing in the United States; the lifetime risk is approximately 3%, with the highest rates found among Caucasians.[144] Unlike the other skin cancers, which are relatively easy to treat and have excellent prognoses, melanoma has an increased risk of metastasis. Disease-free survival depends on early diagnosis and prompt surgical excision. Five-year survival rates for individuals diagnosed with a localized tumor are greater than 90%; by comparison, metastatic disease has a 5-year survival rate of only 15%.[145]

Melanoma is more common among women with large numbers of moles, and among women with dysplastic nevi. Dysplastic nevi are larger than common moles, with a rough texture, often irregular in outline, and of different colors. Among women, melanomas are more common on the legs and trunk. Lesions also can appear on the genital area, and they should be carefully noted for follow-up. The midwife should initiate a referral to a dermatologist for any woman with a suspicious lesion.

Common Skin Infections

Herpes Zoster

Herpes zoster, also called shingles or zoster, is a reactivation of the varicella virus (chickenpox) as immunity to the virus wanes. This virus lies dormant along one or more dermatomes, in the dorsal root of sensory nerve ganglia. Approximately 1 million cases of zoster occur in the United States annually. The incidence rises with age and in persons in whom cellular immunity is impaired. Other risk factors are unclear, but some studies report female gender as a risk factor.[146] The incidence of zoster among whites is approximately twice that among African Americans.[147] Physical trauma at the affected dermatome and psychological stress may also play a role in the reactivation of varicella virus.

Clinically, zoster often presents with a prodrome of burning pain along the affected dermatome, accompanied by headache, fatigue, and malaise. The typical painful rash of papules on an erythematous base that progresses to vesicles, ruptures, and crusts over may take 7 days to develop. The entire cycle from initial rash to complete healing takes 2 to 3 weeks.

Management of acute herpes zoster includes antiviral drugs such as acyclovir (Zovirax) or valacyclovir (Valtrex), with the addition of analgesics to treat the pain. Initially, acetaminophen (Tylenol) or an NSAID should be given, with opioids being reserved for severe or intractable pain. When postherpetic neuralgia develops, severe pain may persist for

months. Treatment of women with postherpetic neuralgia is complicated and requires referral to a pain specialist.

Transmission from direct contact with lesions is possible, but such contact will produce chickenpox, not herpes zoster. Only those persons who have not been vaccinated or who have not experienced prior varicella infection are at risk for chickenpox. Unlike with chickenpox, droplet transmission does not occur with zoster. Vaccination against herpes zoster is now recommended for adults age 60 years or older.[147] Such vaccination reduces the likelihood of ever having an outbreak by more than 50%, and is described in detail in the *Health Promotion Across the Lifespan* chapter.

Herpes Simplex Labialis

The herpes simplex labialis virus (HSV-1) is most commonly transmitted nonsexually and during childhood. Many cases of HSV-1 infection are subclinical, diagnosed only by serology; many individuals do not realize that their cold sore is, in fact, a herpes lesion.[148] Direct exposure of mucous membranes or abraded skin to viral shedding is required for transmission of HSV-1. However, the virus can be shed asymptomatically and can be transmitted by droplet or persist on skin or objects for brief periods of time.

After initial exposure, the development of symptoms depends on the inoculum—that is, the amount of virus to which the individual was exposed—and may occur up to 3 weeks after exposure. The first HSV-1 outbreak, which is typically the most widespread and painful, lasts for 10 to 14 days. Clusters of 1-mm to 2-mm painful vesicles develop on an erythematous base and open to create ulcerative lesions. Lymphadenopathy and flu-like symptoms often accompany a primary outbreak. Pain may be severe enough to lead to avoidance of food and fluids, thereby producing dehydration.[149]

Herpes viruses enter a latent state in the nerve ganglia after the initial infection. Recurrent outbreaks are typically briefer and milder, moving through the stages of prodrome, lesion formation, ulceration, and crusting within 3 to 4 days. Complete skin healing can take an additional week.[150] In addition to causing oral lesions, HSV-1 can infect other locations including the eye (herpes keratitis), fingers (herpetic whitlow), and genital region.[149,151]

Management of primary oral lesions includes acyclovir (Zovirax), valacyclovir (Valtrex), or famciclovir (Famvir) for 7 days; the use of a topical anesthetic; and fluid/nutritional supplementation when needed.[150,152] Promptly providing oral antiviral treatment for recurrent lesions can reduce shedding and duration of lesions; topical products, however, have not been shown to have the same benefit. Single-day therapy can be prescribed in the form of either valacyclovir (2 g twice in 24 hours) or famciclovir (1500 mg once); acyclovir requires multiple doses over 5 days, but can be used if other medications are not accessible. Suppression therapy with acyclovir 400 mg twice daily, valacyclovir 500 mg or 1 g daily, or famciclovir 500 mg twice daily can be provided if the woman experiences frequent recurrences.[150,152]

Tinea Versicolor (Pityriasis)

Tinea versicolor is a chronic infection, in which normal skin fungi that are present on the outer layer of the epidermis, in hair follicles and sebaceous glands, overgrow and become pathogenic. Factors that increase the risk for outbreaks include heat and humidity; oily skin; other conditions including steroid therapy, immune suppression, malnutrition, burns, adrenalectomy, and Cushing's disease; and oral contraceptive use.

The associated rash consists of flat macules of discolored skin on the neck, upper back, or chest. Light-skinned individuals will notice hyperpigmented lesions, while those with darker skin may notice either hypopigmented or hyperpigmented lesions. The macules spread to form large patches, and pruritus may be associated with outbreaks.

Topical antifungals, including clotrimazole (Lotrimin, Mycelex) and ketoconazole (Nizoral), can be applied daily for 2 to 4 weeks. Ketoconazole shampoo (Nizoral) can also be used daily for 3 days as a body wash from the scalp to the hips. When the spread of the rash is extensive or treatment with topical medications proves ineffective, a course of oral antifungals can be prescribed. The affected woman should be advised that the presence of tinea versicolor is not secondary to poor hygiene, and that she needs to continue the prescribed treatment even if lesions resolve during therapy. She also may have areas of hypopigmentation when tanning until the melanocytes destroyed by the fungus have become reestablished.

Bacterial Skin Infections

Bacterial skin infections are a common reason for healthcare visits. Knowledge of the presentation of the most common bacterial skin infections informs proper management of these conditions. Among the normal skin flora, *Staphylococcus epidermis* and *Staphylococcus aureus* are the most common bacteria, along with the corynebacteria and mycobacteria; *Streptococcus pyogenes* is less common. The most significant pathogens within this group are *S. aureus* and *S. pyogenes* (group A beta-hemolytic *Streptococcus*). Risk factors for the development of cellulitis and other soft-tissue infections include trauma, bite injuries, prior cellulitis, diabetes, comorbidities that damage the venous or lymphatic systems, chronic renal disease, cirrhosis, and intravenous drug use.

Impetigo

Impetigo is a superficial infection that produces small vesicles on an erythematous base that open and crust; these vesicles are most often found on the mouth, nares, and arms. If left untreated, the infection may spread and can persist for weeks. Mupirocin (Bactroban) ointment applied three times daily is first-line therapy for individuals with small numbers of lesions. Oral antibiotics should be used for resistant or widespread infection.[153]

Erysipelas

The most likely source of erysipelas is infection with *S. pyogenes*. This infection most often presents as a superficial infection of the skin producing a painful erythematous plaque with well-defined edges. It is treated similarly to cellulitis.[153]

Cellulitis

Penetration of fluid into subcutaneous tissue, causing erythema, warmth, edema, pain, and possibly lymphadenopathy, is the hallmark of cellulitis. An elevated white blood cell count and fever may be present. When ulcers or other open lesions are present, *S. aureus* infection is the likely culprit. By comparison, diffuse infection occurs more commonly when *S. pyogenes* is the pathogen present.

Both erysipelas and cellulitis usually are treated orally with penicillinase-resistant semi-synthetic penicillin (e.g., dicloxacillin [Dynapen]), cephalosporin (e.g., cephalexin [Keflex]), or clindamycin (Cleocin). When systemic symptoms are present, infections require parenteral therapy.[153] When purulent cellulitis is present, treatment should include an antibiotic effective against methicillin-resistant *S. aureus* (MRSA). When cellulitis or erysipelas do not resolve with antibiotic treatment, the woman should be referred to a specialist for further assessment.

Purulent Soft-Tissue Infections

Conditions such as furuncles and carbuncles are most commonly found in areas of the body where restrictive clothing is worn or chafing occurs. A small, firm, red papule will enlarge and become painful as pus increases inside, then may either resolve or open and drain spontaneously. When several furuncles coalesce into a carbuncle, the affected individual may experience systemic symptoms of fever or chills in addition to the local pain. Carbuncles will become fluctuant, thinning the skin above the site and drain, possibly from several openings. Treatment of women with sporadic lesions can be accomplished with warm soaks to help open and drain the site. More severe lesions may require incision and drainage. Treatment with trimethoprim–sulfamethoxazole, clindamycin, or doxycycline is appropriate.[154]

Necrotizing Soft-Tissue Infection

Necrotizing fasciitis is a soft-tissue infection caused by secondary or polymicrobial extension of a surface infection, often involving mixed anaerobes or group A *Streptococcus*. The infection encompasses the subcutaneous tissue and extends to the muscle layer. In 80% of cases, there is a direct relationship to a skin lesion.[153] To distinguish cellulitis from a necrotizing infection, the following signs and symptoms can be identified in necrotizing infections: severe pain, development of bullae, skin bruising, rapid progression to systemic symptoms, edema, surface anesthesia, and a wooden or rigid texture to the underlying tissue.[153] Any suspicion of deep-tissue infection associated with a skin lesion should result in an immediate referral of the woman to a physician, urgent care, or an emergency department to be evaluated for antibiotic and possible surgical treatment.

Methicillin-Resistant Staphylococcus aureus

S. aureus is one of a group of common bacteria—including *Enterococcus*, *E. coli*, and *Neisseria gonorrhoeae*—that have developed increasing resistance to the traditional antibiotic therapies. More than half of all serious skin and soft-tissue infections are now caused by

MRSA.[155] In office settings, MRSA is most commonly seen as boils or carbuncles. These lesions begin with a “spider bite” appearance before enlarging and becoming painful.

The Infectious Disease Society of America’s guidelines continue to recommend incision and drainage of boils as the most appropriate treatment. Its recommendation is to defer antibiotics unless the individual has cellulitis or severe disease with multiple locations. If MRSA is suspected, antibiotics that offer adequate coverage, including TMP-SMX (Bactrim), clindamycin (Cleocin), a tetracycline, or linezolid (Zyvox), should be prescribed for 5 to 10 days, and a culture taken prior to treatment.[155] Regardless of the midwife’s choice to use supportive measures, incise the lesion for drainage, or prescribe antibiotics, women should be advised that signs of worsening infection or failure to begin healing are indications to seek further care.

Substance Use: Screening and Counseling

Screening for alcohol, tobacco, and substance use can be an uncomfortable process. As with questions about sexuality or abuse, some positive answers may lead to a discussion the midwife feels poorly prepared to address.

All women should be screened for tobacco use and alcohol misuse; counseling interventions should be provided for women who use tobacco and for whom alcohol misuse is suspected. A detailed discussion of screening for these conditions, including recommended screening tools, is provided in the *Health Promotion Across the Lifespan* chapter. Counseling interventions aimed at smoking cessation and reducing alcohol misuse are also provided in that chapter.

Screening for Substance Use

Screening for illicit or prescription drug overuse is not currently recommended as a standard screen for all adults in the United States by some organizations, but is recommended by others. Screening may be particularly important in some geographic locales. Screening for illicit drug use can be performed with any of several validated tools. However, legal and social complexities abound. Who should be screened? At what intervals? For which indications? If medical marijuana (or recreational marijuana) is legal in the state, how does that affect counseling about the risks? What about women with chronic pain? All of these issues should be considered and clearly described, both in practice guidelines and to women coming to the practice. Consent for drug testing should always be obtained.

Two areas of specific concern for every midwife are marijuana and opioid use: marijuana because it is transitioning from an illicit drug to a legal one, and opioids because of the epidemic at the intersection of heroin and prescription abuse. If screening is performed, it should be performed for all individuals. The National Institute of Drug Abuse (NIDA) quick screen starts with asking, “In the past year, how often have you used [fill in substance]?” The answer then leads the provider to follow-up questions and responses. The NIDA screen can be found in the Resources section at the end of this chapter.

Marijuana

Marijuana is the most frequently used illicit drug in the United States, as well as being a legal medication in an increasing number of jurisdictions and being legal for recreational use in a few states. Like alcohol use, marijuana use carries health risks as well as benefits. Those risks include development of substance use disorder, increased risk of psychosis, respiratory disorders, cardiovascular disease, and both short-term and cumulative declines in memory and attention.[156–158] Moreover, at least 1 in 10 marijuana users will develop addictive behavior.[157] Assessing women for use or misuse of marijuana can be done similarly to the way in which alcohol misuse is evaluated.

Opioid Use

Overall, 6 of 10 drug overdoses in the United States are now opioid related.[159] Moreover,

81% of cocaine overdose fatalities have opioids as a contributing factor.[160]

Even without the risk of overdose, prescription opioid use has both significant economic and health costs. These include the direct costs of excess prescriptions, lost days of work, and drug-related comorbidities.[161] A number of proposals have been made, and some states have enacted laws, to help reduce the risk of such use. Most states have implemented changes in health policy in an attempt to address the opioid epidemic, including mandating provider participation in prescription drug monitoring programs, making naloxone widely available, and limiting the way opioid prescriptions are written and filled. Professional organizations have also published guidelines that promote the use of NSAIDs and other non-opioid options for managing moderate pain; these measures are intended to decrease the risk of promoting dependency.[159,162] The Substance Abuse and Mental Health Services Administration (SAMHSA) provides an opioid overdose prevention toolkit with recommendations for prescribers and individuals.[163]

One contributor to the opioid epidemic is healthcare provider diversion—that is, the diversion of opioids to patients through the inappropriate provision of opioid prescriptions.[164] Halting inappropriate opioid-prescribing practices is an important first step in the fight against opioid use. Steps that individual prescribers can take include participating in prescription drug monitoring programs, limiting initial opioid prescriptions to immediate-release medications in the lowest dose, and prescribing the smallest number of doses that are consistent with pain guidelines and the anticipated need.[162] Seven-day limits for initial opioid prescriptions for acute pain should meet most individual requirements. Non-opioid alternatives and nonpharmacologic approaches to pain management should be provided and encouraged, along with health education about risks and benefits for each. When requests for opioid refills are made, the woman should be evaluated, as most midwives will not manage chronic pain conditions.

Conclusion

Modern midwifery has often been viewed as possessing a limited, albeit well-recognized, scope of practice—that is, midwives take care of healthy pregnant women and help them give birth. Historically, however, midwives have never limited their care exclusively to healthy pregnant women, if for no other reason than that many of the women traditionally served by midwives were at risk because of age, socioeconomic status, or lack of access to other healthcare resources. In addition, midwives have often provided first-line care for women and sometimes larger populations. As has always been true, the needs of the community and the skills of the midwives who serve that community help define midwifery scope of practice. Today midwifery is practiced in a healthcare system in which the only provider whom many women see is their "women's health" provider. While midwives still care for pregnant women, they are also obliged to correctly identify common conditions, minor and major, and facilitate treatment of women with these conditions by themselves or through other strategies such as collaborative management and referral.

Midwives must be able to describe abnormal or unusual symptoms or signs accurately, even when they cannot make a diagnosis with any certainty. These professionals must be prepared to assess and triage women with these conditions, and determine which can be managed autonomously by the midwife and which cannot. All midwives need to know to whom they will refer cases beyond their personal or professional scopes of practice. Referral can be to nurse practitioners or physician assistants, as well as to physicians, dieticians, social workers, physical therapists, and others. Creation of a large network of healthcare contacts serves both midwives and women well.

The astute reader may have noticed the frequency with which reference has been made to lifestyle changes that can help prevent or treat diseases. One of the assets that midwives bring to primary care for women is an understanding of the importance of health maintenance and disease prevention. Every midwife has the ability and responsibility to provide this aspect of primary care for all women.

Finally, the importance of combining an evidence-based approach with a holistic awareness of the various factors playing into the woman's life is increasingly recognized as positively influencing an individual's ability to adhere to treatment recommendations, whether these recommendations focus on lifestyle changes, medication, or other therapeutic interventions. The provision of effective health care requires an awareness and assessment of how poverty, insurance coverage, literacy, mental health, and other factors affect a woman's ability to achieve the goals she has set with the midwife from whom she obtains care.

Resources

Organization	Description	Webpage
Online Calculators for Atherosclerotic Cardiovascular Disease		
American College of Cardiology (ACC)	10-year calculator for atherosclerotic cardiovascular disease. This risk calculator can also be downloaded from iTunes as an app.	http://tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate/
Framingham Risk Assessment Tool	Based on the Framingham Heart Study	https://www.framinghamheartstudy.org/risk-functions/cardiovascular-disease/10-year-risk.php
Multi-Ethnic Study of Atherosclerosis (MESA)	Coronary artery calcification calculator	https://www.mesa-nhlbi.org/CAC-Tools.aspx
Reynolds Risk Score	This calculator is particularly sensitive for women in assessing risk for coronary event	http://www.reynoldsriskscore.org
Diabetes		
American Association of Clinical Endocrinologists and American College of Endocrinology (AACE/ACE)	Diabetes treatment algorithms	https://www.aace.com/files/aace_algorithm.pdf
Indian Health Service (IHS)	Multiple diabetes treatment guidelines and algorithms based on the American Diabetes Association Guidelines	https://www.ihs.gov/diabetes/clinician-resources/soc/
Hypertension		
American College of Cardiology (ACC)	*High Blood Pressure in Adults: Guideline for the Prevention, Detection, Evaluation and Management* 2017 American Heart Association and American College of Cardiology Hypertension guidelines. This site has multiple links to the executive summary, slides, patient handouts, and key points for clinicians.	http://www.acc.org/guidelines/hubs/high-blood-pressure
National Heart, Lung, and Blood Institute (NHLBI)	JNC 7 reference card and link to complete guideline	https://www.nhlbi.nih.gov/health-pro/guidelines/current/hypertension-jnc-7/reference-card
American Society of Hypertension (ASH)	Website lists different professional association hypertension guidelines	http://www.ash-us.org/About-Hypertension/Hypertension-Guidelines.aspx
Migraines		
Migraine Research Foundation	Migraine resources and guidelines	http://migraineresearchfoundation.org/resources/resources-and-links/

Tuberculosis		
Centers for Disease Control and Prevention (CDC)	Core Curriculum on Tuberculosis: What the Clinician Should Know	https://www.cdc.gov/tb/education/corecurr/index.htm

References

1. American College of Nurse-Midwives. Position statement: midwives are primary care providers and leaders of maternity care homes. 2012. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000273/Primary%20Care%20P Accessed October 1, 2017.
2. Donaldson MS, Yordy KD, Lohr KN, Vanselow NA, eds. *Primary Care: America's Health in a New Era*. Washingtonm DC: National Academy Press; 1996.
3. Linde-Feucht S, Coulouris N. Integrating primary care and public health: a strategic priority. *Am J Public Health* 2012;102(S3):S310-S311.
4. Bunn HF. Overview of the anemias. In: Aster JC, Bunn HF, eds. *Pathophysiology of Blood Disorders*. 2nd ed. New York, NY: McGraw-Hill Education; 2017:32-46.
5. Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. *MMWR Recomm Rep*. 1998;47(RR-3):1-36.
6. U.S. Preventive Services Task Force. Final recommendation statement: iron deficiency anemia in pregnant women: screening and supplementation. 2015. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/iron-deficiency-anemia-in-pregnant-women-screening-and-supplementation. Accessed March 13, 2017.
7. National Institutes of Health. Iron: dietary supplement fact sheet. 2016. Available at: https://ods.od.nih.gov/factsheets/Iron-HealthProfessional/#h2. Accessed September 29, 2017.
8. Rund D, Rachmielewitz E. β-Thalassemia. *N Engl J Med*. 2005;353:1135-1146.
9. Sayani FA, Kwiatkowski JL. Increasing prevalence of thalassemia in America: implications for primary care. *Ann Med*. 2015;47(7):592-604.
10. Muncie H, Campbell J. Alpha and beta thalassemia. *Am Fam Physician*. 2009;80(4):339-344.
11. Olivieri N. The β-thalassemias. *N Engl J Med*. 1999;341:99-109.
12. Centers for Disease Control and Prevention. Incidence of sickle cell trait—United States, 2010. *MMWR*. 2014;63(49):1155-1158.
13. Centers for Disease Control and Prevention. Sickle cell disease: data & statistics. 2016. Available at: https://www.cdc.gov/ncbddd/sicklecell/data.html. Accessed March 13, 2017.
14. Paulukonis S, Eckman J, Snyder A, et al. Defining sickle cell disease mortality using a population-based surveillance system, 2004–2008. *Public Health Rep*. 2016;131:367-375.
15. Dilley A, Drews C, Miller C, et al. Von Willebrand disease and other inherited bleeding disorders in women with diagnosed menorrhagia. *Obstet Gynecol*. 2001;97:630-636.
16. National Heart, Lung, and Blood Institute. *The Diagnosis, Evaluation and Management of von Willebrand Disease*. NIH Publication No. 08-5832. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute; 2007.
17. Centers for Disease Control and Prevention. Leading causes of death in females, 2014. 2017. Available at: https://www.cdc.gov/women/lcod/2014/race-ethnicity/index.htm. Accessed March 13, 2017.
18. Hing E, Rui P, Palso K. National Ambulatory Medical Care Survey: 2013 state and national summary tables: tables 1, 11, 16. 2013. Available at: https://www.cdc.gov/nchs/data/ahcd/namcs_summary/2013_namcs_web_tables.pdf. Accessed March 13, 2017.
19. Yoon S, Burt V, Louis T, Carroll M. *Hypertension Among Adults in the United States, 2009–2010*. NCHS Data Brief No. 107. Hyattsville, MD: National Center for Health Statistics; 2012.
20. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2017. [Epub ahead of print]. doi:10.1016/j.jacc.2017.11.006.
21. Gillespie C, Hurvitz K. Prevalence of hypertension and controlled hypertension—United States, 2007–2010. *MMWR*. 2013;62(03):144-148.
22. Weber MA, Schiffrin EL, White WB, et al. Clinical practice guidelines for the management of hypertension in the community: a statement by the American Society of Hypertension and the International Society of Hypertension. *J Clin Hypertens*. 2014;16(1):14-26.
23. James PA, Oparil S, Carter BL, et al. Evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*. 2014;311(5):507-520.
24. National High Blood Pressure Education Program. *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: Complete Report*. Bethesda, MD: National Heart, Lung, and Blood Institute; August 2004.

25. Appel L, Champagne C, Harsha D, et al. Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. Writing Group of the PREMIER Collaborative Research Group. *JAMA*. 2003;289:2083-2093.
26. Wright J, Musini V. First-line drugs for hypertension. *Cochrane Database Syst Rev*. 2009;3:CD001841. doi:101002/14651858CD001841pub2.
27. Law M, Morris J, Wald N. Use of blood pressure lowering drugs in the prevention of cardiovascular disease: meta-analysis of 147 randomised trials in the context of expectations from prospective epidemiological studies. *BMJ*. 2009;338:b1665.
28. Jellinger PS, Handelsman Y, Rosenblit PD, et al. American Association of Clinical Endocrinologists and American College of Endocrinology guidelines for management of dyslipidemia and prevention of cardiovascular disease. *Endocr Pract*. 2017;23(suppl 2):1-87.
29. American Academy of Family Physicians. Summary of recommendations for clinical preventive services. 2017. Available at: http://www.aafp.org/dam/AAFP/documents/patient_care/clinical_recommendations/cps-recommendations.pdf. Accessed March 13, 2017.
30. American College of Obstetricians and Gynecologists. Annual women's health care, well woman recommendations, ages 40–64 years: laboratory and other tests. Available at: http://www.acog.org/About-ACOG/ACOG-Departments/Annual-Womens-Health-Care/Well-Woman-Recommendations/Laboratory-Testing-Ages-40-64-Years. Accessed March 13, 2017.
31. American College of Cardiology, American Heart Association. ASCVD risk calculator. Available at: http://tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate/. Accessed April 29, 2017.
32. Stone NJ, Robinson JG, Lichtenstein AH, et al. ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;63:2889-2934.
33. Grijalva C, Nuorti J, Grigffin M. Antibiotic prescription rates for acute respiratory tract infections in US ambulatory settings. *JAMA*. 2009;302(7):758-766.
34. Aliberti S, Kaye K. The changing microbiologic epidemiology of community-acquired pneumonia. *Postgrad Med*. 2013;125(6):31-42.
35. Chow A, Benninger M, Brook I, Brozek J, Goldstein E, Hicks L. IDSA clinical practice guideline for acute bacterial rhinosinusitis in children and adults. *Clin Infect Dis*. 2012;54(8):1041-1045.
36. Graves BW. Respiratory conditions and cardiovascular conditions. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:549-586.
37. De Sutter A, Saraswat A, van Driel M. Antihistamines for the common cold. *Cochrane Database Syst Rev*. 2015;11:CD009345. doi:10.1002/14651858.CD009345.pub2.
38. Hayward G, Thompson M, Perera R, Del Mar C, Glasziou P, Heneghan C. Corticosteroids for the common cold. *Cochrane Database Syst Rev*. 2012;8:CD008116. doi:101002/14651858CD008116pub2.
39. Meltzer EO, Hamilos DL. Rhinosinusitis diagnosis and management for the clinician: a synopsis of recent consensus guidelines. *Mayo Clin Proc*. 2011;86(5):427-443.
40. Grohskopf LA, Sokolow LZ, Broder KR, et al. Prevention and control of seasonal influenza with vaccines: recommendations of the Advisory Committee on Immunization Practices—United States, 2016–17 influenza season. *MMWR Recomm Rep*. 2016;65(RR-5):1-65.
41. Centers for Disease Control and Prevention. People at high risk of developing flu–related complications. 2016. Available at: https://www.cdc.gov/flu/about/disease/high_risk.htm. Accessed March 13, 2017.
42. Fiore A, Fry A, Shay D, Gubareva L, Bresee J, Uyeki T. Antiviral agents for the treatment and chemoprophylaxis of influenza: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recommend Rep*. 2011;60(RR01):1-24.
43. Shulman S, Bisno A, Clegg H, et al. Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Disease Society of America. *Clin Infect Dis*. 2012;55(10):e86-e102.
44. Becker L, Hom J, Villasis-Keever M, van der Wouden J. Beta2-agonist drugs for treating cough or a clinical diagnosis of acute bronchitis. *Cochrane Database Syst Rev*. 2009;3:CD001841. doi:101002/14651858CD001726pub5.
45. Albert R. Diagnosis and treatment of acute bronchitis. *Am Fam Physician*, 2010;82(11):1345-1350.
46. Braman SS. Chronic cough due to acute bronchitis: ACCP evidence-based clinical practice guidelines. *Chest*. 2006;129(1 suppl):95S-103S.
47. Afshari A, Pagani L, Harbarth S. Year in review 2011: critical care—infection. *Crit Care*. 2011;16(6):242.
48. Mandel L, Wunderink R, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis*. 2007;44(suppl 2):S27-S72.
49. Almirall J, Bolibar I, Serra-Prat M, et al. New evidence of risk factors for community-based pneumonia: a population

based study. *Euro Respir J*. 2008;31(6):1274-1284.
50. Musher DM, Thorner AR. Community-acquired pneumonia. *N Engl J Med*. 2014;371(17):1619-1628.
51. Bartlett J. Diagnostic tests for agents of community- acquired pneumonia. *Clin Infect Dis*. 2011;52(suppl 4):S296-S304.
52. Centers for Disease Control and Prevention. Pertussis (whooping cough) surveillance and reporting. 2016. Available at: https://www.cdc.gov/pertussis/images/incidence-graph.png. Accessed April 29, 2017.
53. Hamborsky J, Kroger A, Wolfe S. Pertussis. In: Hamborsky J, Kroger A, Wolfe S, eds. *Epidemiology and Prevention of Vaccine-Preventable Diseases*. 13th ed. Washington, DC: Public Health Foundation; 2015. Available at: https://www.cdc.gov/vaccines/pubs/pinkbook/pert.html. Accessed March 13, 2017.
54. Centers for Disease Control and Prevention. Postexposure antimicrobial prophylaxis. 2015. Available at: https://www.cdc.gov/pertussis/outbreaks/pep.html. Accessed March 13, 2017.
55. Centers for Disease Control and Prevention. Most recent asthma data. 2016. Available at: https://www.cdc.gov/asthma/most_recent_data.htm. Accessed March 13, 2017.
56. National Asthma Education and Prevention Program. *Expert Panel Report: Guidelines for the Diagnosis and Management of Asthma (EPR-3)*. Bethesda, MD: National Heart, Lung, and Blood Institute; 2007.
57. National Heart, Lung, and Blood Institute. Asthma care quick reference. 2012. Available at: http://www.nhlbi.nih.gov/files/docs/guidelines/asthma_qrg.pdf. Accessed March 13, 2017.
58. Centers for Disease Control and Prevention. Tuberculosis. 2017. Available at: https://www.cdc.gov/tb/. Accessed September 27, 2017.
59. Centers for Disease Control and Prevention. *Core Curriculum on Tuberculosis: What the Clinician Should Know*. 6th ed. Atlanta, GA: Centers for Disease Control and Prevention; 2013. Available at: https://www.cdc.gov/tb/education/corecurr/pdf/corecurr_all.pdf. Accessed April 29, 2017.
60. Peery A, Dello E, Lund J, Crockett S, McGowan C, Bulsiewicz W. Burden of gastrointestinal disease in the United States: 2012 update. *Gastroenterol*. 2012;143(5):1179-1187, e3.
61. Chey WD, Leontiadis GI, Howden CW, Moss SF. ACG clinical guideline: treatment of *Helicobacter pylori* infection. *Am J Gastroenterol*. 2017;112:212-238.
62. National Center for Health Statistics. *Summary Health Statistics for US Adults: National Health Interview Survey 2010*. Series 10, No. 252. Hyattsville, MD: U.S. Department of Health and Human Services; January 2012.
63. Fashner J, Gitu J. Diagnosis and treatment of peptic ulcer disease and *H. pylori* infection. *Am Fam Physician*. 2015;91(4):236-242.
64. Wilhelm S, Johnson J, Kale-Pradhan P. Treating bugs with bugs: the role of probiotics as adjunctive therapy for *Helicobacter pylori*. *Ann Pharmacother*. 2011;45(7-8):960-966.
65. Schmidt M, Dumot JA, Søreide O, Søndenaa K. Diagnosis and management of gallbladder calculus disease. *Scand J Gastroenterol*. 2012;47(11):1257-1265.
66. Buckius M, McGrath B, Monk J, Grim R, Bell T, Ahuja V. Changing epidemiology of acute appendicitis in the United States: study period 1993–2008. *J Surg Res*. 2012;175(2):185-190.
67. Wilms IMHA, de Hoog DENM, de Visser DC, Janzing HMJ. Appendectomy versus antibiotic treatment for acute appendicitis. *Cochrane Database Syst Rev*. 2011;11:CD008359. doi:10.1002/14651858.CD008359.pub2.
68. Scallan E, Hoekstra R, Angulo F, et al. Foodborne illness acquired in the United States—major pathogens. *Emerg Infect Dis*. 2011;17(1):7-16.
69. Connor BA. Travelers' diarrhea. In: *CDC Health Information for International Travel*. 2017. Available at: https://wwwnc.cdc.gov/travel/yellowbook/2018/the-pre-travel-consultation/travelers-diarrhea. Accessed October 1, 2017.
70. Higgins D, Johanson J. Epidemiology of constipation in North America: a systematic review. *Am J Gastroenterol*. 2004;99:750-759.
71. Rome III Diagnostic Criteria for Functional Gastrointestinal Disorders. Available at: http://www.romecriteria.org/assets/pdf/19_RomeIII_apA_885-898.pdf. Accessed March 13, 2017.
72. Weinberg D, Smalley W, Heidelbaugh J, Sultan S. American Gastroenterological Association Institute guideline on the pharmacological management of irritable bowel syndrome. *Gastroenterology*. 2014;147(5):1146-1148.
73. Centers for Disease Control and Prevention. Surveillance for viral hepatitis—United States, 2011. 2013. Available at: https://www.cdc.gov/hepatitis/statistics/2011surveillance/commentary.htm#background. Accessed March 13, 2017.
74. Centers for Disease Control and Prevention. Viral hepatitis. 2017. Available at: https://www.cdc.gov/hepatitis/. Accessed March 13, 2017.
75. Centers for Disease Control and Prevention. Surveillance for viral hepatitis—United States, 2014. 2016. Available at: https://www.cdc.gov/hepatitis/statistics/2014surveillance/commentary.htm#background. Accessed March 13, 2017.
76. Hamborsky J, Kroger A, Wolfe S. Hepatitis A. In: Hamborsky J, Kroger A, Wolfe S, eds. *Epidemiology and Prevention of Vaccine-Preventable Diseases*. 13th ed. Washington, DC: Public Health Foundation; 2015:135-148.
77. Centers for Disease Control and Prevention. Viral hepatitis: hepatitis C information. Available at: https://www.cdc.gov/hepatitis/hcv/index.htm. Accessed March 13, 2017.
78. American Association for the Study of Liver Diseases, Infectious Diseases Society of America. Recommendations for

testing, managing, and treating hepatitis C. 2017. Available at: http://www.hcvguidelines.org. Accessed March 13, 2017.
79. Tohme R, Holmberg SD. Transmission of hepatitis C virus infection through tattooing and piercing: a critical review. *Clin Infect Dis*. 2012;54(8):1167-1178.
80. Emmanuel B, Shardell MD, Tracy L, Kottilil S, El-Kamary SS. Racial disparity in all-cause mortality among hepatitis C virus–infected individuals in a general US population, NHANES III. *J Viral Hepat*. 2016:24(5):380-388.
81. Barber AE, Norton JP, Spivak AM, Mulvey MA. Urinary tract infections: current and emerging management strategies. *Clin Infect Dis*. 2013;57(5):719-724.
82. O'Dell KK. Pharmacologic management of asymptomatic bacteriuria and urinary tract infections in women. *J Midwifery Womens Health* 2011;56:248-265.
83. Gupta K, Hooton TM, Naber KG, Wullt B, Colgan R, Miller LG. International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: a 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases. *Clin Infect Dis*. 2011;52(5):e103-e120.
84. Foxman B. Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden. *Infect Dis Clin North Am*. 2014;28(1):1-13.
85. Nicolle LE. Uncomplicated urinary tract infection in adults including uncomplicated pyelonephritis. *Urol Clin North Am*. 2008;35(1):1-12.
86. Rahn DD, Carberry C, Sanses TV, et al. Vaginal estrogen for genitourinary syndrome of menopause: a systematic review. *Obstet Gynecol*. 2014;124(6):1147-1156.
87. Foxman B, Buxton M. Alternative approaches to conventional treatment of acute uncomplicated urinary tract infection in women. *Curr Infect Dis Rep*. 2013;15(2):124-129.
88. Barbosa-Cesnik C, Brown MB, Buxton M, et al. Cranberry juice fails to prevent recurrent urinary tract infection: results from a randomized placebo-controlled trial. *Clin Infect Dis* 2011; 52:23.
89. McLennan MT. Interstitial cystitis: epidemiology, pathophysiology, and clinical presentation. *Obstet Gynecol Clin North Am*, 2014;41(3):385-395.
90. Berry SH, Elliott MN, Suttorp M, et al. Prevalence of symptoms of bladder pain syndrome/interstitial cystitis among adult females in the United States. *J Urol*. 2011;186:540-544.
91. Morgan MSC, Pearle MS. Medical management of renal stones. *BMJ*. 2016;352:i52.
92. Hollingsworth JM, Rogers MAM, Kaufman SR, et al. Medical therapy to facilitate urinary stone passage: a meta-analysis. *Lancet*. 2006;368(9542):1171-1179.
93. Samplaski MK, Irwin BH, Desai M. Less-invasive ways to remove stones from the kidneys and ureters. *Cleveland Clin J Med*. 2009;76(10):592-598.
94. Scales CD Jr, Smith AC, Hanley JM; Project UDiA. Prevalence of kidney stones in the United States. *Eur Urol*. 2012;62:160-165.
95. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002 Dec 17;106(25):3143-3421.
96. American Diabetes Association. Standards of medical care in diabetes—2017. *Diab Care*. 2017;40(suppl 1):S1-S135.
97. Inzucchi SE, Majumdar SK. Current therapies for the medical management of diabetes. *Obstet Gynecol*. 2016;127:780-794
98. Reis JP, Loria CM, Sorlie PD, Park Y, Hollenbeck A, Schatzkin A. Lifestyle factors and risk for new-onset diabetes in a large population-based prospective cohort study. *Ann Intern Med*. 2011;155(5):292-299.
99. Helfand M. *Screening for Thyroid Disease*. Systematic Evidence Review No. 23. (Prepared by the Oregon Health & Science Evidence-based Practice Center under contract no. 290-97-0018.) Rockville, MD: Agency for Healthcare Research and Quality; 2004. Available at: https://www.ahrq.gov/downloads/pub/prevent/pdfser/thyrser.pdf. Accessed October 5, 2017.
100. U.S. Preventive Services Task Force. Final recommendation statement: thyroid dysfunction: screening. 2015. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/thyroid-dysfunction-screening. Accessed March 13, 2017.
101. Garber JR, Cobin RH, Gharib H, et al. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. *Endocr Pract*. 2012;18(6):989-1028.
102. Maizels M, Houle T. Results of screening with the brief headache screen compared with a modified ID-Migraine. *Headache*. 2008;48:385-394.
103. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). *Cephalalgia*. 2013;33:629-808.
104. Holroyd KA, Stensland M, Lipchik GL, Hill KR, O'Donnell FS, Cordingley G. Psychosocial correlates and impact of chronic tension-type headaches. *Headache*. 2000;40:3-16.

105. Diener H-C, Gold M, Hagen M. Use of a fixed combination of acetylsalicylic acid, acetaminophen and caffeine compared with acetaminophen alone in episodic tension-type headache: meta-analysis of four randomized, double-blind, placebo-controlled, crossover studies. *J Headache Pain*. 2014;15(1):76.
106. Linde K, Allais G, Brinkhaus B, et al. Acupuncture for the prevention of tension-type headache. *Cochrane Database Syst Rev*. 2016;4:CD007587. doi:10.1002/14651858.CD007587.pub2.
107. Christiansen S, Jürgens T, Klinger R. Outpatient combined group and individual cognitive-behavioral treatment for patients with migraine and tension-type headache in a routine clinical setting. *Headache*. 2015;55(8):1072-1091.
108. Verhagena AP, Damena L, Bergera MY, Passchierb J, Koesa BW. Lack of benefit for prophylactic drugs of tension-type headache in adults: a systematic review. *Fam Pract*. 2010;27(2):151-165.
109. Jackson JL, Shimeall W, Sessums L, et al. Tricyclic antidepressants and headaches: systematic review and meta-analysis. *BMJ*. 2010;341:c5222. doi:10.1136/bmj.c5222.
110. Fumal A, Schoenen J. Tension-type headache: current research and clinical management. *Lancet Neurol*. 2008;7(1):70-83.
111. Tepper SJ, Dahlöf CGH, Dowson A, et al. Prevalence and diagnosis of migraine in patients consulting their physician with a complaint of headache: data from the landmark study. *Headache*. 2004;44:856-864.
112. Burch RC, Loder S, Loder E, Smitherman TA. The prevalence and burden of migraine and severe headache in the United States: updated statistics from government health surveillance studies. *Headache*. 2015;55:21-34.
113. Stewart WF, Wood C, Reed ML, Roy J, Lipton RB. Cumulative lifetime migraine incidence in women and men. *Cephalalgia*. 2008;28(11):1170-1178.
114. Deneris A, Allen PR, Hayes EH, Latendresse G. Migraines in women: current evidence for management of episodic and chronic migraines. *J Midwifery Womens Health*. 2017;62(3):270-285.
115. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(RR-3):1-104.
116. Fisher R, Acevedo C, Wiebe S, et al. ILAE official report: a practical clinical definition of epilepsy. *Epilepsia*. 2014;55(4):475-482.
117. Samba Reddy D. Neuroendocrine aspects of catamenial epilepsy. *Horm Behav*. 2013;63(2):254-266.
118. Pack AM, Olarte LS, Morrell MJ, Flaster E, Resor SR, Shane E. Bone mineral density in an outpatient population receiving enzyme-inducing antiepileptic drugs. *Epilepsy Behav*. 2003;4(2):169.
119. Gaffield ME, Culwell KR, Lee CR. The use of hormonal contraception among women taking anticonvulsant therapy. *Contraception*. 2011;83(1):16-29.
120. Yuen AW, Thompson PJ, Flugel D, Bell GS, Sander JW. Mortality and morbidity rates are increased in people with epilepsy: is stress part of the equation? *Epilepsy Behav*. 2007;10(1):1-7.
121. Becker J, Norab DB, Gomesa I, et al. An evaluation of gender, obesity, age and diabetes mellitus as risk factors for carpal tunnel syndrome. *Clin Neurophysiol*. 2002;113:1429-1434.
122. Kozak A, Schedlbauer G, Wirth T, Euler U, Westermann C, Nienhaus A. Association between work-related biomechanical risk factors and the occurrence of carpal tunnel syndrome: an overview of systematic reviews and a meta-analysis of current research. *BMC Musculoskelet Disord*. 2015;16:231. doi:10.1186/s12891-015-0685-0.
123. Page MJ, Massy-Westropp N, O'Connor D, Pitt V. Splinting for carpal tunnel syndrome. *Cochrane Database Syst Rev*. 2012;7:CD010003.
124. Page MJ, O'Connor D, Pitt V, Massy-Westropp N. Exercise and mobilisation interventions for carpal tunnel syndrome. *Cochrane Database Syst Rev*. 2012;6:CD009899.
125. Waterman BR, Owens BD, Davey S, Zacchilli MA, Belmont PJ. The epidemiology of ankle sprains in the United States. *J Bone Joint Surg Am*. 2010;92(13):2279-2284.
126. Chan KW, Ding BC, Mrozcek KJ. Acute and chronic lateral ankle instability in the athlete. *Bull Hosp Joint Dis*. 2011;69(1):17-26.
127. Stiell IG, Greenberg GH, McKnight RD, et al. Decision rules for the use of radiography in acute ankle injuries: refinement and prospective validation. *JAMA* 1993;269(9):1127-1132.
128. Tiemstra JD. Update on acute ankle sprains. *Am Fam Physician*. 2012;85(12):1170-1176.
129. Schoenfeld BJ. The use of nonsteroidal anti-inflammatory drugs for exercise-induced muscle damage implications for skeletal muscle development. *Sports Med*. 2012;42(12):1017-1028.
130. National Center for Health Statistics. Health, United States, 2015. 2016. Available at: http://www.cdc.gov/nchs/hus.htm. Accessed March 13, 2017.
131. Stanton TR, Henschke N, Maher CG, Refshauge KM, Latimer J, McAuley JH. After an episode of acute low back pain, recurrence is unpredictable and not as common as previously thought. *Spine*. 2008;33(26):2923-2928.
132. Golob AL, Wipf JE. Low back pain. *Med Clin North Am*. 2014;98(3):405-428.
133. Casazza BA. Diagnosis and treatment of acute low back pain. *Am Fam Physician*. 2012;85(4):343-350.
134. Goertz M, Thorson D, Bonsell J, et al. *Adult Acute and Subacute Low Back Pain*. Bloomington, MN: Institute for Clinical Systems Improvement; 2012.

135. Qaseem A, Wilt TJ, McLean RM, Forciea MA, for the Clinical Guidelines Committee of the American College of Physicians. Noninvasive treatments for acute, subacute, and chronic low back pain: a clinical practice guideline from the American College of Physicians. *Ann Intern Med*. February 14, 2017. [Epub ahead of print]. doi:10.7326/M16-2367.
136. Cifuentes M, Webster B, Genevay S, Pransky G. The course of opioid prescribing for a new episode of disabling low back pain: opioid features and dose escalation. *Pain*. 2010;151(1):22-29.
137. Hanifin JM, Reed ML; Eczema Prevalence and Impact Working Group. A population-based survey of eczema prevalence in the United States. *Dermatitis*. 007;18(2):82-91.
138. Shaw TE, Currie GP, Koudelka CW, Simpson EL. Eczema prevalence in the United States: data from the 2003 National Survey of Children's Health. *J Invest Dermatol*. 2011;131(1):67-73.
139. Wollenberg A, Schnapp C. Evolution of conventional therapy in atopic dermatitis. *Immunol Allergy Clin North Am*. 2010;30(3):351-368.
140. Menter A, Gottlieb A, Feldman SR, et al. Guidelines for the management of psoriasis and psoriatic arthritis. Section 1. Overview of psoriasis and guidelines of care for the treatment of psoriasis with biologics. *J Am Acad Dermatol*. 2008;5:826-850.
141. Menter A, Korman NJ, Elmets CA, et al. American Academy of Dermatology guidelines of care for the management of psoriasis and psoriatic arthritis. Section 3. Guidelines of care for the management and treatment of psoriasis with topical therapies. *J Am Acad Dermatol*. 2009;60:643-659.
142. American Cancer Society. Basal and squamous cell skin cancers. 2017. Available at: https://www.cancer.org/cancer/basal-and-squamous-cell-skin-cancer.html. Accessed March 13, 2017.
143. Rosen T, Lebwohl MG. Prevalence and awareness of actinic keratosis: barriers and opportunities. *J Am Acad Dermatol*. 2013;68:S2-S9.
144. American Cancer Society. Cancer Statistics Center. 2017. Available at: https://cancerstatisticscenter.cancer.org/#/. Accessed March 13, 2017.
145. Little EG, Eide MJ. Update on the current state of melanoma incidence. *Dermatol Clin*. 2012;30(3):355-361.
146. Thomas SL, Hall AJ. What does epidemiology tell us about risk factors for herpes zoster? *Lancet Infect Dis*. 2004;4(1):26-33.
147. Tseng HF, Smith N, Harpaz R, et al. Herpes zoster vaccine in older adults and the risk of subsequent herpes zoster disease. *JAMA*. 2011;305(2):160-166.
148. Fatahzadeh M, Schwartz RA. Human herpes simplex virus infections: epidemiology, pathogenesis, symptomatology, diagnosis, and management. *J Am Acad Dermatol*. 2007;57:737-763.
149. Usatine RP, Tinitigan R. Nongenital herpes simplex virus. *Am Fam Physician*. 2010;82(9):1075-1082.
150. Cernik C, Gallina K, Brodell RT. The treatment of herpes simplex infections: an evidence-based review. *Arch Intern Med*. 2008;168(11):1137-1144.
151. Bernstein DI, Bellamy AR, Hook EW, et al. Epidemiology, clinical presentation, and antibody response to primary infection with herpes simplex virus type 1 and type 2 in young women. *Clinical Infect Dis*. 2013;56(3):344-351.
152. Kimberlin DW, Whitley RJ. Antiviral therapy of HSV-1 and -2. In: Arvin A, Campadelli-Fiume G, Mocarski E, et al., eds. *Human Herpesviruses: Biology, Therapy, and Immunoprophylaxis*. Cambridge, UK: Cambridge University Press; 2007. Available at: https://www.ncbi.nlm.nih.gov/books/NBK47444/.
153. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2014;59(2):e10-e52. doi:10.1093/cid/ciu444.
154. Dhar DA. Furuncles and carbuncles. In: *Merck Manual*. Available at: http://www.merckmanuals.com/professional/dermatologic-disorders/bacterial-skin-infections/furuncles-and-carbuncles. Accessed March 13, 2017.
155. Liu C, Bayer A, Cosgrove SE, et al. Management of patients with infections caused by methicillin-resistant *Staphylococcus aureus*: clinical practice guidelines by the Infectious Diseases Society of America (IDSA). *Clin Infect Dis*. 2011;e18-e55. doi:10.1093/cid/ciq146.
156. Hall W, Degenhardt L. Adverse health effects of non-medical cannabis use. *Lancet*. 2009;374(9698):1383-1391.
157. Meier MH, Caspi A, Ambler A, et al. Persistent cannabis users show neuropsychological decline from childhood to midlife. *Proc Natl Acad Sci USA*. 2012;109(40):E2657-E2664. doi:10.1073/pnas.1206820109.
158. Centers for Disease Control and Prevention. Marijuana and public health: health effects. Available at: https://www.cdc.gov/marijuana/health-effects.htm. Accessed March 13, 2017.
159. Centers for Disease Control and Prevention. Opioid overdose: understanding the epidemic. Available at: https://www.cdc.gov/drugoverdose/epidemic/. Accessed March 13, 2017.
160. Jones CM, Baldwin GT, Compton WM. Recent increases in cocaine-related overdose deaths and the role of opioids. *Am J Public Health*. 2017;107:430-432. doi:10.2105/AJPH.2016.303627.
161. Meyer R, Patel AM, Rattana SK, Quock TP, Mody SH. Prescription opioid abuse: a literature review of the clinical and economic burden in the United States. *Popul Health Manag*. 2014;17(6):372-387. doi:10.1089/pop.2013.0098.

162. Chou R, Gordon DB, de Leon-Casasola OA, et al. Management of postoperative pain: a clinical practice guideline from the American Pain Society, the American Society of Regional Anesthesia and Pain Medicine, and the American Society of Anesthesiologists' Committee on Regional Anesthesia, Executive Committee, and Administrative Council. *J Pain*. 2016;17(2):131-157. doi:10.1016/j.jpain.2015.12.008.
163. Substance Abuse and Mental Health Services Administration. *SAMHSA Opioid Overdose Prevention Toolkit*. HHS Publication No. (SMA) 16-4742. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2016.
164. Osbore K. Regulation of controlled substance prescribing: an overview for certified nurse-midwives and certified midwives. *J Midwifery Womens Health*. 2017;62(3);341-347.

CHAPTER

7

Nutrition

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Nutrition and Health

Health and nutrition are intimately interrelated. Proper nutrition is essential to human growth, development, and well-being. Increasing attention to the role of the body's microbiome, especially in the gastrointestinal tract, in metabolic, cellular, and neurologic functioning has in turn increased our knowledge about the direct connection between what we eat and our health.[1] Since the average woman can expect to live until at least 80 years, women should be aware of the growing body of evidence that suggests a healthy diet can prevent a number of detrimental conditions, such as cardiovascular disease (CVD), cancer, adult-onset diabetes, osteoporosis, and age-related vision loss. Measures to reduce this disease burden are relatively simple and well researched: maintaining a healthy weight; eating a well-balanced, nutritious diet; not smoking; and exercising regularly. Unfortunately, instead of adopting healthier diets and lifestyle choices, more than 36% of women in the United States today are obese,[2] and since 2009, women have decreased their fruit and vegetable intake and increased their moderate alcohol intake.[3]

For midwives to promote changes that counter these negative trends, nutrition counseling should be incorporated into regular practice. Such health education can help women distinguish among the myriad sources of dietary advice. In addition, women need to understand some basic principles of nutrition, appreciate the physiologic response of the body to ingested food, and be familiar with current evidence-based dietary guidelines. Healthy nutritional intake is essential in all phases of life, including pregnancy, and the key principles remain the same for women whether they want to lose weight, to gain weight, or to maintain their weight. The basics of nutrition and how foods consumed affect normal physiology are discussed in this chapter. Although understanding individual components of the diet is important for health professionals, counseling women about diet should be positively focused on types of preferable foods to eat and the need to eat a variety of foods, rather than on individual macronutrients and micronutrients.

Principles of Nutrition and Nutrient Recommendations

Nutrients are the chemical components of food. Humans require more than 40 different nutrients for good health. These nutrients are classified as either macronutrients (fats, carbohydrates, and protein) or micronutrients (vitamins and minerals). Water is also a necessary nutrient, but it does not fit into either category. Water, vitamins, and minerals provide no calories but are necessary, although not totally sufficient, for the body to be able to utilize the energy provided by fat, carbohydrates, and protein.

Vitamin and mineral supplements have become increasingly popular in recent years, and some women incorrectly think these agents can supply all needed nutrients. Ideally, humans should obtain both macronutrients and micronutrients from a diet composed of a variety of foods rather than from nutritional supplements. This diet must be balanced in a way that prevents nutritional deficiencies and excesses. Variety is essential both to guarantee proper intake of all necessary nutrients and to benefit from the protective effects of certain dietary components against diseases such as cancer and heart disease. Today's research suggests that

decreased health risks associated with a healthy diet are due to combinations of substances in foods and food groups rather than to the effect of any single substance or nutrient.

Nomenclature for Nutritional Standards

The U.S. Dietary Guidelines for Americans is updated regularly and the most recent eighth edition was published in 2015.[4] The 2011 update to the United States Dietary Guidelines, for the first time took a crucial step in recognizing that it is more important to emphasize the *types of foods* people should eat, rather than concentrating on the *individual nutrient levels* they should consume.[5] Nevertheless, labeling on packaged foods regarding the overall nutritive value of a product can still be useful. Therefore, it is important to be familiar with the nomenclature used by various U.S. governmental organizations for nutritional standards setting.

U.S. nutritional standards have two origins: the Health and Medicine Division (HMD) of the National Academy of Medicine (formerly the Institute of Medicine [IOM]) Food and Nutrition Board[5] and the Food and Drug Administration (FDA).[6] The Food and Nutrition Board standards were originally developed during World War II as part of an effort to ensure adequate nutrition for members of the armed services. Since then, these standards have been used to set government policy related to food programs such as the School Lunch Program and food relief efforts globally. The FDA guidelines are more recent and are intended to assist consumers in obtaining recommended nutrients in their diets.

Table 7-1 compares the terms used by the HMD's Food and Nutrition Board and by the FDA.[5,6] An important characteristic of the Daily Reference Intakes (DRIs) is that they not only aim to determine minimum nutrient intake levels necessary to prevent nutritional deficiencies, but also strive to set standards to decrease the incidence of chronic diseases such as osteoporosis and cancer.

Table 7-1	U.S. Nutritional Standards
Food and Nutrition Board, Health and Medicine Division, National Academy of Medicine	
Dietary Reference Intakes	
DRIs aim to determine minimum nutrient intake levels necessary to prevent nutritional deficiencies, and set standards to decrease chronic diseases such as osteoporosis and cancer. This term has more or less supplanted RDAs.	
DRIs are a combination of RDAs and AIs.	
DRIs also specify tolerable UL, RDAs, EAR, and AIs:	
Tolerable UL	
The maximum level of daily nutrient intake that is unlikely to pose risks of adverse health effects to almost all of the individuals in the group for whom it is designed.	
RDAs	
Developed and updated periodically.	
Set the recommended average intake over a 3- to 7-day period for each nutrient specified.	
Fewer than half of the more than 40 necessary nutrients have an established RDA.	
RDAs are set quite high to meet the needs of almost all (97–98%) individuals in a group, so they may be too high for individual needs.	
EAR	
The intake value that is estimated to meet the requirement defined by a specified indicator of adequacy in 50% of an age- and gender-specific group. At this level of intake, the remaining 50% of the specified group would not have their	

needs met.
AIs
Used to develop a recommended intake when a scientific basis for establishing the EAR is lacking.
Food and Drug Administration
DVs
DVs appear on FDA-regulated products.
DVs are not meant to set levels of nutrients to be consumed every day, but rather to help determine how particular foods fit into an overall healthy diet.
DVs are calculated based on a diet of 2000 calories per day (unless otherwise stated).
DVs are calculated from two sources—DRVs and RDI:
DRVs
Recommended proportions of protein, carbohydrate, and fat in the diet.
Recommended grams per 1000 calories of dietary fiber intake.
Recommended daily maximums for sodium and potassium.
RDI
To ensure that the nutritional needs of all age groups are met, the RDI is calculated from the highest DRI for each nutrient.

Abbreviations: AIs, adequate intakes, DRI, dietary reference intake; DRV, Daily Reference Value; DVs, Daily Values; EAR, estimated average requirement; FDA, U.S. Food and Drug Administration; RDAs, Recommended Dietary Allowances; RDI, Reference Daily Intake; UL, upper intake level.

Based on Food and Nutrition Board. *Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals.* Washington, DC: National Academy of Sciences; 2004, updated 2011[5]; Office of Nutrition Labeling and Dietary Supplements. *Food Labeling Guide.* College Park, MD: Food and Drug Administration; 2009.[6]

Most food labels are based on a “typical” diet of 2000 calories per day. However, the number of calories actually needed in a day depends on the individual’s daily expenditure of calories. Several formulae are available to calculate how many calories an individual needs daily,[7] but clinicians may not have the time to make those calculations. Another option is to use an Internet-based website that calculates the number of calories needed to maintain or lose weight when provided with the individual’s specific sex, age, height, weight, and daily activity levels (see the Resources section at the end of this chapter). According to guidelines issued by the American Heart Association, women age 19 to 30 years who are not obese and of average stature will need approximately 2000 calories per day to maintain their current weight if they do not engage in physical activity beyond activities of daily living such as work and walking to the bus (sedentary physical activity level). Women who are obese or very active will need more calories to maintain their current weight. Women who are older, or who are trying to lose weight, will require fewer calories. However, all food is not the same, and calories, therefore, are simply one consideration within a diet. The sections that follow review macronutrients, micronutrients, and the physiologic response to ingested food.

Macronutrients

Macronutrients contain calories and are the energy-providing nutrients for the human body. The three basic macronutrients are fat, carbohydrates, and proteins. Macronutrients are needed in large quantities and meet the bulk of a human body energy needs. One of the primary components of a healthy diet is consuming fat, carbohydrate, and protein in balanced proportions.

Fats

Fats are an important source of energy, providing more calories per gram than either protein or carbohydrates. Fat provides energy at a rate of 9 kcal/g, compared to 4 kcal/g for protein and carbohydrates. Fats are composed of fatty acids and have various roles in the human body, including transport and digestion of the fat-soluble vitamins and components of cell structure. Stored body fat facilitates temperature regulation by serving as insulation and helps to protect vital organs by providing a cushioning effect. In addition, dietary fat intake increases the pleasure of eating and helps signal satiety during a meal.

Since the 1950s, the American Heart Association has urged persons residing in the United States to eat less fat in their diets, particularly by eliminating cholesterol from red meat and eggs. This campaign has been successful in decreasing fat intake from 45% of total calories to 33%.[8] However, the percentage of calories consumed from fats is *not* linked to chronic diseases; instead, what matters is the *type* of fats consumed. The "fat is bad" campaign that caused people to replace fat calories with generally high glycemic (refined) carbohydrates has been shown to worsen serum lipoprotein levels and is one of the underlying causes of the current obesity and type 2 diabetes epidemic.[9]

Body Fats

Body fats need to be distinguished from dietary fat. For example, the cholesterol (a fatlike substance present in all animal tissue) consumed in diet is different than the cholesterol in blood that is measured as part of a lipid panel. Dietary cholesterol is found in foods of animal origin such as meat and eggs, while blood cholesterol is a waxy, fatlike substance manufactured by the body and stored in the liver. Fifty years of evidence has found that although there is a slight association between dietary cholesterol and serum cholesterol, dietary cholesterol has minimal effect on risk for cardiovascular disease due to the complexities of the metabolic effects of ingested food.[10] For example, eggs—a common source of cholesterol—also are rich in other nutrients that may counterbalance the cholesterol's detrimental effects on the cardiovascular system. In addition, more harmful physiologic effects may occur when an individual substitutes an egg for breakfast with a high-glycemic food, such as a large muffin, as discussed later in this chapter.

The body uses blood cholesterol to make steroid hormones such as estrogen and progesterone, as described in the *Anatomy and Physiology of the Female Reproductive System* chapter. Blood cholesterol is also required for production of vitamin D and bile, as well as being an important component of cell membranes.

Several subtypes of cholesterol are found in the body, although the two primary ones are low-density lipoprotein (LDL) and high-density lipoprotein (HDL). LDL transports cholesterol out of the liver to other parts of the body. Cells attach to these particles and extract LDL or cholesterol. Excess amounts of LDL promote the production of fatty plaques on arterial walls, causing them to lose elasticity and narrow, resulting in arteriosclerosis. If a plaque breaks free, it may cause a cardiac event such as a heart attack or stroke. LDL often is characterized as "bad" or "lousy" cholesterol. Conversely, HDL carries excess cholesterol away from the

arteries and back to the liver; thus, it is known as “good” or “healthy” cholesterol. A high level of HDL appears to have a protective effect against coronary heart disease and heart attacks. Exercise is strongly associated with higher levels of HDL in the body and, therefore, with lower risk of cardiovascular disease. In women, unlike in men, the total cholesterol to HDL ratio or the non-HDL level is more predictive of the risk for cardiovascular disease than the total cholesterol level.

The latest dietary guidelines recommend maintaining a daily dietary intake of cholesterol of less than 300 mg per day, and state that serum HDL level should be higher than 35 mg/dL, with a level of 60 mg/dL being optimal. The goal for the total cholesterol to HDL ratio is 5:1, with 3.5:1 being the ideal.

Another way in which fats are transported through the blood to cells is in the form of triglycerides. Serum triglycerides are derived either from consumed fat, especially trans fats, or synthesized de novo by body fat or the liver. The amount of triglycerides synthesized de novo is positively correlated with the consumption of refined carbohydrates. Triglycerides store excess calories as fat and break down stored fat when a source of energy is needed, especially by the brain. Whether a high triglyceride level is an independent cause of heart disease remains controversial, because many individuals with high triglyceride levels also have high LDL and low HDL cholesterol levels, which are known risk factors for heart disease.[11] It does appear, however, that high triglyceride levels are more predictive of cardiovascular disease risk in women than in men and are a risk factor for developing type 2 diabetes, metabolic syndrome, and ischemic stroke in women. Data collected for the National Health and Nutrition Examination Survey (NHANES) reveal that serum levels of total cholesterol, LDL, and triglycerides declined from the 1990s through 2014 in all population subgroups of women, even among those not taking lipid-lowering medications.[12]

Dietary Fats

Dietary fats are characterized by the chemical structure of their fatty acids. They are classified into four types: trans fats, saturated fats, polyunsaturated fats, and monounsaturated fats.

Trans Fats

Trans fats are produced when vegetable oils are heated in the presence of hydrogen gas and a catalyst, resulting in a partially hydrogenated oil. Such oils become a solid when they cool, making them easier to transport; they are more stable and unlikely to spoil, providing a longer shelf life; and they can be reheated without breaking down, so they are ideal for making fried foods. Although trans fats are found in small amounts in beef and dairy fat, they are largely consumed in the form of commercially made products. Trans fats are harmful because they elevate LDL cholesterol and lower HDL cholesterol; they also stimulate production of prostaglandins and other eicosanoids that increase inflammation, platelet aggregation, and vasoconstriction. The combination of these effects results in an increased risk for cardiovascular disease, diabetes, gallstones, weight gain, and ovulatory infertility.[13] The Nurses’ Health Study reported that for each 2% increase in calories from trans fats, the risk of

coronary heart disease increases 23%.[14] Evidence of adverse health effects following consumption of trans fats resulted in product labeling requirements that subsequently prompted the reformulation of many products to eliminate partially hydrogenated oils. Between 2003 and 2009, average trans fat intake decreased in the United States from 4.6 g/day to 1.3 g/day.[15]

Saturated Fats

Saturated fats are derived from both animal and plant sources. These fats often are solid at room temperature and are found in highest amounts in meat fat, butter, whole-milk products, coconut oil, palm oil, and palm kernel oil. Consumption of these fats worsens serum lipoprotein profiles.

Unsaturated Fats

In contrast to saturated fats, unsaturated fats have several beneficial roles in the body, including improving serum cholesterol levels, reducing inflammation, and stabilizing heart rhythms. These fats are found primarily in vegetable oils, nuts, and seeds, and are liquid at room temperature.

Unsaturated fats are of two types: *monounsaturated* and *polyunsaturated*. Monounsaturated fats are found in high concentrations in olives, peanuts, oils such as canola oil, avocados, nuts such as almonds and pecans, and seeds such as pumpkin and sunflower seeds. Polyunsaturated fats are grouped into two types, distinguished by their chemical bonds: omega-6 (n-6) and omega-3 (n-3) fatty acids. Both n-6 and n-3 are considered “essential fatty acids” due to the body’s limited ability to produce them, which means they must be obtained from dietary sources. Omega-6 fatty (linoleic) acids are found in safflower, sunflower, corn, soybean, and flaxseed oils, as well as canola oils. Omega-3 fatty acids are found in oily fish such as salmon, mackerel, anchovies, and sardines; algae; and plant sources such as chia and flax seeds, walnuts, and oils such as soybean and canola.

The amount of n-3 fats in the U.S. diet has decreased significantly since the 1950s, in part due to changes in the food chain. Today most meat comes from grain-fed animals; relatively speaking, grain-fed animals have less n-3 fats in their meat than grass-fed animals. The consumption of oily fish has also decreased over the same period of time, perhaps due to warnings about heavy metals in fish. Observational studies have shown that the incidence of heart disease mortality, stroke, and mood disorders appears to be correlated with differences in n-3 fat intake.[16] Results from supplementation trials with n-3 fats to prevent heart disease have been somewhat mixed, with some evidence suggesting that such supplements provide secondary prevention among those persons with heart disease, but have a limited effect in preventing arrhythmias.[17]

In summary, the total number of calories obtained from fat is unimportant for cardiovascular and general health; rather, it is the type of fats consumed that is important for health outcomes. Trans fats should be eliminated from the diet as much as possible. Replacing saturated fats with monounsaturated and polyunsaturated fats will provide modest improvements in protection against heart disease. Consumers should be cautious about the foods they choose to

substitute for trans fats and saturated fats, as some seemingly healthy alternatives are not necessarily healthier substitutes. Nevertheless, a good recommendation is to reduce consumption of red meat and whole-milk dairy products, increase intake of fish to at least two 6-ounce servings per week, and increase consumption of soy products, nonhydrogenated vegetable oils, and nuts. **Table 7-2** provides the distribution of daily recommended values of macronutrients for women age 19–30 years, and **Table 7-3** shows the recommended DRIs for women.[5,18]

Table 7-2 **Recommended Daily Values for Macronutrients for Women 19–30 Years**

	Percent Daily Calories (Actual Daily Amount)	
	Food and Drug Administration	**Health and Medicine Division**
Fat	30%	20–35%
Saturated fat	< 10%	As little as possible
Cholesterol	(< 300 mg)	As little as possible
Trans fat	0	0
Carbohydrates	60%	45–65%
Dietary fiber	(25 g)	(28 g)
Protein	10%	10–35%

Based on Food and Nutrition Board. *Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals.* Washington, DC: National Academy of Sciences; 2004, updated 2011[5]; Office of Nutrition Labeling and Dietary Supplements. *Food Labeling Guide.* College Park, MD: Food and Drug Administration; 2009.[6]

Table 7-3 **Daily Reference Intakes of Macronutrients for Women**

	Nonpregnant		Pregnancy		
	14–18 Years	**Adults**	**Singleton**	**Multiple**	**Lactation**
Carbohydrate (g)	130	130	175	330	210
Fiber (g)	26	21–25	28	28	29
Fat (g)	ND	ND	ND	156	ND
Protein (g)	46	46	71	175	71
Water (L)	2.3	2.7	3.0	3.0	3.0

Abbreviation: ND, not determined.

Data from Food and Nutrition Board. *Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals.* Washington, DC: National Academy of Sciences; 2004, updated 2011[5]; Goodnight W, Newman R. Optimal nutrition for improved twin pregnancy outcome. *Obstet Gynecol.* 2009;114(5):1121-1134.[18]

Carbohydrates

Carbohydrates, which can be found in grains, vegetables, fruits, and sugars, are the major

dietary source of glucose, the body's essential energy for cellular metabolism. All carbohydrates except insoluble fibers are broken down by the body into the basic sugars and absorbed in the bloodstream. Glucose, galactose, and fructose can be used immediately by the body or can be stored in the liver or muscle tissue in the form of glycogen, which is then converted to glucose whenever there is a need for reserve energy.

In the past, carbohydrates have been described as simple or complex, with people being encouraged to eat more complex carbohydrates. However, there is no distinction in this recommendation between the different physiologic consequences that occur after eating different types of complex carbohydrates. Because the amount of insulin that is stimulated in response to carbohydrate intake is a key factor in the body's physiologic response, researchers have developed two measures to assess this response: the glycemic index and the glycemic load.[19] In general, ingestion of high-glycemic foods results in worsened lipoprotein profiles, more thrombotic activity, abnormal glucose metabolism, increased inflammation, and more cellular proliferation.

Glycemic Index

The glycemic index is a measure of the blood glucose response 2 hours after ingesting 50 grams of a food. This index is influenced by the amount of fiber and fat in the consumed food, both of which slow the absorption of the carbohydrate. Therefore, highly processed grains such as white bread, white rice, and semolina-flour pasta have a high index. For example, white rice's glycemic index is 89, whereas that for brown rice is 48. Vegetables have a low glycemic index due to their fiber content and low natural sugar content; indeed, in some cases, it is hard to even measure their glycemic index. For example, obtaining 50 grams of carbohydrate from broccoli requires ingesting 16 cups. Fruits, although naturally sweet, if consumed as the whole fruit, have a low glycemic index due to the fruit's fiber; for example, an apple has a glycemic index of about 10. In general, low-glycemic foods have a glycemic index less than 55, whereas those with a glycemic index higher than 70 are considered high-glycemic foods. The glycemic index values for a variety of foods have been published[16] and may be obtained online from the University of Sydney in Australia.

Glycemic Load

Compared to the glycemic index, the glycemic load is a better measure of the amount of insulin stimulated by consumption of specific foods. The glycemic load is calculated as the glycemic index of the food multiplied by the amount consumed, expressed per 100 grams. A low glycemic load is less than 10 and a high glycemic load is higher than 20, with values in between considered intermediate. For example, the glycemic index for one cup of brown rice is 45, but the glycemic load is 18. The same amount of white rice would have a glycemic index of 83 and a glycemic load of 43. This is a typical example, in which unprocessed grain products stimulate the production of less insulin than do processed foods such as white rice. The same is true for whole fruits as compared to consuming only the juice of the fruit. A person would need to consume 3 to 4 oranges to register the glycemic load of just 8 ounces of orange

juice.

Fiber

Fiber is a type of complex carbohydrate composed of nonstarch polysaccharides that cannot be digested by enzymes in the small intestine. Fiber is characterized as either soluble or insoluble based on its solubility in water. Soluble fiber types include pectin found in fruits, beta-glucans found in oats and barley, and gums found in beans and cereals. Insoluble fiber types include cellulose found in leaves, root vegetables (e.g., beets and carrots), bran, and whole wheat; hemicellulose found in bran and whole grains; and lignin found in plant stems and leaves. The typical U.S. diet is characterized by being low in dietary fiber. Fiber intake among adults in the United States averages approximately 16 grams, or only 60% of recommended amounts.[17] Research has revealed that dietary fiber is associated with a decreased risk of heart disease, most likely through lowering total cholesterol and LDL cholesterol levels, and with decreased risk of type 2 diabetes, diverticulitis, and constipation.

The Health and Medicine Division of the National Academies of Science, Engineering, and Medicine and the U.S. Department of Agriculture (USDA) recommend that carbohydrates compose 45% to 65% of daily caloric intake. Women should attempt to maximize their intake of low-glycemic foods rich in fiber, such as whole grains, vegetables, and fruits, and to minimize their intake of high-glycemic foods such as sweetened and energy drinks; refined grains such as white bread, white rice, and russet potatoes; and high-sugar snacks. A desirable intake of fiber for women is 20 to 28 grams per day.

Protein

Protein is the basic component of cells and is needed for cellular growth, replacement, and repair. Enzymes—the substances responsible for controlling the processes that keep the human body functioning smoothly—are composed of protein. Hormones, hemoglobin, and antibodies also are composed partially or entirely of protein. Protein, in turn, is composed of organic compounds known as amino acids. The different arrangements of amino acids into proteins determine the particular properties of each protein.

Approximately 20 amino acids exist that are necessary for human growth and metabolism. The body is able to produce the majority of these necessary amino acids. Approximately nine amino acids, however, must be obtained through foods—the so-called *essential amino acids*. Foods of animal origin, such as meat, poultry, fish, eggs, and dairy products, provide all of these essential amino acids and are known as complete proteins. Proteins derived from plants, such as legumes, nuts, and grains, lack certain essential amino acids and are termed incomplete proteins. Even so, a vegetarian diet can supply all of the essential amino acids as long as a variety of plant-based proteins are eaten throughout the day.[20]

Proteins cannot be stored in the body, so each day they must be consumed to avoid the body breaking down nonessential tissue, such as muscle, to supply proteins vital for survival. Recommended protein intake is 0.8 to 1.0 g/kg, or 10% to 35% of total calories. While protein intake deficiencies are common in the developing world, most persons in the United States

consume adequate amounts of protein. The average intake of women in the United States is approximately 70 g/day or 16% of kilocalories consumed, with only 8% of adolescents and older women having less than average intakes.[21]

The body does not distinguish between animal and plant sources of amino acids. However, protein food sources usually contain other nutrients, such as saturated fat. Evidence from the Nurses' Health Study suggests that substituting plant-based protein for high-glycemic carbohydrates can decrease a woman's risk for heart disease and type 2 diabetes.[22] **Table 7-4** contains a list of foods rich in complete and incomplete proteins.[23]

Table 7-4 Examples of Food Sources of Protein

Food	Quantity	Amount of Protein (g)
Complete Proteins		
Beef, chuck roasted	3 oz	26
Chicken, roasted	3 oz	27
Tuna, fresh	3 oz	25
Pork, center loin	3 oz	23
Beef, lean ground	3 oz	22
Tuna, canned	3 oz	20
Ham	3 oz	19
Chicken breast, deli-type slices	3 oz	17
Salmon	3 oz	17
Scallops	3 oz	17
Cottage cheese	½ cup	14
Eggs	2 large	13
Shrimp	3 oz	12
Yogurt	1 cup	8–12
Milk, any type	1 cup	8
Cheddar cheese	1 oz	7
Incomplete Proteins		
Lentils	1 cup, cooked	18
Beans, cooked (different types)	1 cup	12–15
Tofu, firm	½ cup	10
Green peas	1 cup	9
Quinoa, cooked	1 cup	8
Peanut butter	2 Tbsp	8
Peanuts	1 oz	7
Egg noodles, cooked	1 cup	7
Brown rice, cooked	1 cup	5
White rice, cooked	1 cup	4
Bread, whole wheat	1 slice	4

Sunflower seeds	1 oz	4

Data from U.S. Department of Agriculture. USDA National Nutrient Database for Standard Reference, Release 28. 2016. Available at: https://www.ars.usda.gov/northeast-area/beltsville-md/beltsville-human-nutrition-research-center/nutrient-data-laboratory/docs/usda-national-nutrient-database-for-standard-reference/. Accessed May 11, 2017.[23]

Physiologic Response to Food Intake

Food is a hormonal stimulant more powerful than most drugs. Understanding this reality may be the impetus providers need to spend time with women assessing their diets and helping them to choose better foods. The body's response to food intake depends on the composition of both the macronutrients and the micronutrients ingested. With regard to prevention of inflammation and other processes that promote disease, especially chronic disease, the most important physiologic response to food intake is how much insulin production is stimulated. Stimulation of insulin production is a direct response to the amount and rate of glucose response from a meal. This glucose response can be measured by estimating the glycemic index of a meal.

Distinguishing high-glycemic meals from low-glycemic meals is important in understanding the body's physiologic response.[19] A high-glycemic meal creates a rapidly rising and high blood glucose response, which results in a similar insulin production response. Subsequently, the blood glucose level falls more rapidly than the insulin level decreases, which results in high insulin levels, low glucose levels, and suppression of glucagon production. Faced with these conditions, the body responds as if there is adequate glucose—that is, it suppresses the secretion of free fatty acids, blocking the body's access to stored fat for energy. Low glucose levels also cause poor brain function, which stimulates the appetite, especially for a high-glycemic meal. After several more hours, to maintain a euglycemic state, skeletal muscles become insulin resistant, decreasing glucose uptake. In turn, free fatty acids and cortisol are secreted to allow access to stored glucose from the liver. In addition, other inflammatory hormones are released, causing oxidative stress and potentially damage from the production of excessive free radicals. Over time, this cycle of increased levels of glucose, insulin, and free fatty acids damages pancreatic beta cells, potentially resulting in type 2 diabetes, as well as affecting other processes associated with a variety of conditions, including cardiovascular disease, cancer, neural tube defects in the fetus (if the woman is pregnant), and gallbladder disease.

Eating a diet rich in carbohydrates from whole grains, beans, fruits, and vegetables results in a much more modulated glucose response, in which insulin levels follow but never exceed glucose levels. Therefore, the result is appropriate glucagon stimulation. Over time, as glucose levels decline, stored fat then is available as an alternative energy source until the next meal.

The metabolism of fatty acids results in another important set of physiologic responses. This metabolism leads to the formation of different types of eicosanoids. Eicosanoids are short-acting, powerful, locally acting hormones that aid intracellular communication and regulate cyclic adenosine monophosphate (cAMP), which is needed for peptide hormones to exert their effects in cells. The first-identified eicosanoid was prostaglandin, but midwives will recognize other eicosanoids such as prostaglandin E_2, prostaglandin F_2-alpha, and thromboxane, which exist endogenously. These specific agents can dampen cAMP activity as well as exert a pro-inflammatory effect, increase platelet aggregation and cellular proliferation, and cause vasoconstriction. Conversely, the hormones that increase cAMP (e.g., prostacyclin, PGE_1) have the opposite effects. The body needs all of these substances for self-regulation and appropriate functioning. With regard to nutrition, the type of eicosanoid produced, either pro-inflammatory or anti-inflammatory, is influenced by such factors as the

presence of insulin, glucagon, omega-3 fatty acids, and trans fats. In brief, the quality of human nutrition encompasses not only the type of food that is consumed, but also the physiologic and chemical responses to it, as well as the presence of viruses and stress.

Micronutrients: Vitamins and Minerals

Micronutrients are dietary elements that the body needs in trace amounts. Metabolism and use of macronutrients by the body require a host of enzymatic and hormonal processes that are also influenced by the necessary presence of micronutrients—specifically, vitamins and minerals, which for the most part cannot be synthesized by the body. Micronutrient deficiencies can have profound adverse health effects. Micronutrient deficiencies are a common health problem in developing nations and in some geographic areas in developed nations.

Vitamins are organic substances used by the body as catalysts for intracellular metabolic reactions, whereas minerals are inorganic substances. Both vitamins and minerals are essential for physiologic function, but must be obtained via the diet. Vitamins are classified as fat-soluble or water-soluble: Water-soluble vitamins are excreted from the body and are not stored, whereas fat-soluble vitamins are stored in body fat.

A few of these micronutrients are highlighted here because they play a special role in women's health or because women in the United States are likely to be deficient in them. The role of vitamins and minerals in pregnancy are discussed in the *Prenatal Care* chapter. **Table 7-5** lists the recommended intakes for selected vitamins and minerals in women age 14 to 70 years, the requirements in pregnancy and lactation, and common food sources of these nutrients.[24]

Table 7-5 **Recommended Daily Reference Intakes of Vitamins and Micronutrients in Women**

Vitamin/Mineral	Nonpregnant Women 14–18 Years	Nonpregnant Women 19–50 Years	Nonpregnant Women 51–70 Years	Pregnancy	Lactation	Dietary Source
Vitamin A (mcg)	700	700	700	770	1200	Yellow/orange vegetables and fruits
Vitamin C (mg)	65	75	75	85	120	Citrus fruits
Vitamin D (IU)	600	600	600	600–4000[a]	600–4000[a]	Fortified dairy
Vitamin E (mg)	15	15	15	15	19	Nuts, vegetable oils
Thiamin (mg)	1.1	1.1	1.1	1.4	1.4	Pork, enriched grain
Riboflavin (mg)	1.1	1.1	1.1	1.4	1.4	Meat, enriched grain
Niacin (mg)	14	14	14	18	17	Meat, nuts, legumes
Vitamin B_6 (mg)	1.2	1.3	1.5	1.9	2.0	Chicken, fish, enriched grains
Folate (mcg)	400	400	400	600	500	Leafy vegetables, liver, fortified cereal
Vitamin B_{12} (mcg)	2.4	2.4	2.4	2.6	2.8	Animal-based food
Calcium (mg)	1300	1000	1200	1300	1300	Dairy
Magnesium (mg)	360	320	320	350–360	310–320	Seafood, legumes, grains
Phosphorus (mg)	1250	700	700	700	700	Meat
Potassium (g)	4.4	4.7	4.7	4.7	5.1	Apricots, figs, nuts, soy, wheat germ
Iodine (mcg)	150	150	150	220	290	Iodized salt, seafood
Iron (mg)	15	18	8	27	9	Meat, eggs
Choline (mg)	400	425	425	450	550	Eggs
Zinc (mg)	9	8	8	11	12	Egg yolk, liver, oysters

[a] Lower values: Food and Nutrition Board; higher values: Hollis and Wagner.

Data from Food and Nutrition Board. *Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals.* Washington, DC: National Academy of Sciences; 2004, updated 2011[5]; Hollis BW, Wagner CL. Vitamin D and pregnancy: skeletal effects, nonskeletal effects, and birth outcomes. *Calcif Tissue Int.* 2012;92(2):128-139.[24]

Fat-Soluble Vitamins

Large doses of fat-soluble vitamins are stored in the body and can reach toxic levels that have dangerous effects. For example, accumulation of vitamin A, which occurs when this nutrient is ingested as retinol, can result in liver toxicity, visual problems, and increased risk for hip fractures.

Vitamin A is important for vision (especially night vision), a healthy immune system, and cell growth. Vitamin A deficiency is rare in the U.S. population, but it may be an important nutrient to attend to in low-resource countries. **Table 7-6** presents the current status of micronutrients in childbearing women in the United States.[25,26]

Table 7-6 Recommended and Actual Status of Micronutrients in U.S. Women 20–39 Years of Age by Race/Ethnicity According to National Nutrition and Health Examination Survey, 2003–2006

		Actual Status for Women 20–39 Years of Age, Geometric Mean Values			
Micronutrient (Measurement)	Adequate Levels in Women	All Women 20–39 Years	Mexican	Non-Hispanic Black	Non-Hispanic White
Vitamin A (mcg/dL)	> 20	50.0	45.0	42.7	53.5
Vitamin B_{12} (pg/mL)	> 400	439	501	489	414
Vitamin D 25(OH)D (nmol/L)	≥ 50 or 75	55.3	43.9	32.7	66.7
Vitamin E (mcg/dL)	> 500	1010	1000	917	1040
Folate as RBC folate (ng/mL)	> 95[a] > 330[b]	253	256	213	267
Iron as serum ferritin (ng/mL)	> 15	38.1	33.7	30.1	41.0
Iodine: urinary excretion (ng/L)	100–199	118	171	118	112
Iodine: during pregnancy[c]	150–249	129[d]			

Abbreviation: RBC, red blood cell.

[a] Amount to prevent hypersegmented RBCs.

[b] Amount to prevent neural tube defects.

[c] Years 2005–2010 (Caldwell).

[d] Below adequate levels.

Data from National Center for Environmental Health. *Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population*. Atlanta, GA: Centers for Disease Control and Prevention; 2012[25]; Caldwell KL, Pan Y, Mortensen ME, Makhmudov A, Merrill L, Moye J. Iodine status in pregnant women in the National Children's Study and in U.S. women (15–44 years), National Health and Nutrition Examination Survey 2005–2010. *Thyroid.* 2013;23(8):927-937.[26]

Vitamin D promotes absorption of calcium and phosphate and helps deposit these minerals in teeth and bones, but also plays a role in health beyond bone formation. Most body tissues have vitamin D receptors; thus, this vitamin is involved with regulation of many systems, such as blood pressure, glucose and insulin regulation, and modulation of the immune system. Vitamin D is a not a true vitamin, as humans synthesize D_3 when the skin is exposed to ultraviolet sunlight. Vitamin D_3 synthesis from sunlight is affected by intensity of sunlight,

especially related to latitude and season; the presence of air pollution; and factors that limit the skin's absorptive ability, such as amount of pigmentation, presence of sunscreens, and increasing age. Oily fish and fish liver oils are other sources of vitamin D_3, although absorption is decreased in people with intestinal malabsorptive conditions. Vitamin D_2—another form of vitamin D—is derived from plant sterols, such as those found in mushrooms. Vitamin D is also consumed through fortified dairy products. Both D_3 and D_2 are inactive forms that require metabolism in the liver to convert the vitamin D to 25(OH)D, the form most commonly used to assess a person's vitamin D levels, followed by metabolism in the kidney to convert it to $1,25(OH)_2D$, the physiologic active form.

Vitamin D deficiency has historically been defined as a serum 25(OH)D concentration less than 10 ng/mL (25 nmol/L), which is the level at which rickets or myopathy develops. Currently, there is some controversy about what constitutes vitamin D deficiency in adults based on whether the goal is bone health only or if other general health conditions are also considered. The Health and Medicine Division's Food and Nutrition Board, using bone health as the goal, has defined a vitamin D level of less than 50 nmol/L (20 ng/mL) as inadequate, with the recommended range for this vitamin being 50–100 nmol/L (20–40 ng/mL).[27] This agency recommends supplement intakes of 600–800 IU in the absence of adequate sunlight, with an upper tolerable limit of 4000 IU. However, the U.S. Endocrine Society, the American Geriatrics Society, and Central Europe guidelines recommend a minimum of 75 nmol/L (30 ng/mL) as the vitamin D level, with a level in the range of 75–155 nmol/L (40–60 ng/mL) considered ideal.[28,29] These organizations acknowledge that individuals may need to consume 1500–2000 IU daily to achieve these levels, with an upper tolerable level of 10,000–40,000 IU. Adults who are obese or who take anticonvulsants, glucocorticoids, some antifungals, or antiretroviral drugs, or who have inflammatory bowel disease or gastric bypass surgery, may require two to three times this dose to achieve healthy levels.[28]

Despite the current controversy about precisely which serum values represent vitamin D deficiency—vitamin D 25(OH)D levels of less than 20 ng/mL (50 nmol) versus less than 30 ng/mL (75 nmol)—there is no question that vitamin D deficiency is prevalent in all ethnic groups of women in the United States (**Table 7-6**). More than 50% of women in the United States have serum vitamin D levels less than 50 nmol/L (20 ng/mL), and non-Hispanic black women and Mexican women have particularly low average levels.[25]

Water-Soluble Vitamins

Folic Acid

Among the water-soluble B vitamins, folic acid (vitamin B_9) has a great import for childbearing women due its role in preventing neural tube defects. Folate is essential for RNA and DNA synthesis. Observational studies have found high folate levels to be associated with lower rates of cancer (especially intestinal cancer), cardiovascular disease, dementia, and depression. Supplementation with folate and other B vitamins has proved effective in lowering homocysteine levels, which are associated with cardiovascular disease. However, randomized trials of fairly large populations have not found that vitamin B supplements decrease the incidence of any of these conditions. Nevertheless, folate deficiency is clearly associated with megaloblastic anemia. Women at risk for megaloblastic anemia include those with poor diets, alcoholics, and those who have malabsorptive disorders or take pharmaceutical agents that act as folic acid antagonists (e.g., anticonvulsants, sulfasalazine [Azulfidine]).

Folate is found in liver and leafy greens such as spinach, but the primary dietary source of this micronutrient in the United States is fortified cereal and multivitamin supplements. Folate status can be assessed by either serum folate or red blood cell (RBC) folate tests, although the latter is a better indicator of tissue levels or long-term status.

In general, the U.S. population is not folate deficient. Since food fortification began in the mid-1990s, the percentage of the population that is folate deficient, defined as being at risk for hypersegmented RBCs, in all age and ethnic groups is 1% or less.[30] Discussion about adequate levels for pregnancy can be found in the *Prenatal Care* chapter. Although there is little risk of toxicity from high folic acid intake, concern has arisen that consumption of excess amounts could exacerbate the anemia and cognitive symptoms associated with vitamin B_{12} deficiency. Therefore, folic acid supplements of 1 mg or more require a provider prescription.

Vitamin B_{12}

Vitamin B_{12} deficiency can lead to anemia, and neurologic and cognitive dysfunction including depresson.[31,32] Everyone needs vitamin B_{12} in their diet; human beings do not make this vitamin on their own, and the vitamin B_{12} that may be produced by bacteria in the colon is mostly not bioavailable. Subgroups at risk for vitamin B_{12} deficiency are those with small bowel or inflammatory bowel disease, history of gastric bypass surgery, vegetarians, and elderly persons. If a pregnant woman is vitamin B_{12} deficient, she should be quickly treated, since this condition may affect the developing fetus.

Beef liver, beef, fatty fish (e.g., salmon), and dairy products are good dietary sources of vitamin B_{12}. For vegetarians, this nutrient may be obtained from soy and other products like nutritional yeast, which is fortified with vitamin B_{12}.

Recommended routine supplementation of vitamin B_{12} and eating vitamin B_{12}–fortified foods is recommended by the Food and Nutrition Board for all adults older than age 50 due to the prevalence of atrophic gastritis in older adults, which inhibits absorption of vitamin B_{12}.[33]

A 2015 meta-analysis of the use of vitamin B_{12} supplements in conjunction with antidepressants among individuals with depression did not show any short-term benefit from this adjunct therapy, but supplementation may be useful in preventing depression relapse.[31]

Vitamin B_{12} supplements may be provided as injections, orally or intranasally. Oral supplements are relatively inexpensive and widely available. The most common form of vitamin B_{12} supplement—and the best studied—is cyanocobalamin. A typical vitamin B_{12} supplementation regimen might be 250–500 mcg/day, which would probably compensate for poor absorption of supplements. However, smokers should be aware that vitamin B_{12} supplementation with cyanocobalamin may not be effective for them; smokers may excrete much more cyanocobalamin in the urine.[32] Research on this topic is far from definitive, but smokers might consider a non-cyanocobalamin form of vitamin B_{12}, such as methylcobalamin. Although some sources suggest that sublingual doses are more readily absorbed, the only study comparing routes found no difference in efficacy between sublingual and oral ingestion of vitamin B_{12}.[33] Intake of relatively high doses of vitamin B_{12} does not appear to be associated with any detrimental health problems, so no upper tolerable limit has been established.[34]

In summary, most women in the United States have adequate intakes of vitamins, with the exception of vitamin D and, in some subpopulations, folate or vitamin B_{12} (e.g., in the elderly). Nutrition scientists have been learning more about the roles that vitamins play in health beyond the basic understanding achieved in the twentieth century. Randomized trials on the use of vitamin supplements as a means to prevent chronic disease have yielded disappointing results, but it may be that, without addressing the content of the entire diet, vitamin supplementation is not powerful enough to overcome the physiologic effects of types and amounts of macronutrients in the diet.

Minerals

Minerals are chemical compounds found in the earth and absorbed by plants or animals, or dissolved in water. The five major minerals found in the human body are calcium, magnesium, phosphorus, potassium, and sodium. The other minerals needed in minute quantities, called “trace minerals,” have specific functions within the body; they include cobalt, copper, iodine, iron, manganese, molybdenum, and selenium. The minerals reviewed in this chapter are those that are most commonly associated with deficiencies and their resulting adverse health conditions.

Calcium

Calcium is essential not only for healthy bone and teeth formation, but also for nerve conduction, muscle contraction, and blood clotting. Because more than 30% of women in the United States older than 50 years are at risk for bone fracture due to osteoporosis,[35] maximizing peak bone mass through adolescence and early adulthood is essential. Unfortunately, adolescent girls have the lowest total intakes of calcium, 918 mg ± 30 mg per day, with only 13% to 15% meeting the daily requirement of 1300 mg from both diet and supplements.[36] In addition to calcium’s role in building bone mass during adolescence and early adulthood, it is important that women obtain adequate amounts of calcium throughout the lifespan to maintain bone mass. Many older women meet their calcium needs by taking a daily supplement or calcium carbonate antacids. **Table 7-7** lists common food sources of calcium.

Table 7-7 **Examples of Sources of Calcium**

	Serving Size	Calcium (mg)		Serving Size	Calcium (mg)
Dairy			**Fruits and Vegetables**		
Yogurt, fruit variety, nonfat	8 oz	345	Orange juice, calcium fortified	6 oz	200
Milk, calcium fortified	8 oz	400	Orange, navel	1	52
Milk, 1%	8 oz.	300	Raisins, seedless	2 oz	28
Lactaid milk	8 oz	300	Broccoli, steamed	½ cup	31
Cheese, American	1 oz	174	Collards, fresh cooked	1 cup	268
Cheese, cheddar	1 oz	204	Collards, frozen, boiled	½ cup	179
Cheese, mozzarella	1 oz	222	Spinach, fresh	1 cup	245
Cottage cheese, nonfat	8 oz	125	Spinach, chopped frozen	½ cup	145
Ice cream	8 oz	176	Kale, fresh, cooked	1 cup	94
Milk, nonfat dry powder	2 Tbsp	104	Mustard greens, fresh	1 cup	165
			Rhubarb, fresh diced	1 cup	105
			Blackstrap molasses	2 tsp	118
Seafood			**Nuts, Seeds, and Beans**		
Clams, canned	3 oz	78	Almonds	1 oz	76
Salmon, pink, canned	3 oz	240	Tahini (sesame paste)	1 Tbsp	64
Sardines, canned in oil	2 oz	216	Beans, white, canned	1 cup	191
Ocean perch	3 oz	29	Beans, navy, canned	1 cup	123
Halibut, baked	3 oz	51			
Shrimp (shelled)	3 oz	60			
Soy Products			**Cereal**		
Tofu, firm prepared with calcium sulfate	4 oz	253	Oatmeal, instant	1 packet	105
			All Bran (Kellogg)	½ cup	121
Tofu, soft	4 oz	150	Cheerios (General Mills)	1 cup	112
Soy milk	8 oz	300	Total (General Mills)	¾ cup	1000
			Tortilla, corn, 6 in.	1	45
			Bread, whole wheat	1 slice	30
Antacids					
Tums, regular	1	200			
Tums, extra strength	1	300			

Based on U.S. Department of Agriculture. USDA National Nutrient Database for Standard Reference, Release 28. 2016. Available at: https://www.ars.usda.gov/northeast-area/beltsville-md/beltsville-human-nutrition-research-center/nutrient-data-laboratory/docs/usda-national-nutrient-database-for-standard-reference/. Accessed May 11, 2017.[23]

Iodine

Iodine is essential for normal thyroid function, and deficiency of this mineral can lead to an enlargement of the thyroid gland known as goiter. Dietary iodine is obtained primarily from iodized salt, fish, and seaweed. From the late 1970s to 2000, iodine levels in the U.S. population declined by 50%, though levels have since stabilized.[26] This decline is probably due to a combination of decreased intake of salt, fish, and possibly dairy products, which

collectively account for 60% of iodine intake. The currently popular exotic salts, such as Fleur de Sel and pink salt, are not iodized. The recommended daily intake of iodine is 150 mcg, which would result in a urinary iodine level of 100 mcg/L. The most recent data show that, in general, the U.S. population obtains sufficient iodine, but 37% of childbearing-age women have urinary iodine levels below this threshold, with the highest proportion of such deficiency occurring among non-Hispanic blacks.[26]

Iron

The most common nutritional deficiency globally is iron deficiency, which disproportionally affects women. Iron is essential to the production of hemoglobin and, therefore, for the transport of oxygen. Red blood cells contain 80% of the body's iron, where it can be measured as hemoglobin. Iron not in red blood cells is stored as ferritin; transferrin is a protein needed for this stored iron to be transported in the circulation. Women who are not pregnant are at risk of anemia because of iron loss during menstruation, coupled with inadequate iron intake or decreased iron stores associated with close spacing of pregnancies. Special populations of women at higher risk for iron-deficiency anemia include pregnant women and those with malabsorption, such as women with inflammatory bowel disease and women who have undergone bariatric surgery. Slightly less than 1 mg of iron is lost from the intestines daily; this daily iron loss is more than 1 mg among women with *Helicobacter pylori* infection, malaria, or infection with intestinal parasites.

Two forms of iron are found in food: heme and non-heme. Heme iron found in animal products is absorbed more efficiently than non-heme iron. For example, 20% to 30% of heme iron is absorbed, compared to 2% to 10% of non-heme iron. **Table 7-8** lists some common dietary sources of iron. Iron uptake also varies according to the specific needs of a woman's body. If a woman has adequate iron stores, only approximately 10% of ingested iron is absorbed. In the presence of iron deficiency, however, as much as 40% of ingested iron can be absorbed.

Table 7-8 **Examples of Dietary Sources of Iron**

Source	Serving Size	Iron (mg)
Breakfast cereals: varies by brand	1 cup	5–18
Prune juice	1 cup	10
Chicken liver	3 oz	8
Beef liver	3 oz	7
Cream of wheat	½ cup	7
Oysters	6	6
Sunflower seed kernels	2 oz	4
Prunes	10	4
Beans: lentils, kidney, garbanzo	2.3 cup	2–3
Beef	3 oz	3
Pork	3 oz	2

Sesame seeds	2 oz	2
Leafy green vegetables, cooked	1 cup	2
Molasses, blackstrap	1 Tbsp	2
Tortilla, wheat/corn, 6 in.	1	1.8/0.7
Chicken	3 oz	1
Plantains, cooked	1 cup	1
Peanut butter	2 Tbsp	0.6

Based on U.S. Department of Agriculture. USDA National Nutrient Database for Standard Reference, Release 28. 2016. Available at: https://www.ars.usda.gov/northeast-area/beltsville-md/beltsville-human-nutrition-research-center/nutrient-data-laboratory/docs/usda-national-nutrient-database-for-standard-reference/. Accessed May 11, 2017.[23]

Some foods or supplements can enhance or inhibit iron absorption, and these items should be discussed when counseling women. Iron uptake enhancers include animal muscle from meat, chicken, or seafood; foods containing vitamin C and, in populations deficient in vitamin A, vitamin A; and fermented vegetables and sauces (e.g., sauerkraut, kimchee, and soy sauce). Iron uptake inhibitors include foods containing phytates (e.g., whole grains, oats, bran, nuts, spinach), phenols (e.g., tea, especially green tea; coffee; cocoa; and red wine), calcium, and soy proteins. Iron absorption also is decreased in the presence of antacids. *Pica*—a condition characterized by persistent and compulsive eating of non-nutritive substances, including excessive consumption of ice—can be either a symptom of anemia or a contributing cause. Frequently, the correction of the anemia will eliminate the pica.

The loss of iron stores is followed by anemia. Adequacy of iron stores can be measured in several ways. The most commonly available tests assess characteristics of the red blood cells, such as hemoglobin level, and perform a peripheral smear to characterize the morphology of the RBCs. However, biochemical tests of serum ferritin and transferrin saturation measure earlier stages of iron depletion prior to actual iron deficiency as evidenced by a low hemoglobin level. The primary laboratory and clinical findings with differing states of anemia are discussed in the *Common Conditions in Primary Care* and *Pregnancy-Related Conditions* chapters. Although iron-deficiency anemia typically is associated with pale (hypochromic) and small (microcytic) RBCs, changes in RBC morphology occur relatively late in the process.

According to NHANES data, the overall prevalence of iron-deficiency anemia in women who reside in the United States is 11% among nonpregnant women and twice that rate among pregnant women. However, this estimate is based on measures of iron stores, such as serum ferritin or transferrin saturation. If hemoglobin level (the most widely available measurement method) is used to assess for iron-deficiency anemia, the incidence is lower, approximately 6%.[37] Differences in the use of vitamin supplements by race/ethnicity appear to account for the increased prevalence of anemia among blacks and Hispanics. Approximately 10% to 13% of older women have been found to have anemia, with elderly black women having at least twice the rate of elderly white women.[38]

Women diagnosed with iron-deficiency anemia who increase their dietary consumption of iron-rich foods and avoid taking antacids 2 hours before or 4 hours after meals may correct the problem nutritionally. The other option for remedying this deficiency is iron supplementation.

After the addition of 30 mg to 100 mg of elemental iron taken daily, an increase in the reticulocyte count should be noted within 2 weeks and an increase in the hemoglobin noted within 3 to 4 weeks.

Ferrous salts are the first-line choice for oral iron supplementation for the treatment of women with iron-deficiency anemia. Other types of iron supplements are not more effective, do not appear to have a more benign side-effect profile, and cost approximately four times as much as ferrous salts. Because a maximum amount of absorption is possible at one time, divided doses are preferable. Also, separating administration of iron from ingestion of a multivitamin can maximize absorption.

Side effects of iron therapy, such as nausea, bloating, abdominal cramping, and constipation, are proportional to the dose. Frequently women will discontinue therapy due to these side effects. Women should be counseled about the potential side effects and measures they can take to lessen them. An alternative to daily iron supplementation is taking 120 mg of elemental iron once or twice weekly. It is not recommended that women double their multivitamin supplement in an effort to obtain added iron, as this approach could result in excessive levels of fat-soluble vitamins.

Table 7-9 lists types of iron supplements and the amounts of elemental iron in each dose. Enteric-coated and slow-release iron formulations are not recommended because iron is absorbed in the small intestine and these products provide iron that is excreted unabsorbed. A key safety issue is that iron supplements—particularly those prescribed prenatally—are one of the leading causes of childhood poisoning.[39] All women should be cautioned to keep their medications out of the reach of children.

Table 7-9 Iron Supplements

Iron (Fe) Group	Generic Name (Brand)	Dose Elemental Iron (mg)
Iron Salts		
Tablets	Iron sulfate (FeoSol)	325 (60)
	Iron fumarate (Feostat, Hemocyte)	200 (66)
	Iron gluconate (Fergon)	300 (35)
Liquid	Iron sulfate elixir (FeoSol Elixir)	22 (44) per 5 mL 300 (60) per 5 mL
	Iron gluconate + herbs (Floradix or Floravital—gluten free)	(10) per 10 mL—higher absorption rate
Polysaccharide Iron/Carbonyl Iron		
Tablets	Carbonyl iron (FeoSol caplets)	50 (45)
	Polysaccharide iron (Ferrex-150)	150 (150)
Liquid	Polysaccaride-iron elixir (Niferex Elixir)	100 (100) per 5 mL

Role of Vitamin and Mineral Supplementation

Ideally, micronutrients should be obtained through intake of a variety of fruits and vegetables each day—at a minimum, five servings per day—and exposure to adequate sunlight. Unfortunately, fewer than one in four individuals in the United States meets the daily fruit recommendation and only 13% meet the recommendation for daily intake of vegetables.[40]

Use of supplements is not a substitute for a healthy diet. While noting that there is little harm in taking a daily multivitamin supplement, the U.S. Preventive Services Task Force (USPSTF) does not currently recommend routine multivitamin/mineral supplementation for the general population for the prevention of chronic diseases such as cardiovascular disease. However, this recommendation does not apply to populations at risk for deficiencies, including women who have undergone gastric bypass and those who have malabsorptive conditions or take medications that can result in lower absorption. As discussed in the section "Dietary Patterns," limited supplementation should be at least considered by vegetarians, and especially by vegans. However, consumption, by anyone, of large doses of iron and of vitamin supplements, particularly fat-soluble vitamins, can potentially be hazardous.

Although studies of multivitamin supplements have not shown a benefit in preventing chronic disease, several expert panels have highlighted the deficiencies in these studies. First, these studies use a non-uniform definition of a "multivitamin"—that is, differences in the number of vitamins included and the presence or absence of specific minerals. Second, most studies have been cross-sectional and relied solely on self-reports, which may be not be accurate—especially when recalling intake many years in the past.

The longitudinal Physicians Health Study did find multivitamin use was associated with a decrease in epithelial cancer and cataracts.[41] A recent systematic review concluded that, at the very least, multivitamin/mineral supplements do not seem to cause any harm.[42] Therefore, in general, taking a multivitamin/mineral supplement "just in case" seems to do no harm although it may not be money well spent.

Dietary Patterns

When discussing nutrition, a healthcare provider will probably be of most value to a woman by emphasizing healthier dietary patterns that are relevant at any weight and are applicable throughout her life course. The word “diet” is often interpreted by many laypersons too narrowly—that is, as meaning “a weight-loss diet.” It may be helpful, therefore, to use a less-familiar term such as “dietary pattern” with a woman when counseling her on nutrition. The USDA highlights three food patterns that are recommended as healthy dietary patterns: (1) the United States 2010 Dietary Guidelines, which are similar to the Healthy Eating Pyramid; (2) the Mediterranean-style eating pattern; and (3) the healthy vegetarian eating pattern.

Healthy Eating Pyramid and Healthy Eating Plate

The Healthy Eating Pyramid was developed by faculty in the Department of Nutrition at Harvard School of Public Health and adopted by the United States Department of Agriculture (USDA) in 1991.[43] The pyramid is a graphic that illustrated recommended quantities of food intake per day in different food categories.[43] The bottom of the pyramid and bulk of one’s diet is made of vegetables, fruits, health fats and oils, and whole grains. The top of the pyramid are those foods eaten sparingly such as red meat, butter, processed meat, refined grains, sugary drinks, and salt.[43]

In 2011, the USDA replaced the Healthy Eating Pyramid with The Healthy Eating Plate graphic (**Figure 7-1**).[45] The USDA Healthy Eating Plate graphic has the advantage of being a simple illustration but it does not convey important diet choices recommended by the Harvard researchers who created the pyramid (e.g., whole grains are better than any grains). Nonetheless, the USDA Healthy Eating Plate is an easy teaching tool that can be used with the Healthy Eating Pyramid and key principles outlined in **Table 7-10** in discussions about recommended dietary patterns.[8,43-45] The Resources section at the end of this chapter has links to the Harvard versions of the Healthy Eating Pyramid and Healthy Eating Plate for more detail about their use in clinical practice. Essentially, women can see that half their plate should be occupied by vegetables and fruits (minimum of 5 servings per day), one-quarter with whole grains, and one-quarter with protein, with the addition of a healthy unsaturated fat. It is also important to discuss fluid intake, especially if it is a source of high-glycemic, high-calorie intake, such as the empty calories found in sugared beverages.

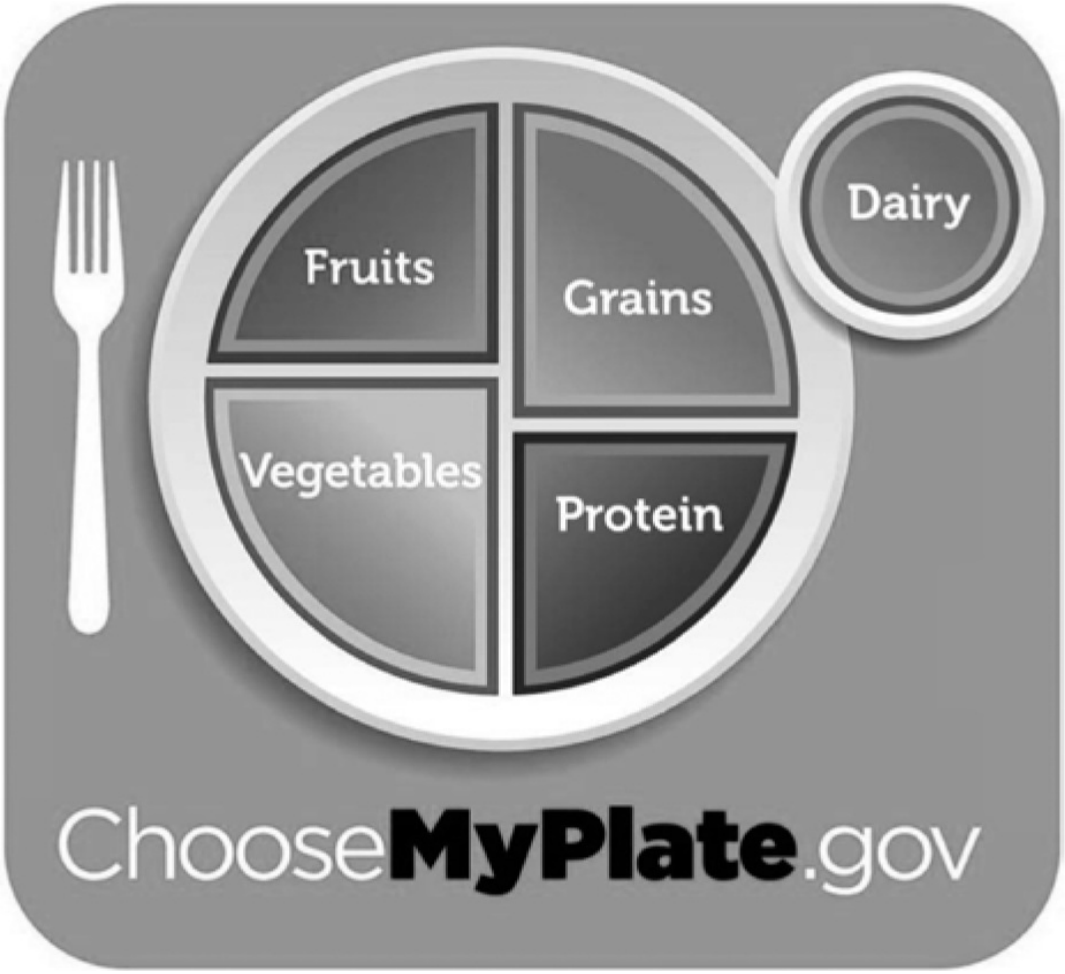


Figure 7-1 The Healthy Eating Plate.
Reproduced with permission from U.S. Department of Agriculture. MyPlate. Available at: https://www.choosemyplate.gov/MyPlate. Accessed August 30, 2017.[45]

Table 7-10 **Key Principles in the Healthy Eating Pyramid**

1. Choose healthy fats instead of unhealthy fats.
   - Avoid trans fats; choose monounsaturated and polyunsaturated fats.
2. Choose slowly digested carbohydrates over highly refined ones.
   - Emphasize whole fruits, vegetables, beans, and nuts.
   - Choose whole grains, such as brown rice, bulgur, barley, quinoa, and wheat berries.
3. Choose proteins that are plant based at least half of the time, and choose fish, eggs, and poultry the rest of the time, eating little red meat.
4. Eat large amounts of fruits and vegetables—a minimum of 5 servings per day.
5. Choose low-calorie hydration—water is best.
   - Drink coffee and tea in moderation.
   - If milk is part of the diet, choose low-fat types.
   - Limit juice to one small glass per day.
   - Avoid high-sugar drinks.

- Limit alcohol intake to one glass per day.

6. Meet daily recommendations for vitamins and minerals.
7. Get daily exercise—30 minutes of brisk walking daily.
   - Calories expended are important to maintain a healthy weight.

Modified with permission from Skerrett PJ, Willett WC. Essentials of healthy eating: a guide. *J Midwifery Womens Health.* 2010;55(6):492-501.[9] © 2010, with permission from Wiley.

The dietary pattern promoted in the Healthy Eating Pyramid and Healthy Eating Plate is similar to two other dietary patterns that have been associated with decreases in chronic diseases, especially cardiovascular disease: the Mediterranean diet and the DASH (Dietary Approaches to Stop Hypertension) diet.

Mediterranean Diet

The Mediterranean diet emphasizes eating primarily vegetables, grains, beans, nuts, and seeds, with a serving of fish, poultry, or egg, and small amounts of cheese or yogurt, and obtaining 40% of calories from fats, primarily olive oil.[46] Similarly, the DASH diet emphasizes 8 to 10 servings of fruits and vegetables per day and choosing low-fat/nonfat protein sources; it also limits salt consumption and limits fats to 27% of calories. The DASH diet places less emphasis on the type of fat, except to limit saturated fat to less than 7% of calories. A variant of the DASH diet is the OmniHeart (Optimal Macronutrient Intake Trial to Prevent Heart Disease) diet, which somewhat reduces carbohydrate intake relative to the DASH diet, compensating for the caloric loss by substituting either protein or unsaturated fat. The investigators who studied the OmniHeart diet documented further lowering of blood pressure, further improved lipid levels, and further reduced cardiovascular risk compared to the already-healthier standard DASH diet.[47] Finally, the "portfolio" dietary pattern created by researchers at the University of Toronto is primarily a vegetarian diet that appears to reduce LDL levels, but without lowering HDL.[48]

Vegetarian Diets

A person who eats a vegetarian diet does not eat meat, poultry, or fish. A vegetarian diet is subclassified as *lacto-ovo-vegetarian*, which includes dairy products and eggs, or *vegan*, which does not contain any animal products. Both types of vegetarian diet can be nutritionally sound throughout the lifespan, including during pregnancy and lactation. Because vegetarian diets are generally lower in saturated fat and higher in fruits, vegetables, and whole grains, this dietary pattern may reduce the risk of some chronic diseases. Population studies of vegetarians have found a lower incidence of obesity, hypertension, type 2 diabetes, and ischemic heart disease and lower rates of low-density lipoprotein cholesterol levels compared to nonvegetarian populations.[49] The incidence of cancer varies widely, however. Although overall the risk of any type of cancer might be lower in persons who have a vegetarian diet, the data on this point are inconclusive.

When consuming a vegetarian diet, attention must be paid to some nutrients that are not found

in large quantities in vegetarian food sources such as vitamin B_{12}, vitamin D, calcium, and n-3 fatty acids. Essential amino acid needs can be met by eating a wide variety of plant foods.

Vitamin B_{12} deficiency is the most common nutrient deficiency in persons who eat a vegetarian diet. Unfortunately, the foods in a typical vegan dietary pattern cannot guarantee uptake of sufficient dietary vitamin B_{12} for normal functioning. Thus, vegans in particular should assiduously supplement their vitamin B_{12} intake with vitamin B_{12}–fortified foods and supplements. Such fortified foods can include vitamin B_{12}–fortified nutritional yeasts, vitamin B_{12}–fortified breakfast cereals, and vitamin B_{12}–fortified meat alternatives.

No food available to a vegan contains adequate vitamin D and calcium. Fatty fish, eggs, and fortified milk are the principal food sources of vitamin D, with sunshine on human skin being the other significant "source" of vitamin D. Therefore, vegans should consider vitamin D supplementation. Vitamin D_3 seems to be the best source of this nutrient.[50] It should be noted that vitamin D_3 may be derived from animals, so a committed vegan might search for a vegan form of vitamin D_3. Vegans appear to have a 30% increased risk of bone fracture risk,[51] so they need to consciously choose fruit and plant sources of calcium. **Table 7-7** lists non-animal sources of calcium. Vegans can also consider a calcium supplement.

It is recommended that to increase n-3 fatty acid intake, vegans and vegetarians should consider supplements containing eicosapentaenoic acid (EPA) and/or docosahexaenoic acid (DHA).[52] Cardiovascular disease appears to be less prevalent among vegetarians, however, even though vegetarians' n-3 fatty acid concentration is often lower than that of omnivores.

Gluten-Free Diet

The number of people choosing a gluten-free diet has increased rapidly in the United States in the last few years, with the latest estimates suggesting that approximately 17% of people living in the United States adhere to a gluten-free diet, including 1.8 million people with celiac disease and 2.7 million people without celiac disease.[53] It is clear that for those persons with celiac disease, adherence to a gluten-free diet decreases inflammation and symptoms of the disease. Although celiac disease and wheat allergy sensitivity are clearly defined disorders related to gluten intake, the existence of non-celiac gluten sensitivity syndrome remains controversial, despite being the subject of several consensus conferences.[54,55] Nevertheless, the midwife should be aware of potential dietary concerns related to a gluten-free diet. Women consuming gluten-free products are at risk for excessive intake of high-glycemic carbohydrates and saturated fat and deficiencies of fiber, vitamin C, vitamin B_{12}, folic acid, and vitamin D as well as calcium, zinc, and magnesium.[55] However, it should be noted that gluten-free pseudo-cereals, such as quinoa and amaranth, have twice the mineral content (including calcium and iron) of traditional cereals. Therefore, the principle of opting for whole-grain forms of food, instead of processed foods, is an important message to communicate.

Women with celiac disease are at increased risk for poor pregnancy outcomes and osteoporosis. The pregnancy-related risks include prematurity, fetal growth restriction, small-for-gestational-age newborns, as well as stillbirth.[56] Women whose disease is well controlled

have a lower risk than those whose disease is not well controlled. Therefore, preconception counseling about a gluten-free diet in these women has the potential to improve their pregnancy outcomes. Additionally, women with celiac disease are at risk for low bone density that may or may not normalize with a gluten-free diet.[56] Emphasizing adequate intake of calcium and vitamin D or supplementation, starting in adolescence, is important for long-term health.

Weight Management Counseling and Dietary Patterns

Numerous morbidities are associated with obesity: metabolic disorders such as diabetes and polycystic ovary syndrome, cardiovascular disease, osteoarthritis, and malignancies. Thus it would seem clear that midwives should assist women with obesity and other weight management concerns. Consistent weight management is a very challenging task for many individuals, however, and the best evidence shows that the kind of low- to moderate-intensity counseling that a busy clinician can provide is not the most effective approach.[57] In addition, community resources such as those that provide a high-intensity comprehensive lifestyle intervention, entailing a minimum of 14 sessions over 6 months, may be unavailable. When a midwife has limited time, direct referral to a dietician or a specific weight management program may be most helpful. The midwife should seek interventions that are short, precisely targeted, easily administered, understandable to women, and validated.

Dietary Assessment

Assessment customarily begins with a calculation of the woman's body mass index (BMI). Web-based BMI calculators can readily be found on the Internet (sources are listed in the Resources section at the end of this chapter), but the calculation involves only simple arithmetic and can be done by hand using the following formula:

$$BMI = [(\text{weight in lbs}) \div (\text{height in in})^2] \times 703 \text{ or}$$
$$BMI = (\text{weight in kg}) \div (\text{height in m})^2$$

For most individuals, including adolescents, a BMI in the "normal" range for their gender is associated with a reduced risk of obesity-related morbidities. However, BMI is not as accurate for postmenopausal women, who have typically lost some muscle mass and, therefore, might have a "normal" BMI but be at higher than normal risk. This assessment technique is also clearly inaccurate for body builders or elite athletes, who have much less body fat than average and, therefore, might have a "high" BMI but actually be at normal or even reduced risk.

Waist circumference in women more than 35 inches (88 cm) has been found to be an additional indicator of increased disease risk. Data reveal that an even better measure to identify persons at risk for cardiovascular disease and diabetes is a waist circumference-to-height ratio of more than 0.5.[58] For example, a waist measure of 37 inches and a height measurement of 65 inches would translate into a ratio of 0.57. The waist-to-height ratio identifies abdominal adiposity in the many different populations in which it has been tested, and the cut-off ratio of 0.5 is applicable in both men and women and in both young and old. The ease, usefulness, and relative accuracy of the waist-to-height ratio argue strongly for making waist circumference measures a regular part of every health assessment examination.

The 2012 weight management and treatment algorithm developed by the National Heart, Lung, and Blood Institute (NHLBI) uses BMI as the decision indicator and is available on the Internet.[57] Within this algorithm, weight management counseling is not advised if a woman is only overweight but lacks any risk factors for diabetes or cardiovascular disease. However, if

she has (1) two risk factors for diabetes and/or cardiovascular disease and (2) a BMI greater than 25 kg/m^2 or waist circumference larger than 35 inches, or a BMI greater than 30 kg/m^2, then she should be asked about her desire to lose weight. If she is ready to make a behavior change, then referral to a comprehensive lifestyle program is recommended, although the counseling may also be undertaken by the provider.

If weight management will be helpful for the woman, the next step should be a brief assessment of her readiness to change. It may be helpful to conceive of the process of weight management as providing *cues* rather than providing *cures*. If and when a woman is receptive, she will receive and welcome a supportive and factual weight management conversation, and be able to consider strategies proposed by the midwife as cues to her own behavior and incorporate them into her life. If a woman is not ready to change, attempts to intervene from a healthcare professional will seem irrelevant, irritating, or even threatening to her as an advertisement for a product or behavior in which she has no interest.

Accordingly, a midwife seeing a woman who meets the NHLBI criteria for weight-loss management might use a few brief questions to obtain a general concept of where the woman is in her journey using the "stages of change" model. A sample question sequence that is supportive, brief, and factual for the stages of change model is found in **Table 7-11**.

Table 7-11 **Sample Question Sequence for the "Stages of Change" Model**

"There's no right or wrong answer to this question, but for me to better understand where you are right now, have you been thinking about losing weight?"
[In response to either yes or no] "That's fine." (That is, briefly give support).
[If yes] "Have you been thinking about a plan?"
[If yes] "Is this something you intend to do soon, like within the month, or is this more of a long-term goal?"
"Would you like me to be involved in this process with you, and be a support for you, no matter what happens?"

Although it would be ideal to have women complete a 24-hour or 3-day food diary prior to their healthcare visit, and then have the time for the woman and the midwife to analyze these data together for macronutrient and micronutrient content, the reality is that busy midwives do not usually have the time to do this kind of counseling. Another option is to ask all women being seen for an initial or annual visit to complete a "Starting the Conversation About Healthy Eating" questionnaire, which incorporates questions to assess readiness to change, along with seven questions to assess dietary patterns (**Figure 7-2**).[7] Three response categories exist for each question; they are scored 0, 1, or 2, starting from left to right, culminating in a range of scores from 0 to 14. Higher scores indicate a poorer dietary pattern. The form includes practical suggestions for each question on how this aspect of eating can be improved. It also includes a section for making goals for changing if a woman is at that stage of change.

ARE YOU READY TO WORK ON **HEALTHY EATING** DURING YOUR PREGNANCY?
___I'm ready to make some changes and I would like help
___I'm not sure if I'm ready to change the way I eat but I would like to start the conversation
___I'm not ready to change the way I eat at this time

HOW WELL DO YOU EAT?	TIPS TO HELP YOU EAT WELL
How many times a week do you eat food that is fried or high in fat? ☐ Less than 1 ☐ 1–3 ☐ 4 or more	**Eat less fast food.** • If you eat fast food, order grilled chicken, a salad, or plain burger.
How many servings of fruit and vegetables do you eat each day? ☐ 5 or more ☐ 3–4 ☐ 2 or less	**Aim for 5 or more servings of fruits and vegetables a day.** • Add a fruit and a vegetable to each meal. • Raw fruits and vegetables are easy snacks.
How many regular sodas, juice, or glasses of sweetened beverages do you drink each day? ☐ Less than 1 ☐ 1–2 ☐ 3 or more	**Beverage calories add up.** • Drink water throughout the day. • If you drink milk, switch from whole or 2% to 1% or skim. • Limit soda and other sweet drinks to one or less per day.
How many times a week do you eat beans (like pinto or black beans), chicken, or fish? ☐ 3 or more ☐ 1–2 ☐ Less than 1	**Eat more beans, chicken, and fish.** • Beans are a great substitute for meat. • Eat up to 12 oz. of low-mercury fish weekly like canned light tuna, salmon, or pollock. • Eat baked, broiled, or grilled chicken and fish. • Avoid high mercury fish like shark, swordfish, king mackerel, and tilefish.
How many times a week do you eat regular (not low-fat) snack chips or crackers? ☐ 1 or less ☐ 2–3 ☐ 4 or more	**Hold the chips.** • Try popcorn, but limit oil, butter, or salt. • Try nuts such as almonds or walnuts, but limit your serving to 2 Tablespoons.
How many times a week do you eat desserts and other sweets? ☐ 1 or less ☐ 2–3 ☐ 4 or more	**Be smart with your sweet tooth.** • Eat smaller amounts of dessert. • Try desserts with less fat and calories like fruit, sherbet, or angel food cake.
How many butter, lard, and animal fat (visible fat in red meat, chicken skin) do you eat? ☐ Very little ☐ Some ☐ A lot	**Cut back on animal fats.** • Use a small amount of plant-based oils and transfat free margarines. • Season foods with spices and fresh herbs.

0 × (____) + 1 × (____) + 2 × (____) = Total Score _____

IF YOU FEEL THIS WAY	TRY THESE THINGS
Healthy food costs too much.	**Eat well can save you money.** • Eat less meat and more beans. • Eat canned or frozen fruits and vegetables. • Eat at home instead of going out.
Healthy food doesn't taste as good as junk food.	**Don't give up your favorite foods—just eat smaller amounts.** • Try new foods and recipes.
I eat when I'm bored, tired, angry, or depressed.	**Find something else to distract you.** • Work on a hobby, call a friend, go for a walk. • Keep only healthy snacks around.
It's hard to be healthy when I eat out.	**Avoid all-you-can-eat places and restaurants that don't offer healthy options.** • Order grilled or low-fat sandwiches and salads instead of fried foods. • Ask for low-fat dressing on the side. • Ask for half portions, share with a friend, or bring leftovers home.

Figure 7-2 Starting the conversation about healthy eating.
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I eat too much at social events.	**You can still eat healthy at social events.** • Eat a healthy snack before you go. • Choose a few things to eat. • Bring healthy dishes to potlucks.
I eat too much when I'm cooking or cleaning.	**Don't just eat because it is there.** • Chew gum or a toothpick. • Ask someone else to put away leftovers while you wash dishes.
I tend to skip regular meals, but snack in front of the TV and throughout the day.	**Make time for regular meals.** • Sit down at the table and eat healthy meals with friends, family, and/or co-workers. • Pack lunch and snacks to take to work or for travel.

Making a plan
What goal(s) can you set for yourself now? **Before my next visit, I am going to:** ___ Eat fried foods less often ___ Aim to eat 5 or more fruits and vegetables per day ___ Eat smaller portions ___ Instead of regular sodas, juice, and sugar sweetened beverages, drink water or skim milk ___ Keep healthy snacks around ___ Other _____

A more comprehensive and validated instrument is the easy-to-use "Rapid Eating and Activity Assessment for Patients" (REAP), which contains 31 questions. The accompanying "REAP Physician Key for Diet Assessment and Counseling" provides clear directions for further assessment, treatment, and counseling based on responses to each of the questions.[59] All behavioral changes must be made by the woman herself, in a manner that she can freely accept and commit to. Weight is a delicate subject for many women, and repeatedly offering warm support rather than remedies will build trust. When specific plans and strategies seem relevant to discuss, offering a woman cues to become more active in her weight management rather than imposing cures should be the goal. Cures are imposed; cues are freely accepted or freely put on hold. Finally, if the woman will be seeing the same healthcare provider again, this provider can offer continuing support. It is important for the midwife to take into account and be supportive of weight management failures as well as successes, as both may occur. Repeated, honest, and realistic offers of support "no matter what happens" can help build the needed trust.

While a weight-loss intervention can be part of the nutritional choices made by a woman for herself (in consultation with her healthcare provider), the more fundamental issue remains adopting one of the healthier dietary patterns discussed previously in this chapter. The healthiest and safest weight-loss "diets" usually involve following the same healthier dietary patterns that would be recommended for anyone, with the modification of eating smaller portions, so as to create a daily calorie deficit in the range of 500–750 kcal/day. A minimum

weight-loss goal to improve an individual's health has been shown to be 5%. A woman attempting a weight loss should be offered a follow-up assessment with the midwife or another clinician after 6 months. At the 6-month visit, the clinician can evaluate weight loss, review behaviors the woman has attempted to change, and reinforce these efforts. It is important to initiate plans with the woman to maintain her weight loss, since weight loss usually plateaus at this time. To reiterate, a midwife can remind a woman that learning and adopting a healthier dietary pattern will be valuable to her throughout her life, whatever her weight, and whatever her health and life goals.

Women who are unable to meet a healthy weight goal with diet and exercise, especially after attempting intensive comprehensive lifestyle programs, may be candidates for pharmacologic therapy or referral for possible bariatric surgery. Weight-loss drugs work by suppressing appetite, limiting fat, or altering neurotransmitters such as serotonin and catecholamines that regulate appetite and food intake in the central nervous system. Currently there are five FDA-approved drugs for long-term weight loss, listed here in order of their relative weight loss results from smallest to largest mean amount of weight lost:

1. Orlistat (Xenical or Alli, over the counter at a lower dose) is a lipase inhibitor and the oldest drug on the market.
2. Lorcaserin (Belviq) is a Schedule IV drug due to its potential hallucinogenic properties.
3. Phentermine and topiramate combination therapy (Qsymia) is contraindicated for use during pregnancy and is also a Schedule IV drug.
4. Liraglutide (Saxenda), a drug also used for type 2 diabetes, is available only as an injectable.
5. Naltrexone/bupropion (Contrave) is the newest option on the market. Expected weight loss over 12 months ranges from 6 to 9 kg (13–20 pounds).[60]

Women with BMIs greater than 40 kg/m^2, and women with BMIs between 35 and 40 kg/m^2 who have a comorbidity such as hypertension, hyperlipidemia, diabetes, or obstructive sleep apnea, should be offered a referral to a bariatric surgeon in addition to a comprehensive lifestyle program.[56] Bariatric surgery has been shown to be the most successful strategy for weight loss for this population of women.[61] Additionally, recent evidence demonstrates that women who are obese and experiencing infertility (undergoing assisted reproductive technologies) increase their ability to conceive and have a live birth by one-third following bariatric surgery compared to attempts prior to surgery.[62]

Nutrition Across the Lifespan

Nutritional needs change significantly over the course of a woman's lifetime. A review of nutrition and recommended nutrition and counseling for women during pregnancy and nutrition specific to lactating women are addressed in the *Prenatal Care* and *Breastfeeding and the Mother–Newborn Dyad* chapters. This chapter focuses on nutrition during non-reproductive phases in a woman's life.

Nutrition During Adolescence

Adolescence is a period of rapid physical growth and, therefore, high nutritional need. During adolescence, females gain 40% of their skeletal mass and 50% of their body weight. Inadequate nutrition, especially calcium intake, can compromise their peak bone mass. Unfortunately, the diet pattern of adolescents tends to be poor due to the consumption of calorie-dense, nutrient-poor foods; it is also a time when teenage girls typically stop drinking milk. Of perhaps greatest concern is the increased rate of obesity among U.S. children and adolescents. Today, on average, 20% of girl teens are obese, and such an increase in body fat has both health and social implications.[63]

In recent decades, the age of onset of puberty has decreased. The reasons for this trend may include increased exposure to environmental pollutants and chemicals, stress, and increased body fat in childhood. For every one-point increase in BMI, menstruation occurs 1 month earlier.[64] Stage 2 or greater breast development is now not uncommon at ages 7 and 8 years, with its rate ranging from 18% to 43%; black girls have a higher rate of this condition than white girls.

Beyond body fat, type of food consumed plays a role in the development of puberty. Data from national surveys show that the onset of puberty is delayed approximately 7 months in children consuming large amounts of plant protein, whereas those with a high intake of animal protein enter puberty approximately 7 months earlier.[64] Also, greater isoflavone intake—which is found in soy products, chickpeas, and lentils—delays breast development by 7 months. Earlier onset of puberty can pose significant self-image and social problems for young children, who may be seen as more socially and emotionally mature than they actually are.

By developing a young girl's trust and conducting the same kind of sensitive assessment as previously outlined to determine whether she is ready to make at least small alterations in her diet or exercise patterns, a midwife can play a role in helping her establish healthier diet patterns for life. Problem-solving barriers to change for an adolescent must include consideration of where and when food is consumed, especially if in the presence of peers, and the adolescent's ability to control the kinds of food purchased and prepared at home. The two nutrients essential at this phase of development are calcium and iron, as the intake of both of these nutrients tend to be low in adolescents.

Nutrition, the Menstrual Cycle, and Fertility

Nutrition also affects, and is affected by, the menstrual cycle. Studies suggest that caloric

intake varies during the menstrual cycle, peaking once in the luteal phase and once in the follicular phase, and reaching a nadir during menses.[65] These changes are theorized to coincide with cyclical fluxes in a woman's basal metabolic rate that are affected by hormones. Women also appear to increase their intake of carbohydrates and fat during the luteal phase, and their intake of protein and fat in the follicular phase, as compared to the menstrual phase. Women with premenstrual syndrome have similar intakes as women without this condition, but increase their intakes of non-milk sugars and alcohol in the luteal phase.[53] Women with very low body fat become anovulatory.

According to the Centers for Disease Control and Prevention, 1 million married women in the United States are infertile, with six times that number having a reduced fecundity.[66] Women at the extremes in body weight are more likely to have reduced fertility. Therefore, aiming for a healthy body weight is important for improving fertility. Data from the Nurses' Health Study indicate that for women with high BMIs, losing 5% to 10% of body weight may improve ovulation, especially if regular exercise is incorporated into the regimen.[67] Lean women who exercise strenuously should reduce this activity to a moderate level to improve their fertility. Other specific dietary patterns associated with improved ovulatory fertility from the Nurses' Health Study—beyond those emphasized in this chapter, such as eliminating trans fats and eating low-glycemic carbohydrates—include choosing more plant-based proteins, and temporarily trading nonfat or low-fat dairy products for the full-fat versions.

Nutrition and the Mature Woman

As women age, they may find that their abdominal fat and weight increase. Although this change in perimenopausal women is due to a combination of genetics, age, and physical activity, some evidence indicates that loss of estrogen contributes to the redistribution of fat to the abdominal area in middle age and later. Increased abdominal fat increases women's risk for cardiovascular disease and type 2 diabetes. Mature women also become increasingly at risk for osteoporosis, which makes adequacy of vitamin D and calcium levels important. Therefore, a healthy eating pattern is important to this population of women to maintain their quality of life.

Many studies have focused on the use of individual nutrients, particularly isoflavones, to help manage hot flashes. Unfortunately, most systematic reviews have not found these therapies to be effective when tested against placebo, although many studies may be of too short duration to adequately test the comparisons.[68]

Nutrition and Selected Conditions

Eating Disorders

An estimated 5% of all women in the United States may have, or have had, some form of eating disorder, although fewer than half seek treatment for this condition.[69] Primary care providers may not automatically recognize eating disorders. Due to the difficulties of detection and the complex etiology of eating disorders, successful detection, diagnosis, and treatment require a multidisciplinary effort by a team of health professionals, preferably with special education in the field. Although midwives in busy practices may lack education for in-depth counseling and treatment of women with eating disorders, they should be able to rule out some instances of eating disorders through use of a brief screening and provide follow-up referrals when necessary.

The SCOFF is a brief five-question instrument with a high negative predictive value in the general population. That is, when used among a normal clinical population, the SCOFF questionnaire is not reliable in detecting eating disorder, but it is reliable in ruling it out.[70] "SCOFF" is a mnemonic for the five questions found in the questionnaire. The questions assess whether individuals are making themselves vomit to relieve a feeling of fullness (S); whether they are concerned that they have lost control over what or how much they eat (C); whether they have recently lost a significant amount of weight (15 pounds or more) (O); whether they feel that they are "fat" even when others comment that they are thin (F); and whether they feel that thoughts and feelings about food are dominating their life (F). An answer of "yes" to two or more questions is indicative of an eating disorder. The SCOFF has been validated as an effective screening tool to screen for eating disorders.[70] Two questions from the SCOFF that may be particularly associated with pica (another eating order) are the two "F" questions related to feeling fat and thinking a lot about food.[71] Individuals with some symptoms of disordered eating should be assessed in more detail by professionals in the area.

Cancer

Numerous studies of every type have been undertaken to identify the role of diet in the development of cancer. Most of the intervention trials that tested supplements with selected vitamins or minerals have yielded disappointing results in terms of these nutrients' ability to prevent cancer. This fact underlies the USPSTF finding that there is no evidence to support routine vitamin/mineral supplementation to prevent cancer. One reason for these disappointing results has already been discussed in this chapter—namely, that the totality of what we eat, including both macronutrients and micronutrients, exerts a powerful physiologic effect, probably more powerful than just providing a single supplement without changing the overall dietary pattern.

That said, strong evidence from World Cancer Fund International links overweight/obesity or excess abdominal fat to 11 different cancers, including breast, endometrial, and ovarian cancers.[72] This organization notes especially strong evidence that the consumption of nonstarchy vegetables and fruits is protective against mouth, esophageal, stomach, and lung cancers. Colorectal cancer is the cancer for which the strongest evidence shows that what we

eat or do not eat matters. Along with exercise, dietary fiber, garlic, milk, and calcium consumption have preventive effects related to colorectal cancer, whereas consumption of red meat, processed meat, and alcohol, along with greater body and abdominal fat, increase the risk of this cancer. Unfortunately, for breast and reproductive cancers, there is not strong evidence that diet plays a role in their development, beyond the indication that greater body or abdominal fat increases the risk of postmenopausal breast cancer and endometrial cancer. For premenopausal breast cancer, breastfeeding is protective and alcohol use increases risk.

Age-Related Eye Disease

Macular degeneration and cataracts are two eye-related conditions that women may develop as they age. Strong evidence shows that consumption of vegetables and fruits rich in lutein and zeaxanthines, which accumulate in the eye, may protect against free-radical damage to the eye caused by smoking, pollution, sunlight, and metabolism from an unhealthy diet pattern such as consumption of a high-glycemic diet.[73] Spinach, kale, and brightly colored fruits and vegetables are rich in these nutrients. Additionally, higher B vitamin intake is associated with decreased cataracts.[74]

Conclusion

Research increasingly confirms that good nutrition is essential during all phases of life, and the basic principles of good nutrition remain remarkably consistent throughout the life course. Albeit with some caveats, good nutrition is good nutrition—for women who are pregnant, or who want to lose weight, gain weight, or maintain their weight. The midwife should understand the basics of nutrition and how dietary patterns affect normal physiology. Nutrition counseling should be gently incorporated into a midwife's regular practice so that each woman can understand some basic principles of nutrition, the physiologic response of the body to ingested food, and current evidence-based dietary guidelines. Although understanding individual components of the diet is important for health professionals, counseling a woman about diet should remain positively focused on the types of preferable foods to eat and the need to eat a variety of foods, rather than emphasizing individual macronutrients and micronutrients. Of course, a midwife should also be prepared with nutrition information specific to pregnancy, be alert to the possibility of eating disorders and refer women appropriately in such cases, and be ready with encouragement, factual information, and possible referral if a woman indicates a desire to achieve a healthier weight.

Resources

Organization	Description	Webpage
Websites		
National Institutes of Health (NIH)	Body mass calculator (English and Spanish)	https://www.nhlbi.nih.gov/health/educational/lose_wt/BMI/bmicalc.htm
WebMD	BMI calculator and calories for weight goal	http://www.webmd.com/diet/calc-bmi-plus
University of Sydney	Glycemic index for foods	http://www.glycemicindex.com
Brown University School of Public Health	Nutrition tools Rate My Plate (English, Spanish); Rapid Eating Assessment for Patients (REAP) tool; WAVE Assessment (a tool to assess weight, activity, and variety of foods eaten on one page)	https://www.brown.edu/academics/public-health/research/health-equity/tools-and-resources
Centers for Disease Control and Prevention (CDC)	Nutrition topics	http://www.cdc.gov/nutrition/index.html
Harvard University School of Public Health	The Nutrition Source	http://www.hsph.harvard.edu/nutritionsource
U.S. Department of Agriculture (USDA)	ChooseMyPlate.gov has information and educational materials on MyPlate diets, including vegetarian versions.	https://www.choosemyplate.gov
	Interactive nutrient database to look up nutrient content of fresh and prepared foods	https://ndb.nal.usda.gov/ndb/
Smartphone Apps		
U.S. Department of Agriculture (USDA)	SuperTracker is a site that allows a person to track good food, exercise, weight, and set personal goals	https://www.supertracker.usda.gov
MyFitnessPal	Site to track weight, diet calories, and exercise over time with a large database of foods or can be customized to personal intake of food	https://www.myfitnesspal.com
Handouts About Nutrition		
Journal of Midwifery and Women's Health and American	Share with Women series including: *Vitamin D* and *Folic Acid: What's It All About?*	http://www.midwife.org/Share-With-Women/

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References

1. Xu Z, Knight R. Dietary effects on human gut microbiome diversity. *Br J Nutr* 2015;113(suppl):S1-S5.
2. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity among adults: United States, 2011–2012. *NCHS Data Brief*. 2013;131:1-8.
3. National Center for Health Statistics. *Health, United States, 2015: With Special Feature on Racial and Ethnic Health Disparities*. Hyattsville, MD: National Center for Health Statistics; 2016.
4. U.S. Department of Health and Human Services, U.S. Department of Agriculture. *2015–2020 Dietary Guidelines for Americans*. 8th ed. December 2015. Available at https://health.gov/dietaryguidelines/2015/resources/2015-2020_Dietary_Guidelines.pdf. Accessed August 16, 2017.
5. Food and Nutrition Board. *Dietary Reference Intakes (DRIs): Recommended Intakes for Individuals*. Washington, DC: National Academy of Sciences; 2004, updated 2011.
6. Office of Nutrition Labeling and Dietary Supplements. *Food Labeling Guide*. College Park, MD: Food and Drug Administration; 2009.
7. Widen E, Siega-Riz AM. Prenatal nutrition: a practical guide for assessment and counseling. *J Midwifery Womens Health*. 2010;55(6):540-549.
8. Wright JD, Wang C-Y. *Trends in Intake of Energy and Macaronutrients in Adults from 1999–2000 Through 2007–2008*. Hyattsville, MD: National Center for Health Statistics; 2010.
9. Skerrett PJ, Willett WC. Essentials of healthy eating: a guide. *J Midwifery Womens Health*. 2010;55(6):492-501.
10. McNamara DJ. Dietary cholesterol, heart disease risk and cognitive dissonance. *Proc Nutr Soc*. 2014;73(2):161-166.
11. Miller M, Stone NJ, Ballantyne C, et al. Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*. 2011;123(20):2292-2333.
12. Rosinger A, Carroll MD, Lacher D, Ogden C. Trends in total cholesterol, triglycerides, and low-density lipoprotein in US adults, 1999–2014. *JAMA Cardiol*. 2017; 2(3):339-341.
13. Chavarro JE, Rich-Edwards JW, Rosner BA, Willett WC. Dietary fatty acid intakes and the risk of ovulatory infertility. *Am J Clin Nutr*. 2007;85(1):231-237.
14. Mozaffarian D, Pischon T, Hankinson SE, et al. Dietary intake of trans fatty acids and systemic inflammation in women. *Am J Clin Nutr*. 2004;79(4):606-612.
15. Doell D, Folmer D, Lee H, Honigfort M, Carberry S. Updated estimate of trans fat intake by the US population. *Food Addit Contam Part A Chem Anal Control Expo Risk Assess*. 2012;29(6):861-874.
16. Brand-Miller JC, Stockmann K, Atkinson F, Petocz P, Denyer G. Glycemic index, postprandial glycemia, and the shape of the curve in healthy subjects: analysis of a database of more than 1,000 foods. *Am J Clin Nutr*. 2009;89(1):97-105.
17. Hoy MK, Goldman JD. Fiber intake of the U.S. population: what we eat in America, NHANES 2009–2010. *Food Surveys Res Group Diet Data Brief*. September 2014;12.
18. Goodnight W, Newman R. Optimal nutrition for improved twin pregnancy outcome. *Obstet Gynecol*. 2009;114(5):1121-1134.
19. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA*. 2002;287(18):2414-2423.
20. Craig WJ, Mangels AR. Position of the American Dietetic Association: vegetarian diets. *J Am Diet Assoc*. 2009;109(7):1266-1282.
21. Moshfegh A, Goldman J, Ahuja J, Rhodes D, LaComb R. Usual nutrient intakes from food and water compared to 1997 Dietary Reference Intakes for vitamin D, calcium, phosphorus, and magnesium. *What we eat in America, NHANES 2005–2006*. U.S. Department of Agriculture; 2009.
22. Chen M, Li Y, Sun Q, et al. Dairy fat and risk of cardiovascular disease in 3 cohorts of US adults. *Am J Clin Nutr*. 2016;104(5):1209-1217.
23. U.S. Department of Agriculture. USDA National Nutrient Database for Standard Reference, Release 28. 2016. Available at: https://www.ars.usda.gov/northeast-area/beltsville-md/beltsville-human-nutrition-research-center/nutrient-data-laboratory/docs/usda-national-nutrient-database-for-standard-reference/. Accessed May 11, 2017.
24. Hollis BW, Wagner CL. Vitamin D and pregnancy: skeletal effects, nonskeletal effects, and birth outcomes. *Calcif Tissue Int*. 2012;92(2):128-139.
25. National Center for Environmental Health. *Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population*. Atlanta, GA: Centers for Disease Control and Prevention; 2012.
26. Caldwell KL, Pan Y, Mortensen ME, Makhmudov A, Merrill L, Moye J. Iodine status in pregnant women in the National Children's Study and in U.S. women (15–44 years), National Health and Nutrition Examination Survey 2005–2010. *Thyroid*. 2013;23(8):927-937.
27. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Calcium and Vitamin D*. Washington, DC: National Academy Press; 2010.
28. Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an

Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2011;97(7):1911-1930.
29. Pludowski P, Holick MF, Grant WB, et al. Vitamin D supplementation guidelines. *J Steroid Biochem Mol Biology*. February 12, 2017. [Epub ahead of print]. doi:10.1016/j.jsbmb.2017.01.021.
30. Pfeiffer CM, Sternberg MR, Hamner HC, et al. Applying inappropriate cutoffs leads to misinterpretation of folate status in the US population. *Am J Clin Nutr*. 2016;104(6):1607-1615.
31. Almeida OP, Ford AH, Flicker L. Systematic review and meta-analysis of randomized placebo-controlled trials of folate and vitamin B_{12} for depression. *Int Psychogeriatr*. 2015;27(5):727-737.
32. Forsyth JC, Mueller PD, Becker CE, et al. Hydroxocobalamin as a cyanide antidote: safety, efficacy and pharmacokinetics in heavily smoking normal volunteers. *J Tox Clin Toxicol*. 1993;31(2):277-294.
33. Sharabi A, Cohen E, Sulkes J, Garty M. Replacement therapy for vitamin B_{12} deficiency: comparison between the sublingual and oral route. *Br J Clin Pharm*. 2003;56(6):635-638.
34. Food and Nutrition Board. *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B_6, Folate, Vitamin B_{12}, Pantothenic Acid, Biotin, and Choline*. Washington, DC: National Academy of Sciences; 1998.
35. Dawson-Hughes B, Looker AC, Tosteson AN, Johansson H, Kanis JA, Melton LJ 3rd. The potential impact of the National Osteoporosis Foundation guidance on treatment eligibility in the USA: an update in NHANES 2005–2008. *Osteoporosis*. 2012;23(3):811-820.
36. Bailey RL, Dodd KW, Goldman JA, et al. Estimation of total usual calcium and vitamin D intakes in the United States. *J Nutr*. 2010;140(4):817-822.
37. Sekhar DL, Kunselman AR, Chuang CH, Paul IM. Optimizing hemoglobin thresholds for detection of iron deficiency among reproductive-age women in the United States. *Translat Res*. 2017;180:68-76.
38. Patel KV. Epidemiology of anemia in older adults. *Semin Hematol*. 2008;45(4):210-217.
39. Chang TP, Rangan C. Iron poisoning: a literature-based review of epidemiology, diagnosis, and management. *Ped Emerg Care*. 2011;27(10):978-985.
40. Moore LV, Thompson FE. Adults meeting fruit and vegetable intake recommendations—United States, 2013. *MMWR*. 2015;64(26):709-713.
41. Angelo G, Drake VJ, Frei B. Efficacy of multivitamin/mineral supplementation to reduce chronic disease risk: a critical review of the evidence from observational studies and randomized controlled trials. *Crit Rev Food Sci Nutr*. 2015;55(14):1968-1991.
42. Biesalski HK, Tinz J. Multivitamin/mineral supplements: rationale and safety: a systematic review. *Nutrition*. 2017;33:76-82.
43. Willett WC, Skerrett PJ. *Eat, Drink, and Be Healthy: The Harvard Medical School Guide to Healthy Eating*. Free Press/Simon & Schuster: New York, NY; 2005.
44. The Nutrition Source. *Healthy Eating Plate*. 2011. Boston, MA: Department of Nutrition, Harvard School of Public Health. Available at: https://www.hsph.harvard.edu/nutritionsource/. Accessed August 18, 2017.
45. U.S. Department of Agriculture. MyPlate. Available at: https://www.choosemyplate.gov/MyPlate. Accessed August 30, 2017.
46. Dernini S, Berry EM, Serra-Majem L, et al. Med Diet 4.0: the Mediterranean diet with four sustainable benefits. *Pub Health Nutr*. 2017;20(7):1322-1330.
47. Chen ST, Maruthur NM, Appel LJ. The effect of dietary patterns on estimated coronary heart disease risk: results from the Dietary Approaches to Stop Hypertension (DASH) trial. *Cir Cardio Qual Outcomes*. 2010;3(5):484-489.
48. Jenkins DJ, Jones PJ, Lamarche B, et al. Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA*. 2011;306(8):831-839.
49. Melina V, Craig W, Levin S. Position of the Academy of Nutrition and Dietetics: vegetarian diets. *J Acad Nutr Diet*. 2016;116(12):1970-1980.
50. Tripkovic L, Lambert H, Hart K, et al. Comparison of vitamin D_2 and vitamin D_3 supplementation in raising serum 25-hydroxyvitamin D status: a systematic review and meta-analysis. *Am J Clin Nutr*. 2012;95(6):1357-1364.
51. Appleby PN, Key TJ. The long-term health of vegetarians and vegans. *Proc Nutr Soc*. 2016;75(3):287-293.
52. Harris WS. Achieving optimal n-3 fatty acid status: the vegetarian's challenge . . . or not. *Am J Clin Nutr*. 2014;100(suppl 1):449S-452S.
53. Kim HS, Patel KG, Orosz E, et al. Time trends in the prevalence of celiac disease and gluten-free diet in the US population: results from the National Health and Nutrition Examination Surveys 2009–2014. *JAMA Int Med*. 2016;176(11):1716-1717.
54. Fasano A, Sapone A, Zevallos V, Schuppan D. Nonceliac gluten sensitivity. *Gastroenterology*. 2015;148(6):1195-1204.
55. Vici G, Belli L, Biondi M, Polzonetti V. Gluten free diet and nutrient deficiencies: a review. *Clin Nutr*. 2016;35(6):1236-1241.
56. Saccone G, Berghella V, Sarno L, et al. Celiac disease and obstetric complications: a systematic review and metaanalysis. *Am J Obstet Gynecol*. 2016; 214(2):225-234.

57. Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Obesity Society. *Circulation*. 2014;129(25 suppl 2):S102-S138.
58. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global boundary value. *Nutri Res Rev*. 2010;23(2):247-269.
59. Gans KM, Risica PM, Wylie-Rosett J, et al. Development and evaluation of the nutrition component of the Rapid Eating and Activity Assessment for Patients (REAP): a new tool for primary care providers. *J Nutr Ed Behav*. 2006;38(5):286-292.
60. Daneschvar HL, Aronson MD, Smetana GW. FDA-approved anti-obesity drugs in the United States. *Am J Med*. 2016;129(8):879e1-879e6.
61. Colquitt JL, Pickett K, Loveman E, Frampton GK. Surgery for weight loss in adults. *Cochrane Database Syst Rev*. 2014;8:CD003641. doi:10.1002/14651858.CD003641.pub4.
62. Milone M, Sosa Fernandez LM, Sosa Fernandez LV, et al. Does bariatric surgery improve assisted reproductive technology outcomes in obese infertile women? *Obesity Surg*. 2017;27(8):2106-2112.
63. Biro FM, Greenspan LC, Galvez MP. Puberty in girls of the 21st century. *J Pediatr Adolesc Gynecol*. 2012;25(5):289-294.
64. Cheng G, Buyken AE, Shi L, et al. Beyond overweight: nutrition as an important lifestyle factor influencing timing of puberty. *Nutr Rev*. 2012;70(3):133-152.
65. Davidsen L, Vistisen B, Astrup A. Impact of the menstrual cycle on determinants of energy balance: a putative role in weight loss attempts. *Int J Obes*. 2007;31(12):1777-1785.
66. Centers for Disease Control and Prevention. Infertility FastStats. 2016. Available at: https://www.cdc.gov/nchs/fastats/infertility.htm. Accessed August 18, 2017.
67. Chavarro JE, Rich-Edwards JW, Rosner BA, Willett WC. Diet and lifestyle in the prevention of ovulatory disorder infertility. *Obstet Gynecol*. 2007;110(5):1050-1058.
68. Lethaby A, Marjoribanks J, Kronenberg F, Roberts H, Eden J, Brown J. Phytoestrogens for menopausal vasomotor symptoms. *Cochrane Database Syst Rev*. 2013;12:CD001395.
69. Duncan AE, Ziobrowski HN, Nicol G. The prevalence of past 12-month and lifetime *DSM-IV* eating disorders by BMI category in US men and women. *Eur Eating Disord Rev*. 2017;25(3):165-171.
70. Solmi F, Hatch SL, Hotopf M, Treasure J, Micali N. Validation of the SCOFF questionnaire for eating disorders in a multiethnic general population sample. *Int J Eating Disord*. 2015;48(3):312-316.
71. Santos AM, Benute GR, Nomura RM, Santos NO, De Lucia MC, Francisco RP. Pica and eating attitudes: a study of high-risk pregnancies. *Matern Child Health J*. 2016;20(3):577-582.
72. World Cancer Research Fund International. *Continuous Update Project*. Available at: http://wcrf.org/int/research-we-fund/continuous-update-project-findings-reports. Accessed July 14, 2017.
73. Glaser TS, Doss LE, Shih G, et al. The association of dietary lutein plus zeaxanthin and B vitamins with cataracts in the age-related eye disease study: AREDS Report No. 37. *Ophthalmology*. 2015;122(7):1471-1479.
74. Weikel KA, Garber C, Baburins A, Taylor A. Nutritional modulation of cataract. *Nutr Rev*. 2014;72(1):30-47.

CHAPTER

8

Mental Health Conditions

BARBARA W. GRAVES

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Mental health disorders affect millions of individuals and families in the United States. Primary care providers, including midwives, care for women who are affected by mental health disorders, either as a sole disorder or comorbid with other illnesses. This chapter presents an overview of mental health conditions that commonly affect women; a framework for identification of common disorders; and screening strategies for determining the need for urgent referral to a mental health expert. An introduction to treatment modalities for many of the most common conditions is included.

Controversy exists regarding use of the terms “mental health” and “behavioral health.” Since no clear consensus is available, this chapter uses the term “mental health.” Substance use, which may be included under the umbrella of behavioral health, is reviewed in the *Common Conditions in Primary Care* chapter.

Mental health disorders are the most common and costly cause of disability in the United States.[1,2] Almost one third of persons who reside in the United States will suffer from an anxiety disorder in their lifetime, and between 15% and 20% will experience a major depressive episode.[3] Unfortunately, many individuals with mental health disorders do not seek treatment, and of those who do, many are not able to access the mental health system due to widespread shortages of mental health providers or lack of financial resources. Members of uninsured, underinsured, low income and minority populations are the least likely group to receive treatment.[3]

Mental health disorders affect more than the individual; they also affect entire families. This phenomenon is especially true when women have such disorders, as they are most often the primary care providers for children. In addition, women are at risk for mental health disorders that are hormonally associated such as premenstrual mood disorder, perinatal and postpartum mood disorders. **Figure 8-1** presents the lifetime prevalence of selected mental health

disorders in men and women.[3] A cross-national survey of 72,933 individuals in Africa, Asia, Europe, the Middle East, and the Americas found that among all cohorts in all countries, women had an odds ratio (OR) of 1.9 (95% confidence interval [CI], 1.8–2.0) for experiencing depression and an OR of 1.7 (95% CI, 1.6–1.8) for experiencing an anxiety disorder when compared to men. No significant gender difference was found among those with a bipolar disorder, and the difference for social phobia (OR, 1.3; 95% CI, 1.2–1.4) was less than other anxiety disorders.[4]

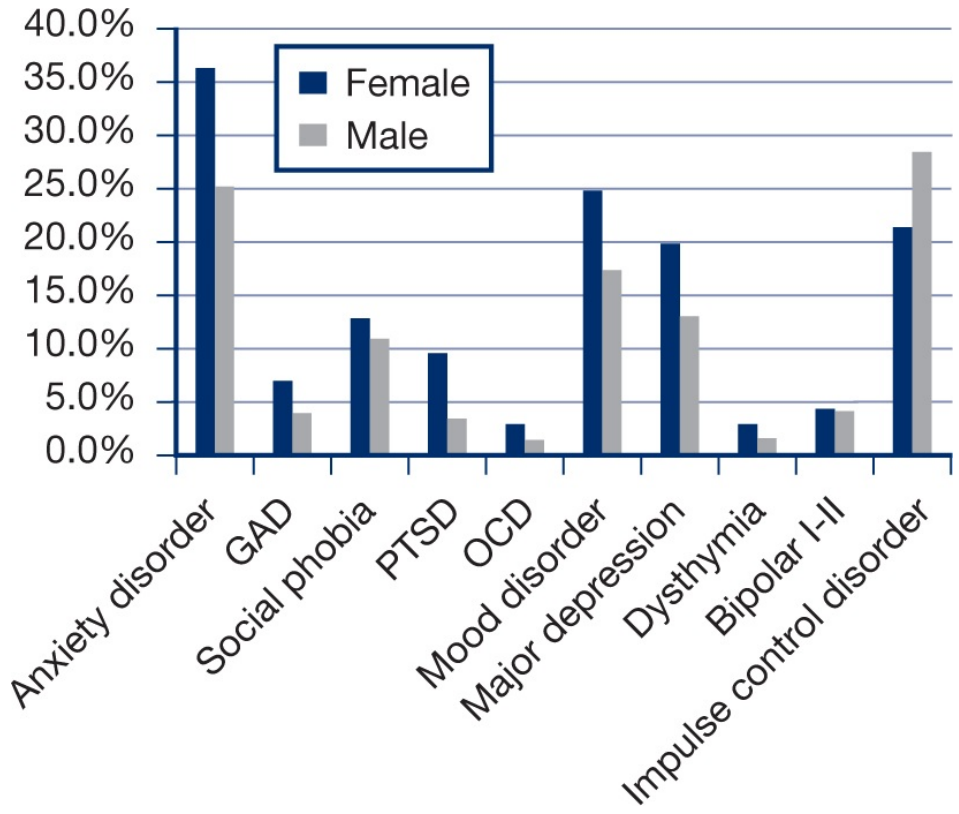


Figure 8-1 Lifetime prevalence of selected disorders identified in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*) from the World Mental Health Survey version of the Composite International Diagnostic Interview.

Abbreviations: GAD, generalized anxiety disorder; OCD, obsessive–compulsive disorder; PTSD, post-traumatic stress disorder.

Data from Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of *DSM-IV* disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry.* 2005;62(6):593-602.[3]

The etiology of mental disorders is complex, with genetic,[5] epigenetic, and environmental factors all intertwined.[6] The two most frequently encountered mental health disorders in primary care are depression and anxiety, both of which are multifactorial in origin. Twin studies have found that genetic factors are a significant contributor to the development of both major depression and anxiety disorders, although perhaps more for depression than for anxiety.[7]

Attenuation appears to diminish the influence of genetics as individuals move from childhood to adulthood.[8-10] Genetics appears to have a greater influence in women than in men,

owing to hormonal shifts related to puberty, menstruation, and childbearing that influence both the neurologic and immune systems. Women are also more likely than men to exhibit somatic symptoms associated with mood disorders.[2] Environment and experiences are equally important in determining whether an individual will develop depression or anxiety. Culture plays a significant role in the meaning an individual assigns to mental illness and the ways in which symptoms present. Environmental risk factors include childhood adversity such as poor parental attachment, witnessing or experiencing trauma, and subsequent stressors such as relationship problems and poor social support. Brain imaging studies have demonstrated altered connectivity between the limbic system, including the amygdala, and the frontal cortex among individuals with mood disorders.[7,11,12]

The diagnostic standard for psychiatric disorders is the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*). This manual has evolved as knowledge about mental health has increased, with the most recent revision as of this writing, the fifth edition *DSM-5*, being published in 2013.[13] *DSM-5* delineates specific diagnostic criteria for each distinct psychiatric diagnosis, and is the reference for the tables throughout this chapter.

The management of women with major mental health disorders, such as schizophrenia, bipolar disorder (BPD), obsessive–compulsive disorder (OCD), and eating disorders, is beyond the scope of care for most primary care practitioners. The role of the midwife in caring for women with one of these disorders is usually to perform screening and referral. In contrast, midwives working as primary care providers may diagnose, initiate, and perhaps, continue treatment for women with depression, including perinatal and postpartum depression, and some women with anxiety disorders. Although most primary care providers do not have the training for counseling or therapy, they often can prescribe or renew psychopharmacologic medications and recommend lifestyle changes, as well as work in concert with psychiatric professionals. Proper education is necessary to prescribe psychotropic medications appropriately and safely.

Screening for Mental Conditions

The U.S. Preventive Services Task Force recommends that all adults, pregnant women, and women who recently gave birth be screened for depression.[14] Several screening tools are available for use in clinical practice and those commonly used are presented in this chapter. Some depression screening instruments may miss detection of individuals with other psychiatric disorders, so it is important to choose the tool that is best for the population being screened.

A recently developed questionnaire, the M-3 Checklist, is a 23-item checklist that screens an individual for depression, anxiety disorders, and bipolar disorder, and includes questions specific to functional impairment secondary to their symptoms. The validity and feasibility of this tool were examined using a study population that included 723 English-speaking individuals attending a family medicine clinic, 647 of whom completed all aspects of the study. Participants completed the M-3 Checklist while waiting to be seen by their clinician; they also answered a brief exit questionnaire about the checklist. The checklist was completed in less than 5 minutes. A master's-prepared diagnostic interviewer contacted each participant by phone within 30 days of the visit to administer the Mini Neuropsychiatric Interview (MINI); the mean time from the clinic visit to the follow-up phone call was 8.8 days. The checklist was scored in a two-step method, beginning with functional impairment, which eliminated 53.9% of the population who would be eliminated from further scoring. This first step would have eliminated approximately 11% who met the MINI criteria for a psychiatric diagnosis. Of the participants who passed this "gateway," 62.4% of the remaining group were diagnosed with a psychiatric disorder. In total, 36.4% of the participants were diagnosed with one or more psychiatric disorders. Among the participants, 4.2% were diagnosed with depression without anxiety, 12.1% with depression and anxiety, 9.1% with anxiety without depression, and 9.3% with bipolar disorder. The sensitivity, specificity, positive likelihood ratio, and negative likelihood ratios for depression, bipolar disorder, anxiety, and post-traumatic stress disorder (PTSD) were comparable to the corresponding statistics for single-disorder screening instruments.[15]

Overview: Psychotherapy and Pharmacotherapy

The two primary therapeutic modalities for mental health disorders are psychotherapy and pharmacotherapy. Several different forms of psychotherapy exist, and several different classes of drugs have effects on the neurotransmitters relevant to mental health disorders. For some conditions, such as depression, a combination of pharmacotherapy and psychotherapy is most effective as treatment; for others, such as anxiety disorders, psychotherapy is more effective than pharmacotherapy.

Psychotherapy

In brief, psychotherapy helps individuals understand emotions, behaviors, and ideas that contribute to their mental disorder, and identifies ways to help modify emotions, behaviors, and/or ideas. Therapy can be conducted individually, in groups, or for families. **Table 8-1** presents an introduction to the primary forms of psychotherapy used to treat the most common mental health conditions that a midwife may identify in clinical practice.

Table 8-1 Common Types of Psychotherapy

Therapy	Description	Conditions for Which Effectiveness Is Established
CBT	Well-established, effective short-term therapy. Focus is on identifying and changing thinking and behavior patterns. Benefits are usually seen in 12–16 weeks. Therapy may involve reading and keeping records between therapy sessions.	Depression[a] Anxiety disorders Eating disorders Phobias
Exposure therapy	A form of CBT. Exposure therapy is used to reduce anxiety and fear responses via gradual or simulated exposure to the anxiety-provoking stimulus.	Phobias Obsessive–compulsive disorder
Dialectical behavioral therapy	A blend of CBT and Eastern meditation that focuses on combining acceptance and change. May include individual and group therapy. Originally developed for persons with suicidal thoughts. Useful for anyone with high emotional reactivity.	Depression[a] Panic disorder Post-traumatic stress disorder Eating disorders Obsessive–compulsive disorder
Interpersonal therapy	Limited structured therapy usually delivered over a 16-week period. Focuses on how interpersonal difficulties affect mental health and, in particular, depression.	Depression[a]
EMDR	Eye movements can reduce the intensity of disturbing thoughts. EMDR may be similar to what happens during REM sleep.	Post-traumatic stress disorder Panic attacks Phobias

Abbreviations: CBT, cognitive-behavioral therapy; EMDR, eye-movement desensitization and reprocessing; REM, rapid eye movement.

[a] Includes perinatal depression.

Cognitive-behavioral therapy (CBT)—a time-limited therapy that aims to change maladaptive patterns of thinking and behaviors—has been the subject of the most research and is effective for treating individuals with major depressive disorders and anxiety disorders. Topics, or "lessons," for sessions often include psychoeducation, behavioral activation, cognitive restructuring, problem solving, graded exposure, relapse prevention, and assertiveness skills. While CBT has traditionally been delivered by mental health specialists, the scarcity of such providers has limited its impact. More recently, CBT has also been delivered or supported by primary care providers, specially trained nurses, and social workers. In this approach, CBT may be delivered on a face-to-face basis, via the Internet, or as a computer-based therapy.

Pharmacotherapy

Psychotropic medications used to treat mood disorders and anxiety disorders include antidepressants, tricyclic antidepressants, and anxiolytics. These medications primarily affect the neurotransmitters serotonin, norepinephrine, and dopamine, either singly or in combination. Although neurotransmitters have many complex functions, they exert those functions only when released into the synaptic space between neurons. Thus, antidepressants such as selective serotonin reuptake inhibitors (SSRIs), which block reuptake of the neurotransmitter into the presynaptic neuron, will augment the effects of that neurotransmitter. Drugs that decrease anxiety are called *anxiolytics*. Benzodiazepines such as alprazolam (Xanax) are very effective for reducing anxiety but have a risk of dependence and tolerance so must be used with caution. This chapter presents a broad overview of these therapies for midwives who may collaborate with a mental healthcare professional in the care of women with mental disorders.

Mood Disorders

Mood disorders include forms of depression known as dysthymia (a mild, chronic depression) and major depressive disorder (MDD); bipolar disorder; and premenstrual dysphoric disorder. MDD is the most common mood disorder among adults in the United States, and is four times more prevalent than either dysthymia or bipolar I/II. While dysthymia and bipolar disorders are less prevalent, each of these disorders is associated with significant suffering and financial costs for individuals, families, and society.[16]

Figure 8-2 presents a simplified algorithm that can aid the differential diagnosis of mood disorders. This algorithm is based on a thorough process that is outlined in the *DSM-5*.[13,17] Note that, in some situations when mood disorders and anxiety disorders are discussed together, there is a tendency to use abbreviations when documenting health care visits. Perinatal mood and anxiety disorders should not be referred to using the acronym PMAD due to the negative connotations associated with this term.

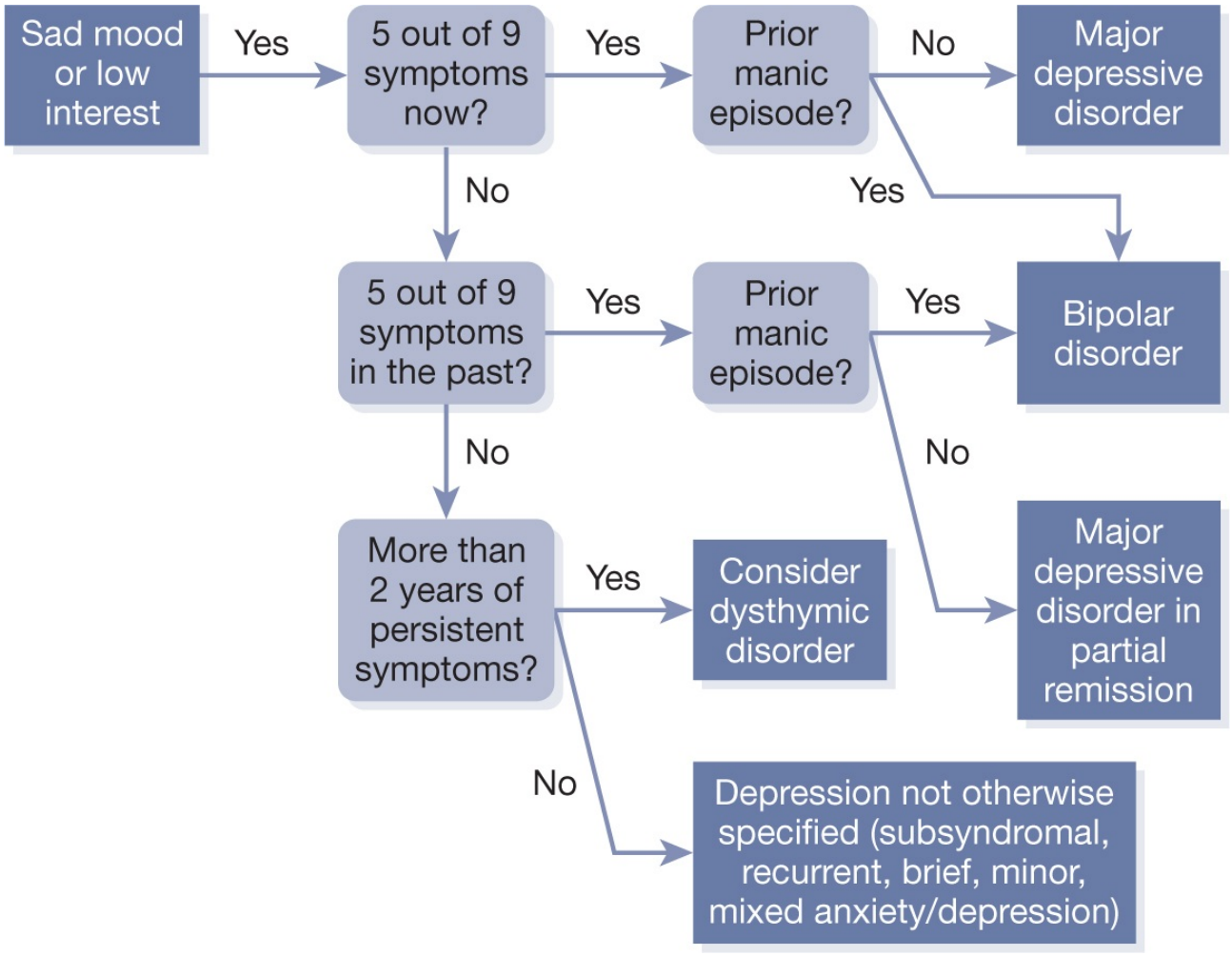


Figure 8-2 Differential diagnosis of mood disorders.

Reproduced with permission from U.S. Department of Health and Human Services. *Depression in Primary Care: Volume 1. Detection and Diagnosis.* AHCPR Publication No. 93-0550. Rockville, MD: Agency for Health Care Policy and Research; 1993:20.[17]

Depression

Depression affects millions of persons in the United States. Approximately 16.6% of the U.S. population have a major depressive disorder during their lifetime.[3] When Wittayanukorn et al. analyzed a sample of adults who completed the National Health and Nutrition Examination Survey (NHANES) between 2007 and 2010, they found that 9.8% met the criteria for moderate to severe depression.[18] An additional 15.8% of respondents reported symptoms of mild depression.[18] Women are at an increased risk compared to men, as are individuals who are ethnic minorities, living in poverty, or lacking adequate health insurance. Additional risk factors are listed in **Table 8-2**.

Table 8-2	Risk Factors for Major Depression
Stressful life events such as divorce, job change, or financial problems	
Recent death of a loved one or friend	
Family members with depression	
History of abuse or trauma	
Exposure to traumatic event (e.g., car accident, hurricane)	
Intimate-partner violence	
A serious or chronic medical condition	
Alcohol or drug abuse	
Prior episodes of depression	
Many medications may contribute to depression or cause symptoms of depression	

Depression has a major impact on quality of life and the ability to work and is second only to back and neck pain as the cause of disability days.[19] Depressive disorders are often associated with other comorbid conditions such as chronic disease, and may lead to decreased quality of life for those whose health conditions are aggravated by depression.[20] Depressed individuals may also have other psychiatric disorders, including personality disorders, anxiety disorders such as obsessive–compulsive disorder, eating disorders, and substance abuse disorders.

Women with combined diagnoses, including those exhibiting any psychotic features such as delusions or hallucinations, require specialist mental health services. They should be referred appropriately by the midwife.

Depression is the mental health disorder most frequently diagnosed by primary care providers. Even so, it is common for depression to be undiagnosed or treated suboptimally depending on the demands of the practice and attitudes of the provider. A 2012 systematic review of studies that invesigated the diagnosis of depression made by nonpsychiatric physicians found that the diagnosis of depression was missed more than half the time. Only 24% of those individuals with major depression are estimated to receive treatment, with only 9% receiving adequate therapy.[21]

The diagnostic criteria for major depression and dysthymia are described in **Table 8-3**.

Individuals with mild major depression will present with the minimum of symptoms that meet the diagnostic criteria; those with moderate depression exhibit more symptoms, but typically are able to carry out their usual activities; and those with major depression often exhibit difficulty even when performing normal activities.[22,23]

Table 8-3 **Common Depressive Disorders**

Major Depression

Symptoms (SIGECAPS)

Symptoms must include either depressed mood or anhedonia (lack of interest) PLUS any of the following symptoms (to total five symptoms), which occur nearly every day for at least 2 weeks, and are severe enough to impede function:

1. **S**leep disorder (insomnia or hypersomnia)
2. **I**nterest deficit or a lack of feeling pleasure
3. **G**uilt (worthlessness, hopelessness, regret)
4. **E**nergy deficit (fatigue or loss of energy nearly every day)
5. **C**oncentration deficit
6. **A**ppetite disorder (increased or decreased), unplanned weight loss or gain
7. **P**sychomotor retardation or agitation
8. **S**uicidality (recurrent thoughts of death)

There has never been a manic or hypomanic episode.

Symptoms are not attributable to a medical condition or substance use.

Must be qualified as a single episode or recurrent.

Dysthymia

Symptoms

Depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least 2 years.

Presence of two or more of the symptoms listed under major depression.

There has never been a manic or hypomanic episode.

Based on American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 5th ed. Arlington, VA: American Psychiatric Association; 2013*13*; Hackley B, Sharma C, Kedzior A, Sreenivasan S. Managing mental health conditions in primary care settings. *J Midwifery Womens Health.* 2010;55(1):9-19.*22*

Women with bipolar disorders (defined in **Table 8-4**) may also present with depression. However, depression associated with bipolar disorder is distinct from unipolar depression. It is extremely important to also screen for mania or hypomania when screening for depression, because treating women who have bipolar disorder with antidepressants can aggravate the manic condition. While management of women with depression may fall within the scope of primary care, individuals with symptoms of mania or hypomania should be referred to a mental health specialist.

Table 8-4 **Bipolar Disorder**

Diagnosis includes major depression AND history of mania (bipolar I) or hypomania (bipolar II), leading to a marked impairment in occupational function, social activities, or relationships; may lead to hospitalization or psychotic features. The manic phase must last a minimum of 7 days.
Symptoms of Mania
Inflated self-esteem or grandiosity
Decreased need for sleep
More talkative than usual or pressure to continue talking
Flight of ideas or feels thoughts are racing, agitation
High energy, irritability, or pleasure-seeking
Must have three symptoms (or four, if the only symptom is irritability) during the time of mood disturbance.
Symptoms of Hypomania
Lasts a minimum of 4 days
Briefer duration and less severe symptoms
Associated with unequivocal change in functioning that differs from normal function for that individual
Not severe enough to cause marked impairment in social or occupational functioning
Not associated with psychosis

Based on American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013[13]; Hackley B, Sharma C, Kedzior A, Sreenivasan S. Managing mental health conditions in primary care settings. *J Midwifery Womens Health.* 2010;55(1):9-19.[22]

Screening for Depression

The U.S. Preventive Services Task Force and other professional organizations recommend regularly screening women for depression and establishing a system for referring individuals to mental health services.[15] Several screening instruments can be used to assess for depression, ranging from a questionnaire with two questions to a questionnaire that has 21 items.

The Patient Health Questionnaire-2 (PHQ-2), the two-question instrument (**Table 8-5**), was developed and validated in New Zealand, and has a sensitivity of 97% (95% CI, 83–99%) and a specificity of 67% (62–72%).[24] The likelihood ratio for a positive test with the PHQ-2 was found to be 2.9 (95% CI, 2.5–3.4), and the likelihood ratio for a negative test was 0.05 (95% CI, 0.01–0.35).[25] Adding a third question about whether the individual desires help for the depression improved the specificity of the original two questions[26] but may decrease the sensitivity, with the ultimate result that some individuals who are depressed may not be identified.[27]

Table 8-5 **PHQ-2: Two-Question Screen for Depression[a]**

Over the 2 weeks how often have you been bothered by any of the following problems?	Not at All	Several Days	More Than Half the Days	Nearly Every Day
Little interest or pleasure in doing things	0	1	2	3
Feeling down, depressed, or hopeless	0	1	2	3

Abbreviation: PHQ-2, Patient Health Questionnaire-2.

[a] The PHQ-2 score ranges from 0 to 6. A cut off score of 3 is optimal for screening purposes.

Developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke and colleagues, with an educational grant from Pfizer Inc. Available at: http://www.phqscreeners.com.

Management for individuals who respond positively to either question on the PHQ-2 depends on the expertise of the clinician and the resources of the setting for management of mental health disorders. If either of these is lacking, the clinician should screen for immediate risk of harm to the individual or others; if no risk for harm is present, the clinician should refer the woman to an appropriate mental health provider.

The PHQ-9 (**Table 8-6**) is available in the public domain, has nine items, and is commonly used for monitoring response to treatment as well as screening for depression.[28] Depression screening can be conducted quickly, using a two-step screening process. A woman is asked the two questions of the PHQ-2, which is then followed up with the PHQ-9 as needed. This two-step screen provides a balance of sensitivity and specificity.[29] The midwife can then provide primary care or refer the woman to a mental health specialist depending on available resources. Additional screening for depression should include an evaluation for conditions or diseases that may be causing the depression. Thyroid function tests are indicated, and possibly a complete blood count to evaluate for anemia. Drug screening may be indicated as well.

Table 8-6 **PHQ-9: Nine-Question Screen for Depression**

Over the last 2 weeks, how often have you been bothered by any of the following problems? (use "x" to indicate your answer)	Not at All	Several Days	More Than Half the Days	Nearly Every Day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed; or the opposite—being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead, or of hurting yourself in some way	0	1	2	3
	Column Totals	____	____	____
	TOTAL SCORE	____		

10. If you checked off any problems, how difficult have those problems made it for you to do your work, take care of things at home, or get along with other people?

☐ Not difficult at all ☐ Somewhat difficult ☐ Very difficult ☐ Extremely difficult

Total Score	*Depression Severity*
1–4	Minimal depression
5–9	Mild depression
10–14	Moderate depression
15–19	Moderately severe depression
20–27	Severe depression

- Consider a depressive disorder if there are at least four "x" marks in the shaded section (including Questions 1 and 2). Add up the score to determine severity.
- Consider a major depressive disorder if there are at least five "x" marks in the shaded section (one of which corresponds to Question 1 or 2).
- Consider another depressive disorder if there are two to four "x" marks in the shaded section (one of which corresponds to Question 1 or 2).

Abbreviation: PHQ-9, Patient Health Questionnaire-9.

Question 10 Note: All responses should be verified by the clinician. Diagnoses of major depressive disorder or other depressive disorder also require impairment of social, occupational, or other important areas of functioning (Question 10) and ruling out normal bereavement, a history of a manic episode (bipolar disorder), and a physical disorder, medication, or other drug as the biological cause of the depressive symptoms.

Developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke and colleagues, with an educational grant from Pfizer Inc. Available at: http://www.phqscreeners.com.

The Center for Epidemiologic Studies-Depression Scale (CES-D), Patient Health Questionnaire-2 (PHQ-2), Patient Health Questionnaire-9 (PHQ-9), Beck Depression Inventory (BDI), and Edinburgh Postnatal Depression Screen (EPDS) have all been validated, and have sensitivities ranging from 0.8 to 0.9, and specificities from 0.7 to 0.85. All of these instruments are written at the sixth-grade level and are easy to complete, and several can be completed and scored online. The accuracy of the questionnaires appears comparable for identifying individuals with depression.[29] While all of these instruments have been widely

employed for depression screening, each has some benefits and some detractors. In particular, although the EPDS has been extensively validated for use during pregnancy and the postnatal time period,[30] it has not been validated for use outside the perinatal period. The BDI is copyrighted, and requires a fee for each use. The CES-D is the longest of the commonly used screening instruments for depression.

Suicidal Ideation and Intent

The midwife should always assess for suicidal ideation in women who have screened positive for depression and in anyone who expresses a sense of hopelessness. Common statements that are red flags for suicidal ideation include "Life is not worth living;" "My family would be better off without me;" "I won't be around to deal with that;" "I would be better off dead;" and "There is nothing I can do to make this better." Women at highest risk for suicide include those who have a specific plan or have access to the means to commit suicide, such as guns, cutting, or prescription or street drugs. Other risk factors include previous hospitalization or suicide attempt; family history of suicide; sense of hopelessness; experiencing family, romantic, or legal conflicts; social isolation; and insomnia.[31]

Screening instruments used to identify individuals at risk for suicide are listed in the Resources section at the end of this chapter. Although no single instrument is considered the best, or even the most frequently used, they all address the questions listed in **Table 8-7**. The midwife has a responsibility to refer any woman deemed at risk for suicide to emergency mental health services.[32]

Table 8-7 Questions to Assess for Suicidal Ideation and Intent

Question	Clinical Notes
1. Are you currently thinking about or have you recently thought about death or harming yourself?	If answer is yes, consider additional questions that assess frequency, intensity, and duration of suicidal ideation
2. Have you thought about how you would harm yourself?	If answer is yes, consider additional questions that assess frequency of suicidal thoughts about a possible method of suicide
3. Do you have access to a method such as gun and bullets or pills?	If answer is yes, consider additional questions that assess location of methods
4. Do you have any intention of following through with thoughts of self-harm?	If answer is yes, consider additional questions that assess: • Preparatory acts (e.g., collected pills, wrote a suicide note, obtained a gun) • Rehearsals for suicide (e.g., loading gun) versus nonsuicidal self-injurious acts

Additional Questions
What has kept you from acting on these thoughts?
What are your plans for the future?
Have you or a family member ever attempted suicide in the past?
Are you currently using drugs or alcohol (illicit or prescription)?
Have there been any changes in your employment, social life, or family?
Do you have friends or family with whom you are close? Have you told anyone that you have these thoughts?
Do you tend to be impulsive with your decisions or behavior?

Dysthymia Versus Major Depression

The term *dysthymia* comes from a Greek word meaning "bad state of mind" or "bad humor."

Dysthymia is a chronic form of depression in which the symptoms are neither as severe nor as numerous as they are in major depression. This disorder has been identified in cultures worldwide, although the prevalence appears to be higher in high-income countries when compared to low- to middle-income countries.[33] Dysthymia can be just as disabling as major depression due to its chronic nature even though its symptoms are less severe.

Similar to major depression, dysthymia occurs more commonly in women, and is often underdiagnosed except when complicated by comorbid major depressive episodes.[34] The lifetime community prevalence is between 3% and 6%, although a higher prevalence between 5% to 15% has been noted in primary care settings.[35] While the specific depressive symptoms may be less severe, dysthymia may be associated with slower and less complete recovery than major depression.[35] It is not uncommon for an individual with dysthymia to experience a major depressive disorder, a phenomenon known as "double depression."[36]

Treatment of Depression: General Principles

Many treament modalities exist for women with depression, including cognitive-behavioral therapy, counseling, faith-based therapy, group work, and psychotropic medications. Regardless of treatment modality selected, the goal is remission of symptoms. Education and recommendations for sleep hygiene, participation in pleasurable activities, and exercise are beneficial adjuncts.[32]

Evidence-based guidelines for the management of depression on the Internet are available for primary care providers and are listed in the Resources section at the end of this chapter. Complementary treatments that have been found to be efficacious as adjuncts to psychotherapy or medication include omega-3 fatty acid supplementation, *S*-Adenosyl methionine (SAMe), bright light therapy, massage, yoga, and acupuncture.[37] While some evidence suggests that St. John's wort (*Hypericum perforatum*), an herbal/botanical product, may benefit individuals with mild or moderate depression, products containing it are not regulated for purity or consistent dosing. In addition, St. John's wort is known to interact with many medications, including other antidepressants, protease inhibitors used for treatment of human immunodeficiency virus (HIV) infection, and combined oral contraceptives.

Medication and nonpharmacologic therapy are associated with similar rates of initial improvement, but individuals treated with medication alone are more likely to experience relapse compared to those treated with therapy.[38] Treatment with both nonpharmacologic therapy and psychotropic medication appears to result in the best remission rates. Current guidelines published in the United States recommend psychotherapy and pharmacotherapy together for treatment of MDD when possible.[32] The combination of both pharmacotherapy and psychotherapy improves the likelihood of remission and decreases the risk of relapse.[32,38–40] If combined therapy is not available, psychotherapy is recommended for persons with mild to moderate MDD, with pharmacotherapy being added if there is no response to the nonpharmacologic therapy.[32] Pharmacotherapy should be the initial therapy for anyone with severe major depression.[32] Gender does not appear to influence the response to cognitive-behavioral therapy as compared to the response to drug therapy.[39] The decision to initiate treatment with psychotherapy or medication is based on many factors, such as preferences of the individual, severity of depression, access to and acceptability of counseling, insurance coverage, and the individual's ability to commit to the time involved in therapy.[41]

Several studies have suggested that antidepressant therapy is more effective for treatment of dysthymia than for MDD. In 2011, Levkovitz et al. published the results of a meta-analysis of trials comparing antidepressants to placebo in persons with a diagnosis of MDD or dysthymia. Antidepressant therapy was more effective than placebo for persons with dysthymia (relative risk [RR], 1.75; 95% CI, 1.42–2.04; $p < 0.0001$).[41] In addition, the response to placebo in persons with dysthymia was lower than the response to placebo in persons with depression, further validating the effect of antidepressants for this population.[42] While interpersonal psychotherapy may be useful for persons with dysthymia,[43] it is not believed to be as effective as pharmacotherapy.[44]

The University of Washington's Advancing Integrated Mental Health Solutions (AIMS) Center has developed the IMPACT model, which recommends a team approach to following individuals who are diagnosed with depression.[45] Implementation of collaborative care using this model, which involves a case manager (either a specially trained nurse or social worker) and a psychiatrist, has been found to improve outcomes when compared to usual care.[46] The case manager educates the woman about depression, monitors depression symptoms, coaches her in behavioral activation and pleasant-event scheduling, encourages physical activity, and supports the mutually agreed-upon plan of care with close follow-up; in addition, the case manager may provide initial adjustments to medication dosages. The case manager also helps in coordinating referrals and reinforcing follow-through. The psychiatric consultant regularly reviews all cases in which improvement does not occur as expected and is available for consultation for any specific concerns.

Psychotherapy

Several types of psychotherapy are available for treating persons with MDD. To date, cognitive-behavioral therapy has the most evidence for effectiveness.[42] Most midwifery practices are not structured to provide ongoing psychotherapy. Nevertheless, the midwife's

responsibility for follow-up continues even after referral to a mental health specialist to ensure that the woman who has depression has not stopped therapy prior to achieving remission. The midwife may be in a unique position to provide targeted time-limited counseling from having developed an ongoing therapeutic relationship with the woman. This relationship provides an opportunity to discuss behaviors that may improve depression symptoms, such as an exercise program[32,47] and engaging in pleasurable activities. Bright light therapy has been shown to be effective in treating depression that has a seasonal component, and has benefits as an adjunct therapy for individuals with nonseasonal depression as well.[37]

When discussing psychotherapy, the woman can be reassured that therapy is short-term in nature for most persons with depression. Although the strongest evidence supports CBT, behavioral activation and interpersonal therapy have also been found to be effective, and both are recommended by the Canadian Network for Mood and Anxiety Treatments (CANMAT) as first-line psychological acute treatments for depression.[48] Early studies of Internet-delivered psychotherapy with guidance from a therapist have been promising. A recent meta-analysis concluded that Internet-based CBT intervention has superior short-term effectiveness compared to controls, although there is inadequate research to evaluate its long-term effectiveness.[49]

Pharmacologic Therapies

Due to the challenges of entering into psychotherapy, many individuals with depression will opt to initiate pharmacotherapy rather than counseling. Prior to beginning pharmacotherapy for depression, it is critical that the clinician has ruled out bipolar disorder, as treatment with an antidepressant may trigger mania,[50] suicidal ideation, or psychosis in persons with this condition.[51] During screening to rule out bipolar disorders, important signs include a family history of bipolar disorder, risky behaviors, legal issues, history of taking multiple antidepressants without improvement, history of suicide attempts, and a history of agitation or irritability while taking antidepressants in the past.[52]

Psychotropic medications affect the three neurotransmitters that modulate mood—dopamine, serotonin, and norepinephrine. **Figure 8-3** depicts the effects and interactions of these neurotransmitters.[51] Second-generation antidepressants, which include SSRIs, norepinephrine–dopamine reuptake inhibitors (NDRIs), and serotonin–norepinephrine reuptake inhibitors (SNRIs), are the first-line medications for treating depression.[52] The overall effects of SSRIs are considered modest.[52] In addition, most psychotropic medications have some adverse effects that can lead to discontinuation of the medication. Thus the cost–benefit ratio for an individual woman can be difficult to determine.

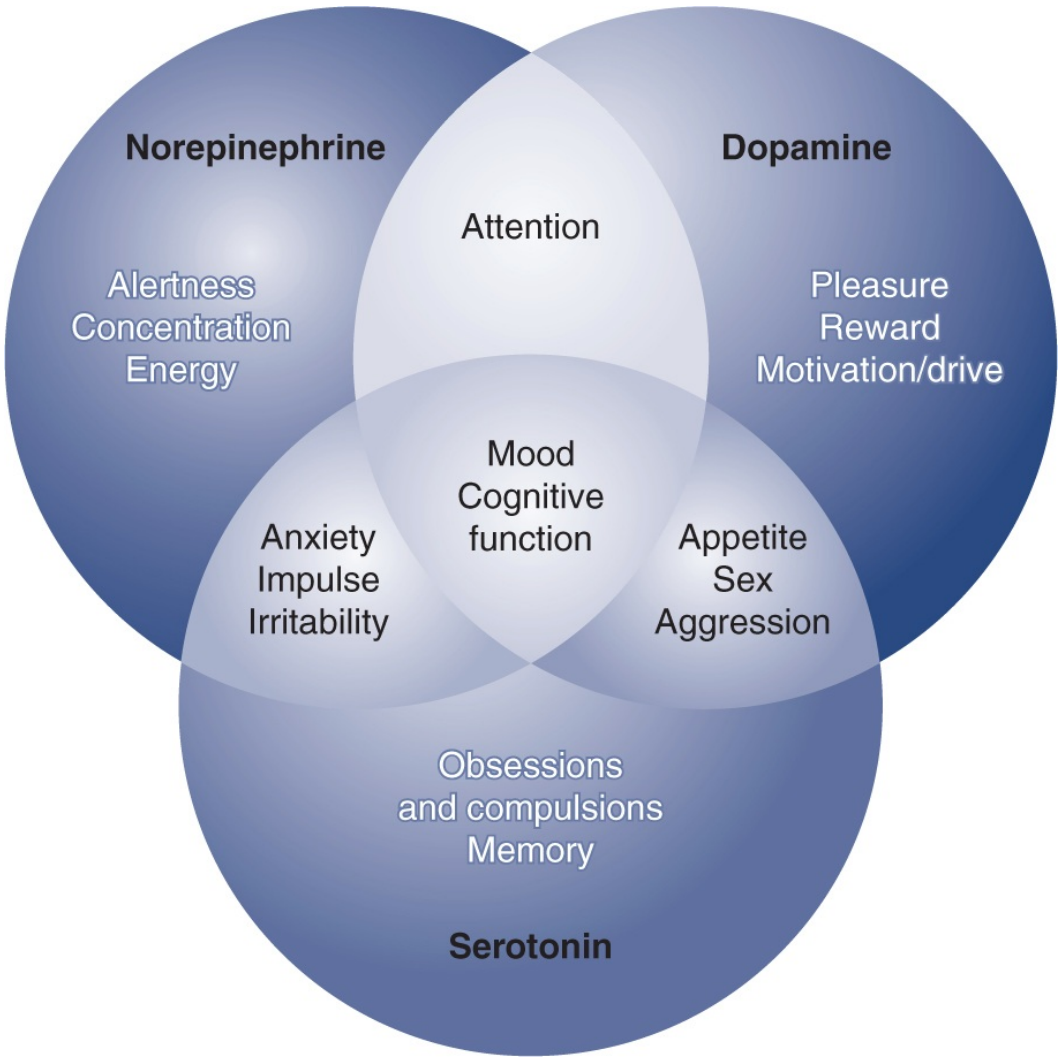


Figure 8-3 Role of dopamine, norepinephrine, and serotonin.

Reproduced with permission from Early NK. Mental health. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:727-764.[51]

Some providers may opt to prescribe bupropion (Wellbutrin, Zyban), an NDRI that has less effect on sexual function compared to SSRIs. Bupropion, however, lowers the threshold for seizures, so it should not be used with other medications that also lower the seizure threshold or for individuals with risk factors for seizures. Bupropion has no effect on anxiety, and may even aggravate anxiety.[53]

Classes of drugs that have been used in the past for depression, and may be prescribed by psychiatric clinicians in specific situations, include monoamine oxidase inhibitors and tricyclic antidepressants. These older medications have a narrow therapeutic range, potential for high toxicity, and serious interactions with many foods and other medications; thus, they are rarely prescribed by primary care providers.

The SSRIs are metabolized through the CPY450 enzyme system. As such, they interact with a wide variety of other medications that are metabolized by the same system. Some of the specific medications that are known to interact with these second-generation antidepressants include older antidepressants such as tricyclic antidepressants, digoxin (Lanoxin), warfarin

(Coumadin), anticonvulsants, beta blockers, cisapride (Propulside), and theophylline (Theo-Dur).[51] Prior to prescribing any psychotropic medication, the clinician is responsible for reviewing all the medications the woman is taking so as to avoid potentially serious interactions.

Research has failed to demonstrate significant differences in effectiveness among the various SSRIs, SNRIs, or NDRIs.[54] Nevertheless, individual medications in these classes do have different side-effect profiles, as summarized in **Table 8-8**.[55,56] The choice of which medication should be used to initiate therapy is based on the individual's preference, past successful treatment with a specific medication, and the predominate symptoms that accompany the depression. For example, all the SSRIs may be associated with weight gain, but fluoxetine (Prozac) is more activating, while sertraline (Zoloft) is less so.[55–58] Thus, fluoxetine would be a better choice to treat a woman with hypersomnia and low energy. As a group, the SNRIs are less frequently associated with weight gain than SSRIs.[22] The woman's ability and motivation to follow therapeutic recommendations is another consideration in selecting which SSRI or SNRI to use when initiating treatment. Abrupt discontinuation of SSRIs or SNRIs usually leads to serotonin withdrawal (or discontinuation) syndrome, whose symptoms include dizziness, ataxia, agitation, headache, tremor, and confusion.[22] Fluoxetine has the longest half-life of the second-generation antidepressants, and it may be the most appropriate medication for those who may have difficulty taking their pills consistently.

Table 8-8 **Antidepressant Medications Prescribed by Primary Care Providers**

Generic Agent (Brand Name)	Starting Dose	Dosage Range and Maximum Dose	Side-Effect Profile	Pharmacokinetic Half-life
Selective Serotonin Reuptake Inhibitors				
Citalopram (Celexa)	20 mg/day for 7 days, then may increase to 40 mg/day. Maintain initial dose for 4 weeks before increasing it.	20–40 mg/day	Minimal drug interactions. Weight gain is common, and more likely after 6 months of use. Good choice for women with anxiety and women who are pregnant. May cause QT prolongation. Lowest risk for liver toxicity; higher risk for sexual dysfunction.	Half-life of 35 hours; few metabolites
Escitalopram (Lexapro)	10 mg to start Increase to 20 mg if partial response after 4 weeks.	10–20 mg/day	Minimal drug interactions. Weight is gain common, and more likely after 6 months of use. Good choice for women with anxiety and women who are pregnant. May cause QT prolongation.	Half-life of 27–32 hours
Fluoxetine (Prozac)	10–20 mg/day May increase to 20 mg/day after 7 days Maintain at 20 mg/day for 4–6 weeks; then 30 mg for 2–4 weeks before additional increases.	20–80 mg/day	May worsen anxiety. Higher risk for switching to the mania phase if the woman has bipolar disorder. Lowest risk for sexual dysfunction. Weight gain is common, and more likely after 6 months of use. More activating than other SSRIs.	Longer half-life of 3–4 days; long elimination of active metabolites. Withdrawal symptoms are less likely than with all other SSRIs.
Paroxetine (Paxil)	10–20 mg/day for 7 days May increase dose by 10 mg/week	Maximum dose: 50 mg/day	Not recommended during pregnancy. Weight gain is more likely than with other SSRIs; may be sedating. Higher risk for sexual dysfunction. Anticholinergic. Recommended for women who breastfeed.	Short half-life; no active metabolites. Users are more likely to experience serotonin withdrawal symptoms.
Sertraline (Zoloft)	25–50 mg for 7 days, then may increase in 25-mg increments	Maximum dose: 200 mg/day	Weight gain is common, and more likely after 6 months of use. Limited CPY450 interactions. Mildly stimulating. Recommended for women who breastfeed.	Half-life of 2–4 days; some minimally active metabolites. Withdrawal symptoms are less likely.
Selective Norepinephrine Reuptake Inhibitors				
Duloxetine (Cymbalta)	20–60 mg as one or two doses/day 30 mg/day for 7 days, then may increase to 60 mg/day	Maximum dose: 60 mg/day	Weight-neutral in the short term; uncertain if weight gain occurs in the long term. Fewer sexual side effects than some SSRIs in the short term; no long-term difference. No impact on blood pressure.	Half-life of 12 hours. Withdrawal symptoms have been reported, but their frequency is unknown.

Venlafaxine (Effexor)	37.5 mg	75–300 mg/day in 2–3 divided doses	Weight-neutral. Fewer sexual side effects than SSRIs. Increases blood pressure in a dose-dependent fashion. More agitation and gastrointestinal side effects than SSRIs.	Withdrawal symptoms have been reported. Frequency of withdrawal symptoms is unknown.
Venlafaxine IR	37.5 mg	37.5–300 mg/day		
Norepinephrine–Dopamine Reuptake Inhibitors				
Bupropion (Wellbutrin, Zyban)	200 mg in divided doses, twice daily	450 mg in 4 divided doses. Single dose should not exceed 150 mg.	May be stimulating. No sexual side effects. Weight-neutral or minor weight loss. Lowers seizure threshold	Half-life is 29 hours for sustained release. Withdrawal symptoms have not been reported.
Bupropion SR (Wellbutrin SR)	300 mg in divided doses, twice daily	Maximum dose: 400 mg given in divided doses, twice daily. Single dose should not exceed 200 mg.		

Abbreviations: IR, immediate release; NDRI, norepinephrine–dopamine reuptake inhibitor; SNRI, serotonin–norepinephrine reuptake inhibitor; SR, sustained release; SSRI, selective serotonin reuptake inhibitor.

Based on Hackley B, Sharma C, Kedzior A, Sreenivasan S. Managing mental health conditions in primary care settings. *J Midwifery Womens Health.* 2010;55(1):9-19[22]; MacArthur Initiative on Depression and Primary Care. Depression management tool kit. 2009. Available at: http://www.integration.samhsa.gov/clinical-practice/macarthur_depression_toolkit.pdf. Accessed July 22, 2017[55]; Department of Veterans Affairs, Department of Defense. The management of major depressive disorder: clinical guideline summary. 2016. Available at: https://www.healthquality.va.gov/guidelines/MH/mdd/. Accessed July 22, 2017.[56]

Side Effects of Psychotropic Medications

Once the clinician has made the diagnosis of major depression and ruled out bipolar disorder, the decision to begin psychotropic medication is made jointly with the woman. All of the SSRIs and SNRIs may cause gastrointestinal upset, jitteriness, and headache after initiation, as the body adapts to the higher levels of neurotransmittors. Within approximately 2 weeks, the receptors are desensitized and downregulated, leading to the disappearance or significant decrease in these side effects.[51] Unfortunately, these adverse side effects often occur prior to a therapeutic effect becoming evident, and may lead the woman to discontinue the medication. Psychotropic drugs should be initiated at a low dose for the first 1 to 2 weeks to minimize these symptoms. The lower dose, when accompanied by anticipatory guidance, will improve the likelihood of the woman continuing the medication.

Adverse Effects of Psychotropic Medications

Serotonin syndrome is a potentially life-threatening reaction that results from excess serotonergic activity. Although it remains rare, its incidence is suspected to be rising due to the more frequent prescribing of serotonergic medications, the potential for overdose, and a possible toxic reaction when therapeutic doses of an SSRI or SNRI are potentiated by an

interaction with other agents. In 2005, 8585 cases of serotonin syndrome were reported in the United States with moderate to major, but not life-threatening, effects; in addition, 118 deaths were attributed to serotonin syndrome.[59] As many as 16% of individuals who overdose on SSRIs develop serotonin syndrome.[60] Combining treatment with more than one SSRI or SNRI, monoamine oxidase (MAO) inhibitors, lithium, or concurrent use of St. John's wort may precipitate serotonin syndrome and should be avoided. Some other classes of drugs—such as triptans, anticonvulsants, and some opioids—also have serotonergic activity. Many recreational drugs, including amphetamines, cocaine, and ecstasy, can trigger serotonin syndrome as well.

Signs and symptoms of serotonin synrome include autonomic instabilty such as hypertension or hypotension, hyperthermia, tachycardia and tachypnea, and diaphoresis; changes in mental status, including agitation, confusion, or hypomania; and neuromuscular changes such as myoclonus, hyperreflexia, tremor, ataxia, and rigidity.[61,62] The clinical manifestations can be highly variable, however, and referral of women who are suspected of experiencing serotonin syndrome to the emergency department is appropriate.[61,62]

The risk of suicide and suicide attempts has been found to increase during the first 1 to 2 months after beginning treatment with SSRIs, especially among adolescents and young adults.[63] In 2004, the U.S. Food and Drug Administration (FDA) placed a black box warning on these agents' labels for providers who prescribe SSRIs for children, adolescents, and young adults, younger than 25 years.[63] Depression is also associated with suicide attempts and because these medications can take several weeks to become effective, close monitoring is indicated for the first few months after therapy is initiated for all persons.

Follow-up and Monitoring

Ongoing monitoring of psychotropic medications is recommended at least monthly until full remission is achieved, and then every 2 to 3 months. Response to and side effects from the many SSRIs, SNRIs, and NDRIs vary greatly, and most women will require adjustments to the initial dose. Monitoring should begin within 1 to 2 weeks after initiating therapy, which can be accomplished either by phone or in a face-to-face visit. This encounter is an important time to discuss whether the individual has filled the prescription, begun taking it, and is continuing the medication, and if not, to identify the barriers or reasons. This first follow-up visit allows for assessment of side effects and possible increases in suicidal thoughts or behaviors.

Some women may experience an improvement in their depression by their 1- to 2-week visit, although many will not appreciate a response until as late as 6 weeks after beginning medication. It is recommended that a reassessment of depressive symptoms be repeated 4 to 8 weeks after initiating therapy. Repeat use of the screening instrument employed during the initial diagnosis allows the provider and the woman to determine whether the woman has achieved an objective, quantifiable response to the therapy.

Follow-up enables several assessments to be made, including how the woman is taking the drug, barriers to appropriately taking the medication, and side/adverse effects. The undertreatment of women with depression may be evidence of failure to provide the necessary follow-up. A partial response to treatment may be an indication that an increase in the dose of

the antidepressant is needed. If the woman has less than a 25% decrease in symptoms, either an increase in the dose or a switch to a different antidepressant is appropriate. Referral for concurrent psychotherapy may be useful as well. Further treatment of women who fail to respond is best managed with either referral to or in consultation with a psychiatric provider.

The initial goal is for the woman to experience a 50% reduction in symptoms within 10 to 12 weeks. Complete remission from depression would mean a score of less than 5 on the PHQ-9. **Table 8-9** provides guidance for alterations in management based on the woman's response to the PHQ-9.[20]

Table 8-9 **Using the PHQ-9 to Assess Clinical Response to Treatment of Depression**

PHQ-9 Score	Treatment Response After 4 Weeks	Treatment Plan
Drop of 5 points from baseline	Adequate	No treatment change needed. Follow up in 4 weeks.
Drop of 2–4 points from baseline	Possibly inadequate	May warrant an increase in antidepressant dose.
Drop of 1 point or no change or increase	Inadequate	Increase dose of medication; augmentation; switch; informal or formal psychiatric consultation; add psychological counseling.

Abbreviation: PHQ-9, Patient Health Questionnaire-9.

Based on Unützer J, Park M. Strategies to improve the management of depression in primary care. *Prim Care*. 2012;39(2):415-431.[20]

All clinicians who care for women with depression have the responsibility to follow the response to complete remission, as adequate treatment decreases the risk of relapse.[64] Inadequate treatment is a continuing concern. Stepped care—similar to the stepped care used for asthma management—is a strategy to assess ongoing improvement, and adjust the treatment regimen if the woman is not moving toward full remission.[20]

Relapse

Relapse is common in persons who suffer from depression, with 20% to 85% of depressed individuals experiencing a second episode of depression within 2 years of the initial episode.[45] The risk of relapse is lower when antidepressant therapy is continued for 4 to 9 months after remission has been achieved, for a total duration of 6 to 12 months.[14,20,21,57] Individuals who have had or are at high risk for relapses will benefit from more long-term maintenance therapy. Characteristics of women for whom maintenance therapy should be considered are listed in **Table 8-10**. These women should be encouraged to continue therapy for at least 2 years, during which time provider–woman interactions may take place every 3 to 12 months as long as the woman's mental health status remains stable.[14,20,45,53] Due to the chronic nature of dysthymia and risk of episodes of double depression, many women with this disorder will benefit from ongoing, or even lifelong, treatment.[65]

Table 8-10	Indications for Possible Maintenance Antidepressant Therapy
Residual symptoms after adequate treatment	
Persistent sleep disturbance	
Family history of mood disorders	
Ongoing psychosocial stressors	
Three or more previous episodes of major depression	
Two or more closely spaced episodes	
Dysthymia	
Dysthymia with an episode of major depression (double depression)	

Based on American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013[13]; MacArthur Initiative on Depression and Primary Care. Depression management tool kit. 2009. Available at: http://www.integration.samhsa.gov/clinical-practice/macarthur_depression_toolkit.pdf. Accessed July 22, 2017.[55]

Discontinuing Psychotropic Medications

When a decision is made to discontinue psychotropic medications, the dose should be tapered over several weeks to months to avoid serotonin withdrawal syndrome.[32] It is beneficial to provide women with information regarding discontinuation when initiating therapy, as otherwise they may consider stopping the medication without consulting their provider. This caution should be reinforced at each follow-up encounter.

Anxiety and Stress Disorders

Anxiety disorders, a group of disorders characterized by anxiety and/or fear, include generalized anxiety disorder (GAD), panic disorder, and phobias (e.g., social anxiety disorder, specific phobias, and agoraphobia). Phobias are the most prevalent subcategory of anxiety disorders. Stress disorders and obsessive–compulsive disorders were previously categorized as anxiety disorders, but have been recategorized in *DSM-5* as separate categories —"Obsessive–Compulsive and Related Disorders" and "Trauma and Stressor-Related Disorders," respectively, with the latter category including PTSD.[13] As a group, anxiety disorders are more prevalent than mood disorders in the United States, yet they are less often screened for or detected in primary care practices.[66] Women are almost twice as likely as men to experience both anxiety and mood disorders.[4,66] Anxiety disorders cause significant stress; lead to impairment of social, family, or occupational functioning; and are out of proportion to any actual threat.

There are significant racial and ethnic disparities in the prevelence of anxiety disorders in the United States.[67] Most of the research on anxiety has been conducted with European-white participants, yet persons from underrepresented groups are less likely to have access to mental health services, are more likely to have comorbid conditions, and are differentially exposed to many of the risks for anxiety.[67] These factors need to be taken into consideration by clinicians, as they have significant effects on affected individuals.

All anxiety disorders, including OCD and PTSD, are frequently compounded by other anxiety or mood disorders—more so than other psychiatric disorders.[68] It is also common to have a three-way interaction that involves anxiety disorders, depression, and somatic symptoms, including respiratory disease, irritable bowel syndrome, chronic pain or malaise, fatigue, atopic disorders, and even cardiac disease. Anxiety disorders are highly internalizing conditions, which contributes to the associated somatic complaints.[68] Individuals with anxiety disorders may experience such somatic symptoms as palpitations, chest pain, shortness of breath, and dizziness that mimic symptoms of other diseases or conditions and lead to increased healthcare utilization and reduced quality of life.[68]

The reported lifetime prevalence of anxiety and its related disorders varies considerably from 14% to 33% perhaps secondary to different survey techniques and populations used as samples in the various studies.[69,70] The prevalence of other anxiety disorders also varies. Based on surveys administered in 2001 to 2003, lifetime prevalence rates in the United States are 13% for social anxiety disorder, 10.3% for PTSD, 4.1% for GAD, 2.7% for OCD, and 6.8% for panic disorder.[69,70]

Phobias and obsessive–compulsive disorder typically develop during childhood, adolescence, or early adulthood. Panic disorders and generalized anxiety disorder have significant variations in age of onset, as does post-traumatic stress disorder, due to the fact that trauma may occur at any age. Neuroimaging data suggest a significant neurobiological contribution to the development of anxiety disorders.[71]

While much public effort has been made to identify individuals with major depression, less attention has been directed at screening for individuals with anxiety disorders. An approach

for evaluation of a suspected anxiety disorder that is recommended by the British Association for Psychopharmacology is presented in **Figure 8-4**.[68] The *DSM-5* includes a more complex algorithm that captures all the possible differential diagnoses,[13] but this version can be helpful for initial screening performed by a midwife.

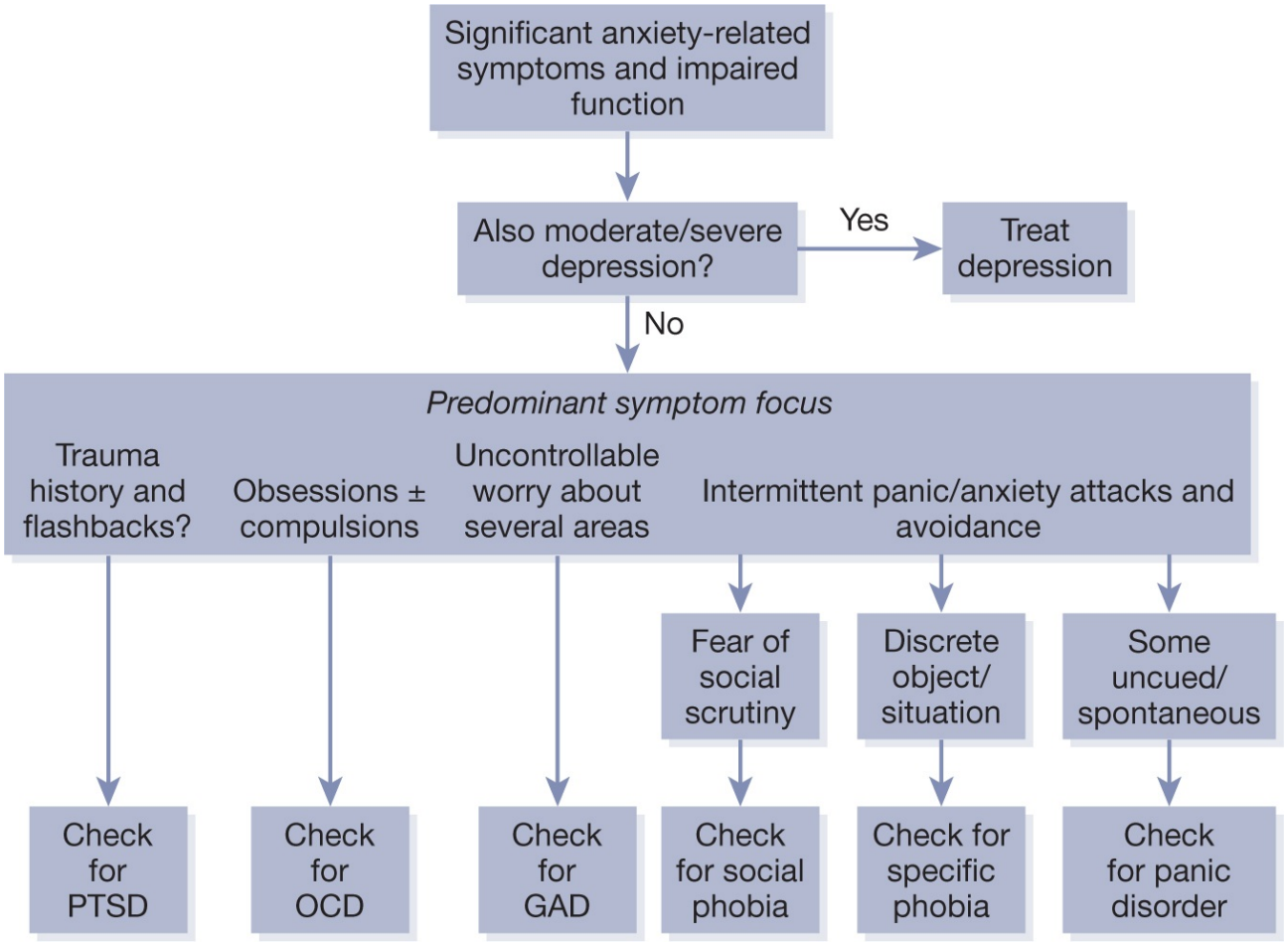


Figure 8-4 One scheme for exploration of a suspected anxiety disorder.

Abbreviations: GAD, generalized anxiety disorder; OCD, obsessive–compulsive disorder; PTSD, post-traumatic stress disorder.

Reproduced with permission from Baldwin DS, Anderson IM, Nutt DJ, et al. Evidence-based pharmacological treatment of anxiety disorders, post-traumatic stress disorder and obsessive-compulsive disorder: a revision of the 2005 guidelines from the British Association for Psychopharmacology. *J Psychopharmacol.* 2014;28(5):403-439.[68] Reprinted by permission of SAGE.

Screening for Anxiety Disorders

Anxiety disorders are underdetected and, therefore, undertreated. Similar to the PHQ-2 and PHQ-9 screening instruments, the GAD-2 and GAD-7 questionnaires **(Table 8-11)** have been developed and validated as means to screen for anxiety disorders. While the GAD-7 was initially developed specifically to screen for GAD, it has since been validated as a screen panic, social anxiety, and post-traumatic stress disorder, too.[72] This instrument was also found to be equally effective at screening among four racial/ethinc groups of undergraduate students:

African American, Caucasian, Hispanic/Latino, and Asian.[73] The GAD-2 questionnaire consists of the first two questions of the GAD-7, with a score 3 or higher suggesting the presence of an anxiety disorder.

Table 8-11 GAD-7: Generalized Anxiety Disorder Screening Tool

Over the last 2 weeks, how often have you been bothered by the following problems?	Not at All	Several Days	More Than Half the Days	Nearly Every Day
1. Feeling nervous, anxious, or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it is hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3
Column Scores	_____	_____	_____	_____

Total Score (add column scores) _____

Scores of 5, 10, and 15 are cut-off points for mild, moderate, and severe anxiety, respectively.
Further evaluation is recommended for a score ≥ 10.

Developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke, and colleagues, with an educational grant from Pfizer Inc. Available at: http://www.phqscreeners.com.

Phobias

A phobia is described as extreme or irrational fear or anxiety about a specific object or situation. This fear or anxiety is persistent, lasting more than 6 months.[13] Social anxiety disorder, also referred to as social phobia, is a specific phobia related to social situations in which the individual is exposed to potential scrutiny by others.[13] Studies suggest that phobias can be caused by a traumatic experience or may be learned behavior within a family environment. Maternal stress, overprotection, hypercriticism, abuse, and neglect may all contribute to the development of social phobia.

Generalized Anxiety Disorder

Generalized anxiety disorder is characterized by excessive worry or anxiety concerning family, health, finances, and work or school. A majority of women with GAD also experience other mental health disorders—most commonly depression or dysthymia, but also panic disorder and agoraphobia. As with all anxiety disorders, women are at greater risk for GAD compared to men.[74] Generalized anxiety disorder is associated with numerous somatic symptoms, leading to frequent primary care, specialist, and emergency department visits, and

absences from work.[74]

Generalized anxiety is characterized by the following signs:

- Excessive anxiety and worry (apprehensive expectation) about two (or more) domains of activities or events (e.g., family, health, finances, and school/work difficulties) occurs on more days than not, for 3 months or more.
- Anxiety and worry are difficult to control.
- Anxiety and worry are associated with restlessness and/or muscle tension.
- Anxiety and worry are associated with marked avoidance of activities or events with possible negative outcomes, excessive time and effort preparing for activities or events with possible negative outcomes, marked procrastination in behavior or decision making due to worries, and repeatedly seeking reassurance due to worries.[13]

Panic Attacks

Panic attacks are periods of intense anxiety or fear that have a sudden onset. Although these attacks can be expected to occur in the presence of a particular trigger, they can also be unexpected. The symptoms of a panic attack are dramatic and usually frightening to the person who is experiencing the event. The attack usually peaks at approximately 10 minutes, but full recovery can take days. Symptoms can include sweating, trembling or shaking, sensation of shortness of breath or smothering, nausea, chest pain or discomfort, dizziness, lightheadedness, unsteadiness or fainting, fear of losing control or going crazy, fear of dying, paresthesia, and chills or hot flashes. Individual attacks may occur in conjunction with any of the anxiety disorders, although the presence of recurrent attacks is characterized as panic disorder.[13]

Panic Disorder

The primary symptom of panic disorder is recurrent unexpected panic attacks, followed by (1) persistent concern or worry about additional panic attacks or their consequences (e.g., losing control, having a heart attack, “going crazy”) or (2) significant maladaptive change in behavior related to the attacks (e.g., behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).[13] Persons experiencing a panic attack may have tachycardia, chest pain, pounding heartbeats, nausea, dizziness, sweating, or shortness of breath.

Individuals who experience frequent anxiety or have a history of beaing fearful are more likely to have panic disorder. Additional risk factors include childhood physical or sexual abuse, as well as parents with a history of depression, anxiety, or bipolar disorder. There may be a genetic component to panic disorder as well.

Medications such as SSRIs, SNRIs, beta blockers, and benzodiazepines can be used to ease the symptoms and reduce the number of panic attacks, but the primary treatment for panic disorder is psychotherapy. Beta blockers can reduce some of the physical symptoms. Benzodiazepines are strong sedatives and can quickly ease anxiety; because they can cause tolerance and dependence, they are not recommended as a regular therapy. CBT appears to be

the most useful approach for treating women with this disorder.

Post-Traumatic Stress Disorder

Post-traumatic stress disorder is a response to a traumatic event that includes re-experiencing the event with nightmares, flashbacks, or other intrusive thoughts; avoidance of anything associated with the traumatic event and/or emotional numbing; and alterations in arousal such as sleep disturbance, difficulty concentrating, or aggressive behavior.[13] Precipitating events may include mass conflict, a natual disaster, population displacement, combat, sexual violation, serious illness, or life-threatening accident. The person with PTSD has either directly experienced a trauma, witnessed a traumatic event, or learned of a life-threatening event experienced by a close family member.

The diagnosis of PTSD is made when the symptoms cause distress or impairment in normal daily function for at least a month. The diagnostic criteria for PTSD are listed in **Table 8-12**. All of these criteria include significant distress or impairment in social, occupational, or other areas of function. To make a diagnosis of PTSD, the criteria cannot be attributable to direct physiologic effects of substances such as medication, drugs, or alcohol, nor can they be ascribed to another disease or condition.

Table 8-12 **Diagnostic Criteria for Post-Traumatic Stress Disorder**

1. Exposure to actual or threatened death, serious injury, or sexual violation by direct experience of the traumatic event or by witnessing the event affect a significant other, learning of the event occuring to a significant other, or experiencing repeated exposure to traumatic events
2. Presence of one or more of the following symptoms associated with the traumatic event(s), beginning after it occurred: intrusive, disturbing memories; distressing dreams; dissociative reactions; intense psychological distress in response to cues related to the event(s); and marked physiological reactions to reminders of the event(s)
3. Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the event(s) occurred, as evidenced by avoidance or efforts to avoid one or more of the following: distressing memories of the event, thoughts or feelings associated with the event, and external reminders of the event
4. Negative alterations in cognition and mood associated with the traumatic event(s), beginning or worsening after the event(s) occurred, as evidenced by two or more of the following: inability to remember an important aspect of the traumatic event(s); persistent and exaggerated negative beliefs or expectations about oneself, others, or the world; persistent, distorted blame of self or others about the cause or consequences of the traumatic event(s); persistent negative emotional state; markedly diminished interest or participation in significant activities; feelings of detachment or estrangement from others; persistent inability to experience positive emotions (e.g., unable to have loving feelings, psychic numbing)
5. Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by aggressive or destructive behaviors, hypervigilance, difficulty concentrating, and sleep disturbances

Based on American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 5th ed. Arlington, VA: American Psychiatric Association; 2013.[13]

Some subgroups in the United States have significantly higher prevalence of PTSD than the general population. Notably, women are 2 to 4 times more likely than men to develop PTSD.[75]

Sexual assault is the most frequent trauma leading to PTSD in women. A study of college-age women in 2000 found that approximately 20% to 25% of women in this age group were subjected to sexual assault or attempted sexual assault, leading to a large pool of women at risk for developing PTSD.[76] What we know about the complex relationship between pregnancy, birth, and PTSD is reviewed later in this chapter.

The most common treatments for PTSD are CBT, exposure therapy, and eye-movement desensitization and reprocessing (EMDR) therapy.[77] Psychotherapy employing exposure therapy and CBT has been found to be more effective than pharmacotherapy, although SSRIs are also used as treatment for PTSD. PTSD is often comorbid with depression or substance abuse, and persons with PTSD and substance abuse have more severe symptoms of both disorders.[76] Among the newer therapies for PTSD that appear promising is virual reality exposure therapy, which simulates the traumatic experience. Repeated exposure to the simulation reduces anxiety and fear related to that event.

Many individuals are exposed to traumatic events, yet only a minority develop PTSD. Resilience allows the majority of individuals who experience a traumatic event to recover without any long-lasting mental health disorder.[75] Further research is needed to explore factors that promote resilience.

Treatment of Anxiety Disorders

Both psychotherapy and pharmacotherapy play important roles in the treatment of individuals with any of the anxiety disorders. Like individuals with depression, those persons with anxiety disorders are at an increased risk of committing suicide when compared to the general population. Therefore, the clinician has a responsibility to screen for suicidal ideation in any woman with an anxiety disorder. The effectiveness of pharmacotherapy compared to psychotherapy is similar for individuals with anxiety disorders, and combined treatment has higher response rates than monotherapy.[78,79] The one exception is PTSD, for which psychotherapy appears to be the most effective form of therapy.

Cognitive-behavoral therapy, mindfulness-based cognitive therapy, exposure-based therapy, and therapist-supported Internet-based CBT have all been found to be effective for treatment of women with anxiety.[67,80,81] The same benefits seen with collaborative care models that employ a specially trained case manager for the treatment of women with depression apply to the treatment of women with anxiety.[82] Individuals with anxiety disorders are more likely to prefer CBT than medication alone.[82] It is unclear whether combining psychotherapy and medication offers the same benefit for anxiety as it does for depression.

When pharmacotherapy is indicated, SSRIs are the recommended first-line pharmacotherapy for GAD, generalized social anxiety, and PTSD.[78,83] The benefits of the SSRIs in the treatment of women with anxiety disorders are the same as the benefits for women with depression. These agents have been well studied, are safe and effective, and are well tolerated. As with depression, it may take up to 4 weeks for an individual to show a response; if no clinical response occurs by 6 weeks of treatment, there is a low likelihood of eventual reponse.[84] The same cautions that are decribed in the initiation of SSRIs in the area of depression apply for

anxiety. The woman should be counseled that she may experience a transient increased nervousness in the first few days of treatment, but that this usually dissipates within 2 weeks. The dosages for the treament of anxiety disorders are similar to those for depression.

Benzodiazepines, usually clonazepam (Klonopin) or lorazepam (Ativan), may be useful for short-term use by women with significant impairment while waiting for the therapeutic effect of an SSRI.[85] These medications may also be useful for the episodic treatment of individuals with panic disorder. Benzodiazepines can be highly addictive, cause drowsiness, may aggravate depression, and should be used with caution with older adults. The risk of depression is higher in individuals who have other dependencies.[86] These risks must be balanced against the benefits that benzodiazepines can offer with short-term or intermittent use.

Mental Health Disturbances Related to the Menstrual Cycle

Mental health disturbances related to the menstrual cycle are categorized into "core" and "variant" groups (**Table 8-13**).[13,87-90] The core premenstrual disorders include premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD). Approximately 20% to 30% of menstruating women have PMS, and 1.2% to 6.4% have PMDD, which is a severe form of PMS.[87,89] Variant premenstrual disorders include those not associated with a regular ovulatory cycle.

Table 8-13 Classification of Premenstrual Disorders: Consensus Criteria of the International Society for Premenstrual Disorders, 2008[a]

Definition	Characteristics
Core Premenstrual Disorders	
PMS	Multiple symptoms appear only during the luteal phase of an ovulatory cycle and are relieved within 4 days of the onset of menses without recurrence until the approximate time of ovulation. Must not be an exacerbation of another condition. Symptoms are severe enough to interfere with daily function and cause significant distress. Affective symptoms include angry outbursts, anxiety, depression, irritability, confusion, and social withdrawal. Somatic symptoms include abdominal bloating, breast tenderness, headache, joint or muscle pain, extremity swelling, and weight gain.
PMDD	Classified separately from PMS based on severe psychological symptoms that occur during the luteal phase and remit entirely during the rest of the menstrual cycle.[b] • At least 5 symptoms are present in the final week before menses. They improve within a few days of the onset of menses, and become minimal or absent in the week after menses. • One or more of the following symptoms must be present: marked affective lability, marked irritability, marked depressed mood, and marked anxiety or tension. • One or more of the following must be present to reach the total of five symptoms: decreased interest in usual activities, difficulty concentrating, lethargy, change in appetite, hypersomnia or insomnia, sense of being overwhelmed, and physical symptoms such as breast tenderness or abdominal bloating. • Symptoms are associated with significant distress and not an exacerbation of another disorder or attributable to the effects of a substance or medication.
Variant Premenstrual Disorders	
Premenstrual exacerbation	Luteal-phase exacerbation of another condition such as depression, diabetes, migraine, epilepsy, or asthma
Nonovulatory premenstrual disorders	Symptoms result from ovarian activity other than ovulation
Progesterone-induced premenstrual disorders	Women receiving exogenous progestogens may develop PMS symptoms
Premenstrual disorders without	Symptoms arise from continuous ovarian activity, although menstruation is suppressed

menstruation

Abbreviations: PMDD, premenstrual dysphoric disorder; PMS, premenstrual syndrome.

[a] Diagnosis is based on structured interview, self-report, and prospective recording of at least two menstrual cycles.

[b] See *DSM-5* for a full description of the diagnostic criteria.

Based on American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013[13]; O'Brien PMS, Bäckström T, Brown C, et al. Towards a consensus on diagnostic criteria, measurement and trial design of the premenstrual disorders: the ISPMD Montreal consensus. *Arch Womens Ment Health.* 2011;14(1):13-21[88]; Ismaili E, Walsh S, O'Brien PMS, et al. Fourth consensus of the International Society for Premenstrual Disorders (ISPMD): auditable standard for diagnosis and management of premenstrual disorder. *Arch Womens Ment Health*. 2016;19:953-958.[90]

Premenstrual Syndrome and Premenstrual Dysphoric Disorder

The core premenstrual disorders are characterized by timing of somatic and/or psychological symptoms that occur during all or part of the 2-week premenstrual phase and resolve during or after menses starts in women with ovulatory cycles. Women with one of these disorders have a symptom-free interval between menses and ovulation.

The etiology of premenstrual disorders is poorly understood, but cyclic changes in progesterone and estrogen levels appear to trigger the symptoms. Premenstrual disorders share features associated with anxiety and depression, which have been linked to serotonergic dysregulation. It is thought that changes in estrogen and progesterone levels affect serotonin and dopamine levels, although other neurotransmitters may also be involved.[87,88,91–93] Recent research suggests PMDD may be the result of extreme neurologic sensitivity to normal hormone variations.[92] Genetic vulnerability probably also plays a role in determining which women experience PMDD.[93]

Diagnosis of PMS or PMDD is based on nature and number of symptoms, cyclicity, severity, and chronicity of symptoms.[94-96] The Daily Record of Severity of Problems, an instrument validated by Endicott et al., is the most widely used daily symptom scale because it measures all 11 of the *DSM-5* symptoms of PMDD.[95] This record can be printed and used by women to record their symptoms over a few months.

PMDD is a psychiatric illness that affects approximately 5% of women during their fertile years.[89] It is the most severe, yet least common, of the menstrual disorders. The most frequent and disabling symptoms of PMDD are extreme irritability and mood lability. Disordered sleep (either insomnia or hypersomnia) and extreme changes in tension or energy are also reported. Confusion and alterations in mental status are prominent in PMDD, which can result in women being unable to work. Women experiencing PMDD may have difficulty with relationships on the days that they are symptomatic. They may also experience comorbidity of their PMDD with other mood or anxiety disorders such as MDD, bipolar disorder, or OCD.[89]

Treatments for PMS and PMDD

Treatments for PMS and PMDD include: (1) nonpharmacologic approaches such as diet, exercise and psychotherapy; (2) SSRIs; (3) hormonal agonists and antagonists; and (4)

vitamins and botanical products.[87]

Nonpharmacologic approaches may help stabilize mood and relieve physical symptoms, but there is little evidence to support these approaches as monotherapy. A diet that includes complex carbohydrates, consumed during the luteal phase, may be helpful, as this type of diet boosts the amount of serotonin present in neural synapses.[87] CBT can help women manage their symptoms.

The strongest evidence for effective treatment of women with PMDD involves boosting neurotransmitter presence or suppressing ovulation. Within the neurotransmitter approach, the SSRIs are the most-studied antidepressants used as pharmacotherapy for women with PMDD. All of the SSRIs appear to be equally effective, however, and both continuous and intermittent dosing regimens are used. A moderate dose may be effective and well tolerated.[97] The action of SSRIs for PMDD differs from their action for other psychiatric conditions in two basic ways:

- Relief of symptoms occurs in the first cycle of use. Ordinarily, SSRIs require up to 2 months before reaching full effectiveness.
- The SSRI is effective when taken only at intervals, rather than every day. Withdrawal symptoms do not appear when the SSRI is discontinued after several days or a week of use.

The second strategy aims to create a steady hormonal state, without the cyclic fluctuations that appear to trigger the neurologic cascade resulting in PMDD. Hormonal contraceptives can suppress ovulation and are widely acceptable to women who do not want to become pregnant. Oral contraceptives containing drospirenone (e.g., Yaz, Yasmin) have been the most specifically studied for their effectiveness in treating PMDD and PMS.[98] Women who use drospirenone-containing contraceptives have an increased risk for deep vein thrombosis, which must be taken into consideration before prescribing these agents. Fluctuation of hormone levels can be further minimized by using long-cycle or continuous-dosing regimens.[99] Long-term suppression of ovulation, using leuprolide acetate (Lupron, Lupron Depot) plus add-back estrogen, may be effective.[100]

Premenstrual Exacerbation of Psychiatric Conditions

Much more common than PMDD is the worsening of other psychiatric conditions that follows the menstrual cycle. Mood and anxiety disorders are more prevalent in the menstruating population than is PMDD. Because women themselves often feel confused about the causes of their distress, it is important to carefully identify menstrual-related symptoms so as to correctly diagnose and address the underlying condition. Reviewing the woman's daily symptom diary can be key to distinguishing between PMDD or another psychiatric diagnosis.

Fortunately, the PMDD treatment strategies can help women with other conditions. Hormonal contraceptives have been used as an adjunct treatment for underlying depression that worsens prior to menses,[101] and the midwife who is familiar with the range of products and dosing regimens can fine-tune treatment for an individual woman's needs in consultation with

the woman’s psychiatric provider.

Women with psychiatric illness may have difficulty making sound decisions in their own best interest. Depression, for example, can slow cognition and impair memory; anxiety can be a force for confusion and distraction. Women with bipolar illness may have difficulty with impulse control and irritability. In working with these women to minimize their symptoms across the menstrual cycle, midwives need to remain mindful of the possible impaired decision making that mental illness can cause in some persons. It may be necessary to consult with the woman’s mental health clinicians to determine the most effective plan of care. In some situations, inclusion of significant others or family members in making a plan can be helpful as well.[102]

Perinatal Mental Health

Childbearing is a time of profound change. An increasingly robust body of literature links the mental health of the mother with the optimal outcome of the pregnancy and birth, and with the cognitive and social development of the offspring.[103] Women with mental health disorders may struggle to negotiate the demands of parenthood, and when they falter, their children—and other family members—may be affected as well.*104* Women with a history of depression who are not aware of being depressed after childbirth may be less engaged with their infants than women without a history of depression. This lack of a close bond may, in turn, affect the infant's neurochemical and neuroendocrine development.[105]

Women are more likely to be diagnosed with a psychiatric disorder during their fertile years than at any other time in their lives. In addition, women are most vulnerable to psychiatric illness while they are pregnant or during the postpartum period.[106]

Mental Health Disorders During Pregnancy

The prevalence of various psychiatric conditions during pregnancy mirrors their prevalence in the general population. Mood and anxiety disorders predominate in women across sociodemographic and age groups; they are also the most commonly diagnosed psychiatric conditions during pregnancy.[3] Approximately 10% to 20% of women will experience minor or major depression during pregnancy.[107] Notably, this incidence may be much higher in some populations that are traditionally not included in epidemiologic surveys. For example, approximately one in three women who migrate from low- and middle-income countries have minor or major depression during pregnancy.[107,108]

Untreated depression and anxiety disorders can adversely affect the course and outcome of pregnancy.[109-112] Untreated depression or anxiety during pregnancy is associated with substance abuse, smoking, and other behaviors that can independently affect pregnancy outcomes. Women with untreated depression have increased risks of preterm birth and fetal growth restriction. In addition, these women are at increased risk for postpartum depression and their children are at increased risk for developmental and behavioral problems.[109-112] The strongest predictors of depression during the postpartum period can be identified during pregnancy, including depression or anxiety during pregnancy, stressful life effects and poor social support during pregnancy, and a history of depression at any other time in a woman's life.

Screening for Depression and Anxiety During Pregnancy

The U.S. Preventive Services Task Force, the American College of Obstetricians and Gynecologists, and the American College of Nurse-Midwives all recommend regularly screening women for perinatal depression.[113-115] In addition, many states mandate that providers screen for depression during pregnancy. Any woman who has risks for or symptoms of depression should be screened two or three times during pregnancy, and all women should be screened at least once. The Council on Patient Safety in Women's Health Care has developed an interdisciplinary patient safety bundle to address maternal mental health that addresses readiness, recognition, prevention strategies, response, and reporting systems for both clinical settings and for evaluating individual women.[115] The list of resources at the end of this chapter has more information about accessing this patient safety bundle.

Symptoms of prenatal depression include crying, weepiness, sleep problems, fatigue, appetite disturbances, anhedonia, anxiety, problems with fetal attachment, and irritability. Because many of these symptoms can also be normal transient problems during pregnancy, formal screening to differentiate normal symptoms from major or minor depression is essential.

General screening for perinatal mental health disorders should also include an assessment for thyroid abnormalities that can trigger psychiatric symptoms. Elevations in thyroid-stimulating hormone (TSH), free T_4, and thyroid antibodies have been associated with depression during pregnancy and postpartum. A study by Sylvén et al. found that an abnormal TSH level at the time of birth was associated with an elevated risk for postpartum depression

at 6 months following birth (OR, 11.30; 95% CI, 1.93-66.11).[116]

Many instruments are available for screening during pregnancy (**Table 8-14**).[113-119] The PHQ-9 and the GAD-2[72] are quick, cost-effective ways to identify those at risk, but their inclusion of constitutional symptoms that are common in pregnancy reduces their specificity. The Beck Depression Inventory has been validated for use during pregnancy but is copyrighted and requires a fee for its use. All of these scales have been translated and validated for many different languages.

Table 8-14 Screening Tools for Perinatal Depression

Screening Tool	Methods of Administration	Time to Complete (min)	Sensitivity (%)	Specificity (%)
Beck Depression Inventory II	21 items self-administered or interview	5–10	56–57	97–100
Center for Epidemiologic Studies Depression Scale	20 items self-administered	5–10	60	92
Edinburgh Postnatal Depression Scale	10 items self-administered	< 5	59–100	49–100
Patient Health Questionnaire-9	9 items self-administered	< 5	75	95
Postpartum Depression Screening Scale	35 items self-administered	5–10	91–94	72–98
Zung Self-Rating Depression Scale	20 items self-administered	5–10	45–89	77–88

Based on American College of Obstetricians and Gynecologists. Committee Opinion No. 630: screening for perinatal depression. *Obstet Gynecol*. 2015;125:1268-1271[113]; Gaynes BN, Gavin N, Meltzer-Brody S, Lohr KN, Swinson T, Gartlehner G, Brody S, Miller WC. *Perinatal Depression: Prevalence, Screening Accuracy, and Screening Outcomes. Evidence Report/Technology Assessment No. 119*. AHRQ Publication No. 05- E006-2. Rockville, MD: Agency for Healthcare Research and Quality; 2005.[118]

Originally developed in the United Kingdom by Cox et al., the Edinburgh Postnatal Depression Scale (EPDS) has been validated for use both during the pregnancy and postpartum in a wide variety of populations and is the most commonly used screening tool (**Table 8-15**).[113,118] It consists of a self-report series of 10 questions that takes approximately 5 minutes to complete. It is freely available to any who want to use it. The EPDS has been validated for many populations and several languages, and it has a higher level of accuracy for women in low-resource settings compared to other tools. However, some translations of this tool have not been tested for cultural sensitivity or comprehensibility in different languages, and it may not be as accurate when used by women whose native language is not English. The EPDS, which also assesses for anxiety, can be used to monitor progress in women over the course of pregnancy and postpartum (**Figure 8-5**).[119]

Table 8-15	Edinburgh Postnatal Depression Scale
1. I have been able to laugh and see the funny side of things.	**0** As much as I always could **1** Not quite so much now **2** Definitely not so much now

	3 Not at all
2. I have looked forward with enjoyment to things.	**0** As much as I ever did **1** Rather less than I used to **2** Definitely less than I used to **3** Hardly at all
3. I have blamed myself unnecessarily when things went wrong.	**3** Yes, most of the time **2** Yes, some of the time **1** Not very often **0** No, never
4. I have been anxious or worried for no good reason.	**0** No, not at all **1** Hardly ever **2** Yes, sometimes **3** Yes, very often
5. I have felt scared or panicky for no good reason.	**3** Yes, quite a lot **2** Yes, sometimes **1** No, not much **0** No, not at all
6. Things have been getting on top of me.	**3** Yes, most of the time I haven't been able to cope at all **2** Yes, sometimes I haven't been coping as well as usual **1** No, most of the time I have coped quite well **0** No, I have been coping as well as ever
7. I have been so unhappy that I have had difficulty sleeping.	**3** Yes, most of the time **2** Yes, quite often **1** Not very often **0** No, not at all
8. I have felt sad or miserable.	**3** Yes, most of the time **2** Yes, quite often **1** Not very often **0** No, not at all
9. I have been so unhappy that I have been crying.	**3** Yes, most of the time **2** Yes, quite often **1** Only occasionally **0** No, never
10. The thought of harming myself has occurred to me.	**3** Yes, quite often **2** Sometimes **1** Hardly ever **0** Never

The scores for each item are totaled. Scores greater than 12 require evaluation and possible referral to a mental health specialist. Scores between 10 and 12 indicate the presence of symptoms of distress. The test should be repeated in 1 to 2 weeks and referral considered. Scores between 0 and 9 indicate that any symptoms of distress may be short-lived and not likely to interfere with day-to-day function. If they persist more than 1 to 2 weeks, further evaluation is recommended.

Reproduced with permission from Cox JL, Holden JM, Sagovsky R, Detection of postnatal depression: development

of the 10-item Edinburgh Postnatal Depression Scale. *Br J Psychiatry.* 1987;150:782-786.[117]

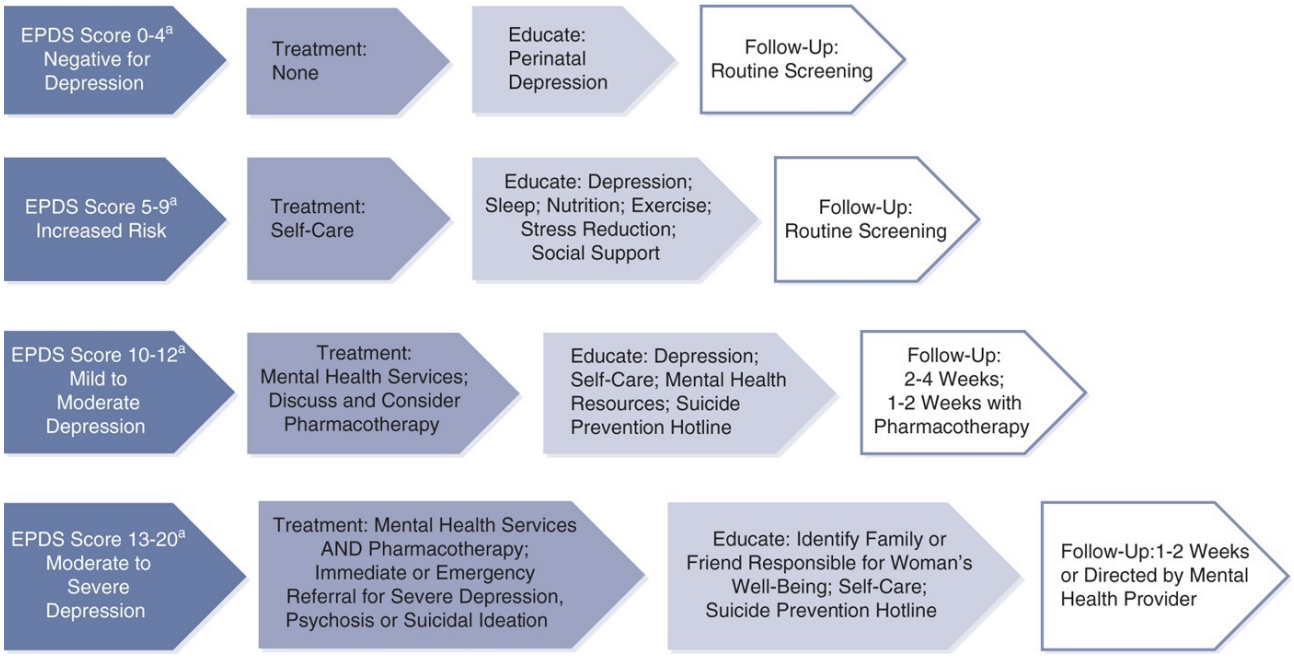


Figure 8-5 Screening and treatment algorithm for perinatal depression.

Abbreviation: EPDS, Edinburgh Postnatal Depression Scale.

a Regardless of EPDS score, a response indicating thoughts of harming self or others should be addressed directly

Reproduced with permission from Latendresse G, Deneris A. Selective serotonin reuptake inhibitors as first line antidepressent therapy for perinatal depression. *J Midwifery Womens Health.* 2017; 62(3):317-328.[119] © 2017, with permission from Wiley.

Three points must be kept in mind when using one of these screening tools:

- The screening tools detect an increased risk for depression, but do not *diagnose* depression. The diagnosis is made via a more in-depth assessment by a mental health professional.
- Any answer that indicates the woman is severely depressed or may have suicidal ideation must be explored further and may be a medical emergency requiring immediate evaluation. Thus, screening should be done within a system that provides access to mental health providers.
- It is extremely important to distinguish unipolar depressive illness from bipolar depression, as the treatment and prognosis for each of these disorders are quite different, and because use of an antidepressant medication may exacerbate bipolar illness.[115]

Psychiatric illnesses have a high comorbidity with each other; therefore, when one disorder is identified in a pregnant woman, the possibility that more than one condition may be present should be considered. A thorough history will reveal previous episodes of mental illness experienced by the pregnant woman or members of her family. Whenever possible, contact the mental health professionals who have cared for the woman in the past to confirm her account, as mental disorders may cloud the memory. Women who have discontinued their psychotropic

medication before becoming pregnant should be counseled about the significant risk of relapse during pregnancy; as many as 68% of women with major depression[106] and 70% of women with bipolar disorder who discontinue psychotropic medication will relapse during pregnancy.[103,120]

Risk factors such as onset of depression before age 18 years, individual or family history of suicide attempts, and multiple unsuccessful trials of antidepressant medications often predict a bipolar origin of depression. Even if a woman with bipolar disorder has been stable for some years before pregnancy, she is at risk for relapse of illness during her pregnancy. Aggressive treatment of women with bipolar illness and protection of sleep are key elements in preventing mania and psychosis postpartum; such measures can reduce but not eliminate these risks.[121]

Anxiety disorders can also develop de novo or relapse during pregnancy, and it can be difficult to differentiate the normal parental anxieties from disabling illness. GAD appears to have a prevalence of approximately 7% among pregnant women, but the risk may be greater for women with a history of child abuse, poor social support, or previous episodes of anxiety.[122] Obsessive–compulsive symptoms can be present during pregnancy, tend to become worse postpartum, and are highly comorbid with depressive illness.[123]

Pharmacotherapy for Perinatal Depression

A mental health disorder that appears or recurs during pregnancy requires collaborative management by the woman and her mental health clinicians. Women who have a history of major mental illness, such as bipolar disorder, eating disorders, and PTSD, need to be comanaged with a mental health clinician who helps to develop the plan of care. The combination of psychotherapy with psychotropic medication has been shown to have higher remission and lower relapse rates than either type of treatment alone.

Midwives who are knowledgeable about both depression and the pharmacology of the medications used to treat depression may prescribe these medications. Safe practice requires that the midwife has access to mental health resources and can provide continuity of care, and that the woman does not have symptoms indicating the need for care by a mental health professional, such as suicidal ideation or a history of hospitalization. Many of the SSRIs are associated with significant drug–drug interactions, so it is important that a prescribing midwife obtain a history of all medications and other products the woman is taking, and be aware of any potential interactions. Women who have multiple comorbidities are best managed by a mental health specialist.

The choice to use medication during pregnancy is complex. Randomized controlled trials (RCTs) are impossible to conduct with pregnant women as participants, leaving clinicians to consider the conflicting findings of many observational, anecdotal, retrospective, and open trials of the various antidepressant medications. In addition, many studies conflate possible medication side effects. Thus, to date, no absolute rules for prescribing antidepressants during pregnancy have emerged. Each woman needs to make an individual choice based on her diagnosis, the likely progression of her illness, and the results of shared decision making. Information provided as part of shared decision making will include possible adverse effects over the course of the pregnancy, including risks of no treatment, fetal effects, newborn effects,

and neurobehavioral effects on the child. The risks of not treating depression must be balanced against possible risks associated with the medication. For example, infants whose mothers had perinatal depression have higher cortisol levels than infants whose mothers were not depressed; these elevations continue through adolescence. Treatment of women with depression during pregnancy appears to normalize this elevation.[124]

Potential Risks Associated with SSRI Use During Pregnancy

Approximately 7% to 13% of women in the United States use an antidepressent during pregnancy.[125] SSRIs are the most commonly used pharmacotherapy for treating perinatal depression and as their use increases, information about positive and adverse effects is rapidly changing. Studies that have found associations between antidepressents and perinatal outcomes have many methodologic challenges, including small sample sizes, recall bias, self-reported data, and outcome measures that are too rare to detect. Thus, large epidemiologic population-based observational studies are the current best resource for detecting an association between medication taken during pregnancy and an adverse outcome, and these studies are not able to conclusively determine an etiologic relationship. That said, **Table 8-16** summarizes the known risks associated with SSRI use during pregnancy and lactation.[119–134]

Table 8-16 Risk Summary for SSRIs Used During Pregnancy and Breastfeeding

Risk	Description
Spontaneous abortion	Well-controlled studies indicate no increase in risk.
Congenital malformations	Well-controlled studies indicate no increase in risk for major congenital malformations in association with SSRIs as a class of drugs.
Congenital cardiac malformations	Well-controlled studies indicate increased risk is associated only with paroxetine (Paxil) (OR, 1.28; 95% CI, 1.11–1.47). May warrant echocardiogram for exposed fetuses.
Preterm birth	Depression and SSRIs are both consistently associated with an increased risk for preterm birth, but it is not clear if this increase is related to depression or to the medication.
Low birth weight	Most well-controlled studies indicate no increase in risk. Possible risk (OR, 2.26; 95% CI, 1.31–3.91).
Postpartum hemorrhage	Controlled studies indicate possible increased risk for postpartum hemorrhage (RR, 1.53; 95% CI, 1.25–1.86) and severe postpartum hemorrhage (RR, 1.84; 95% CI, 1.39–2.44).
Persistent pulmonary hypertension of the newborn	Controlled studies indicate low absolute risk increase (OR, 1.28; 95% CI, 1.01–1.64).
NAS	Controlled studies indicate substantially increased risk for NAS (OR, 5.07; 95% CI, 3.25–7.90). This is more common with fluoxetine (Prozac) and paroxetine (Paxil). Reports of mild NAS in 22% of exposed neonates, hypoglycemia in 19%, and severe NAS in 3% of newborns. May be attributed to SSRI withdrawal or serotonin syndrome. Clinical monitoring of exposed neonates recommended.
Autism spectrum disorders	Conflicting reports. No well-controlled studies. Possibly increased risk (OR, 1.81; 95% CI, 1.47–2.24). Association may be due to psychiatric disorder rather than SSRI exposure.

Effects on breastfeeding infant	Very little evidence of adverse effects on breastfeeding infants. Some case reports of newborn drowsiness, fussiness, irritability, feeding difficulty, hypertonia, hypotonia, weight loss, colic, and withdrawal syndrome with breastfeeding cessation.

Abbreviations: CI, confidence interval; NAS, neonatal adaptation syndrome; OR, odds ratio; RR, relative risk; SSRI, selective serotonin reuptake inhibitor.

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Reputable evidence-based online resources are available to clinicians and women to help guide decisions about medication use during pregnancy and breastfeeding. Some of these resources are listed in the Resources section at the end of this chapter.

Although there is no evidence that some medications work better during pregnancy than other agents do, recommendations for selecting SSRIs for use in pregnant women are evolving. It is generally recommended that treatment be initiated with sertraline (Zoloft) or citalopram (Celexa). Fluoxetine (Prozac) is not considered a first-line choice due to its long half-life. Note that citalopram is associated with elevated serum levels in breastfed infants.

In general, dosing regimens for pregnant women are similar to those for nonpregnant women, albeit with these two considerations:

- Blood volume expansion begins early in pregnancy, and hemodilution usually necessitates progressively higher doses of medication across the pregnancy to achieve a stable clinical effect.[135]
- Because of shifting gestational hormones, metabolism and excretion of medications can vary across pregnancy. This can alter the effectiveness of medication, and is another reason why monitoring psychiatric symptoms throughout the pregnancy is important.

Complementary Therapies for Perinatal Depression

Some women who opt not to participate in therapy or take psychotropic medications consider complementary therapies to help manage their perinatal depression. Omega-3 fatty acids, especially eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have well-established health benefits for pregnancy and infant development. They have also been found to be beneficial in the treatment of individuals with major depression, primarily as adjunct therapy rather than as monotherapy.[37] Results of studies in the treatment of antenatal depression have been mixed. Omega-3 supplements are well tolerated, and psychiatrists at the University of Massachusetts Medical School recommend 1 g EPA plus DHA daily for pregnant women with depression.[37]

S-Adenosyl-methionine (SAMe) also has an antidepressant effect. Although it has been investigated as a treatment for intrahepatic cholestasis of pregnancy, and was not associated with adverse effect, it has not been well studied for use in the management of depression during pregnancy.[37]

St. John's wort (*Hypericum perforatum*) is an herbal dietary supplement that is used in the treatment of individuals with depression. Studies of its effectiveness in reducing depression

have yielded varied results; one meta-analysis done in Germany found that St. John's wort was comparabe to SSRIs, and better for a subgroup of individuals with mild to moderate depression.[136] St. John's wort induces the cytochrome P450 system, and has multiple drug interactions. There have been only limited studies of its use during pregnancy or breastfeeding, so it is not recommended for use in the perinatal period.[37]

Bright light therapy is a safe and effective treatment for pregnant and breastfeeding women once bipolar disorder has been ruled out. Moderate exercise for 30 minutes per day reduces depressive symptoms, and has been recommended by the American College of Obstetricians and Gynecologists for general well-being during pregnancy. Massage therapy can also be recommended as an adjunct therapy. Acupunture therapy may have a benefit in some women.[37]

Post-Traumatic Stress Disorder During the Perinatal Period

The perinatal period can be especially difficult for women who have a history of physical, emotional, or sexual abuse. Women who have PTSD may experience pregnancy, labor, or birth as traumatic. Women who do not have symptoms of PTSD prior to pregnancy may develop this condition when they experience physical vulnerability during pregnancy, and intimate contact during vaginal examinations, labor, and birth. One in five women has a history of a childhood maltreatment.[137] Midwives need to understand the sequelae of sexual abuse in particular, so as to offer these women safe therapeutic care.[137]

Women who have experienced childhood trauma, sexual abuse, depression, or PTSD preceding the pregnancy are at increased risk for PTSD during the perinatal period.[138] The reported incidence of PTSD during pregnancy ranges from 3% in high-resource settings to more than 14% in low-resource settings.[137] Anxiety sensitivity reflects a tendency to respond fearfully to anxiety itself (as opposed to just responding to stressors) and has been found to increase vulnerability to PTSD.[139]

Depression, anxiety, and fear of childbirth during the pregnancy are also predictors of postpartum PTSD. The traumatic stressor for postpartum PTSD is birth trauma, "an event occurring during the labor and delivery process that involves actual or threatened serious injury or death to the mother or her infant."[140] Thus, it is the *perception* of risk that predisposes women to PTSD rather than the *absolute* risk. For example, a woman may perceive her birth as traumatic depsite the perception by the clinicians that everything was routine.[140]

Not surprisingly, emergency cesarean birth and instrumental birth are associated with PTSD—but so are negative emotions such as a negative experience of labor and birth and a loss of control. A sense of not receiving adequate support—especially from care providers, but also from a partner—can contribute to the risk for postpartum PTSD.[138]

Being alert to risk factors for PTSD and signs of crisis, such as withdrawal, signs of acute distress during labor, or dissociative behaviors, may allow midwives to identify women at high risk for PTSD, and provide the opportunity to offer trauma-informed care during pregnancy,[137] provide more intensive support and communication during labor, and make an early referral if indicated at any time in the perinatal period. Limited research has addressed treatment for PTSD specifically following childbirth; those studies that have been conducted

suggest that many of the same therapies that are employed for PTSD in general have a benefit for women with postpartum PTSD. These include counseling, cognitive-behavioral therapy, and eye-movement desensitization and reprocessing.

Postpartum Mood Disorders

Changes in mood are experienced by a majority of women after childbirth. Estrogen levels, which have a significant influence in neurotransmitter systems, decrease during the postpartum period. In addition, lower serotonergic activity following birth has been noted. Downregulation of the hypothalamic–pituitary–adrenal axis following birth may also contribute to increased vulnerability to depression.[115] While these physiologic changes do not lead to depression in all postpartum women, when they are coupled with a history of prior depression or bipolar disorder, a genetic predisposition, inadequate social support, and multiple stressors, it is not surprising that childbirth can present a challenge to a woman's mental health and well-being. Disturbances in sleep patterns and circadian rhythms are also universal in new mothers. Melatonin release is suppressed by exposure to light when a person would normally be sleeping.[141] Postpartum insomnia may both contribute to and be aggravated by MDD.[142]

A description of and diagnostic criteria for the most common postpartum mood disorders appear in **Table 8-17**.[143]

Table 8-17	Diagnostic Criteria for Postpartum Depression Disorders

Postpartum Blues (Baby Blues)
Affects 80% of new mothers Onset: first 7–10 days after birth Peak prevalence: postpartum days 3–5 No specific diagnostic criteria Most common symptom: weepiness Other symptoms: mood lability, feeling overwhelmed, sadness and frustration, fatigue/exhaustion
Postpartum Depression
Affects 10–20% of new mothers Onset: first 2–3 months postpartum Peak prevalence: 2–6 months postpartum Must include depressed mood or anhedonia and any of the following symptoms, which occur nearly every day for at least 2 weeks and are severe enough to impede function: 1. Sleep disorder (insomnia or hypersomnia) a. Must be evaluated in the context of the expected sleep pattern with a newborn b. Can the woman sleep when her infant is asleep? 2. Interest deficit or a lack of feeling pleasure a. Inability to appreciate infant, not bonding b. Persistent sadness 3. Guilt (worthlessness, hopelessness, regret) a. More common than feeling sad b. Feeling inadequate as a mother 4. Fatigue or loss of energy nearly every day a. Feeling of being overwhelmed, unable to cope

5. Concentration deficit
    a. Persistent difficulty with normal daily tasks
    b. Difficulty remembering things
6. Appetite disorder (increased or decreased)
    a. Is the woman drinking enough and urinating regularly based on normal postpartum physiology?
7. Psychomotor retardation or agitation
    a. Unable to get out of bed or go to sleep
    b. Loss of interest in caring for oneself
    c. Obsessive thoughts of harm to the infant
8. Suicidality (recurrent thoughts of death)
    a. Thoughts of harming oneself
    b. Do you wish you could go to sleep and not wake up? (passive suicidal ideation)
    c. Do you think about ways to kill yourself? (active suicidal ideation)

Postpartum Psychosis
Affects 12 women per 1000 live births
Onset: usually starts within 2 to 4 weeks of birth, but can start as early as 2 to 3 days after birth and often progresses rapidly
Emergency psychiatric condition characterized by symptoms of:
Delusional beliefs, hallucinations, and disordered thinking
"Everything is black or dark"
Feeling of hopelessness
Delusions about the infant
Auditory hallucinations, which can be instructions to harm the infant
Visual hallucinations

Modified with permission from NIHCM Foundation. *Identifying and Treating Maternal Depression: Strategies and Considerations for Health Plans.* Issue Brief. Rockville, MD: U.S. Public Health Service, Health Resources and Services Administration, Maternal and Child Health Bureau; 2010. Available at: https://www.nihcm.org/pdf/FINAL_MaternalDepression6-7.pdf. Accessed June 21, 2017.[143]

Postpartum Blues

Postpartum blues, also known as baby or maternity blues, is the most common mood change, occurring in 50% to 75% of new mothers.[144] The symptoms of crying, anxiety, emotional lability, irritability, and fatigue peak within 2 to 5 days of their onset, but usually do not interfere with functioning.[144,145] Although postpartum blues is usually benign and self-limited, it can be frightening to a woman. If a woman in the early postpartum period calls with concerns about her emotional symptoms, it is prudent to ask the two questions of the PHQ-2 about (1) finding pleasure in things and (2) feeling predominately down, depressed, or hopeless. If the woman reports anhedonia, a more thorough screen for postpartum depression is warranted. Many practices regularly use the Edinburgh Postpartum Depression Scale to screen for postpartum depression, especially for women who report emotional distress.

The primary differences between postpartum blues, postpartum depression, and postpartum psychosis relate to duration and severity of the symptoms. Characteristics to help the clinician

distinguish between these conditions when assessing a woman who reports disordered mood during the postpartum period are listed in **Table 8-18**.

Table 8-18 Common Clinical Features That Distinguish Postpartum Blues and Postpartum Depression[a]

	Characteristics				
Disorder	**Onset**	**Duration**	**Characteristic Symptoms**	**Degree of Disability**	**Suicidal Ideation**
Postpartum blues	7–10 days after birth	Less than 2 weeks	Crying, sad, labile mood	Minimal; cares for infant without problem	Unlikely
Postpartum depression	2 months to 1 year after birth	More than 2 weeks	Anhedonia, sleep disturbance, guilt, feeling of isolation and loneliness	Severe; may have difficulty caring for infant without help	Possibly present
Postpartum psychosis symptoms	First 2–4 weeks after birth	Mean duration: approximately 40 days	Acute onset of psychosis and delusional thoughts	Severe; psychiatric emergency	Likely; may also have thoughts that the infant is being harmed

[a] This list is a selected group of symptoms that can aid a clinician during the initial evaluation of a woman on the phone or in a triage setting.

Once postpartum blues are determined to be the likely cause of symptoms, anticipatory guidance that these mood swings are commonly experienced and usually resolve spontaneously within 10 to 14 days can reassure new mothers that they are not “going crazy.” Women should also be counseled to seek further evaluation if these symptoms do not resolve within 2 weeks following birth, because as many as 1 in 5 women with postpartum blues will develop a major depression.

Postpartum Depression

Postpartum depression (PPD) is a major depressive disorder. PPD affects women of all cultures, ages, incomes, races and ethnicities.The peak onset for postpartum depression is in the second month after childbirth, and the risk for this disorder remains elevated up to 6 months, or even 1 year, following the birth. The point prevalence of MDD and minor depression range from 6.5% to 12.9% through the first postpartum year, and as many as 19.2% of women will have a major depressive episode at some time during the first 3 months postpartum.[146]

Risk factors for PPD are listed in **Table 8-19**. Adolescent mothers have almost twice the risk for PPD compared to adult women.[147] Adolescents are still achieving their normal developmental tasks, and often have unrealistic expectations of motherhood or the demands of parenting. In addition, adolescents are likely to be more socially isolated from their peers than adult mothers.[147] Immigrant women may also have a greater chance of experiencing PPD, depending on the circumstances of their immigration, social and language isolation, and the unavailablity of traditional childbirth rituals.[147]

Table 8-19	Risk Factors for Postpartum Depression
Strong Risk Factors	**Additional Risk Factors**
Anxiety during pregnancy	Biologic vulnerability
Depression during pregnancy	Family history of depression or postpartum depression
Stressful life events during pregnancy	Unplanned pregnancy
Low level of social support: single marital status	Young maternal age
History of depression	Lower socioeconomic status, financial insecurity
Postpartum depression after a prior pregnancy	History of interpersonal violence
	Thyroid dysfunction

When assessing a woman for PPD, it is critical to rule out postpartum bipolar disorder, which can be severe and possibly lead to postpartum psychosis. Asking women the following two questions will assess for bipolar disorder:

- Have you ever had 4 continuous days when you felt so good, high, excited, or "hyper" that other people thought you were not your normal self or that you got into trouble?
- Have you ever had 4 continuous days when you were so irritable that you found yourself shouting at people or starting fights?[145]

Even women who do not meet the criteria for MDD in the postpartum period often experience negative thoughts that are associated with depression symptomatology. In the United Kingdom, Hall and Wittkowski developed a questionnaire of postpartum negative thoughts (PNTQ) through qualitative interviews with 10 postpartum women who were suffering from postpartum depression.[148] They then distributed 354 questionnaires, along with the Edinburgh Postnatal Depression Scale, to potential participants at 42 baby-weight clinics. Of the 185 mothers who returned the questionnaires, 22 women who scored 12 or higher on the EPDS were excluded from the analysis, as the purpose of the study was to assess negative thoughts in women who did not screen positive for depression. Items on the negative thoughts questionnaire were scored as "not at all," "occasionally," "frequently," or "almost always." Three of the items on the questionnaire were experienced either frequently or almost always by more than 15% of participants: "Everything in my life should revolve around my baby" (26.6%), "I must show everyone that I am coping" (19.8%), and "As a mother, everything must be perfect" (15.9%). The feeling that "my baby could die" was experienced either frequently or almost always by 9.5% of participants, and 9.8% responded with the same frequency that "If there is something wrong with my baby, it's my fault." This study, as well as others,[149] shows that depressive symptoms exist in many more women than meet the diagnostic threshold for "postpartum depression."

Effects of Postpartum Depression on the Family

Partners of women with postpartum depression are also at risk for depression.[141] In addition, women who are depressed may be less responsive to their infants and less likely to engage in

face-to-face interactions that would contribute to infant communication skills, such as vocalizing, smiling, imitation, and game-playing, compared to mothers who are not depressed. They also can display less synchrony in their mother–baby interactions. In turn, untreated depression has been associated with impaired child development. Studies have found consistent evidence for the long-term adverse effects of untreated PPD on subsequent childhood development and behavior. Children of mothers with PPD are more likely to have poor cognitive funtioning, disruptive behaviors, and emotional maladjustment.[150] Finally, because many women with PPD were also depressed during the pregnancy, it can be difficult to separate the contribution of prenatal depression from that of postpartum depression.

Screening for Postpartum Depression

The U.S. Preventive Services Task Force and the American College of Obstetricians and Gynecologists recommend regularly screening for depression, including screening in the postpartum period. Several instruments are available to screen for PPD. Whereas the PHQ-9 is gaining favor as a screening tool for depression in the general population, it does not include items that are specific to childbirth.The EPDS, which was developed in the mid-1980s specifically for use in the postpartum period, has been well validated for use both during pregnancy and postpartum.[147] The Postpartum Depression Screening Scale (PDSS) is another instrument developed for use in the postpartum period. It is self-adminstered, and is available as a long form of 35 items or an abbreviated "short" form of 7 items.[151] The PDSS also is well validated, and may be used to evaluate response to treatment in a depressed women.

The EPDS and PDSS both appear to be more accurate in identifying postpartum depression than generic screening instruments for depression such as the CES-D or BDI. A major advantage of the EPDS over the PDSS is the ability to employ the questionnaire free of charge; in contrast, a charge is levied for the PDSS manual and forms.

A positive result on any screen should be followed up with a diagnostic evaluation by a qualified clinician so as to establish an accurate diagnosis. The screening questionnaires do not differentiate between unipolar or bipolar depression, so women identified as being depressed should be evaluated for a history or symptoms of mania or hypomania. A positive response to the questions that assess suicidality (question 9 on the PHQ-9 and question 10 on the EPDS) requires immediate attention by the clinician. The diagnosis of postpartum thyroiditis should be considered for depressed women with or without a history of autoimmune disorders.[152]

The optimal time to screen for PPD is betweeen 2 weeks to 6 months postpartum,[144] as the point prevalence of postpartum depression has bimodal peaks at 2 and 6 months postpartum. Following a 6-week postpartum visit, women are less likely to be in contact with their midwife, so many women who develop PPD may go unrecognized if screening depends on self-recognition or recognition by family or friends. For this reason, it has been recommended that screening for PPD be incorporated into the infant's well-child visits to the pediatric care provider. The American Academy of Pediatrics suggests that pediatricians should have a low threshold of suspician for parental depression, and consider, at a minimum, asking parents the two questions in the PHQ-2.

Prevention of Postpartum Depression

Primary prevention and secondary prevention addressing early detection and intervention are always preferable to dealing with the sequelae of an illness. Identifying a vulnerability to PPD may allow the midwife an opportunity to recommend interventions that may decrease the woman's risk for PPD. Interventions that may help prevent postpartum depression include interprsonal psychotherapy, regular physical activity, improving or developing a social support system, and arranging to obtain sufficient sleep postpartum.[153,154] Many of these behaviors can be fostered prior to or during pregnancy. For example, counseling regarding exercise (especially outdoors), healthy eating, and good sleep habits can be reinforced at each prenatal visit—not just to ensure physical well-being during pregnancy, but also to promote the mother's mental well-being. The CenteringPregnancy model seeks to improve social support and can be associated with less depression at one year postpartum.[155,156] Joining other postpartum support groups may also help women to develop coping skills.

Psychotherapy may help a woman learn to identify and access sources of support from family and friends, thereby avoiding the development of postpartum depression. Women who are at risk for PPD but have a desire to avoid medication may be especially receptive to this advice. If a woman has experienced depression prior to the pregnancy, but opted to discontinue the medication during the pregnancy, her plan of care may be to restart the medication immediately postpartum and continue it for at least 6 months.

Placing the newly born infant skin-to-skin with the mother has been documented to provide many maternal and infant benefits, including more stable physiologic transition, less crying, and more restful sleep.[153] These neonatal adaptations in and of themselves may allow for less maternal stress. In a quasi-experimental study, Bigelow and colleagues evaluated the effects of continuing daily skin-to-skin contact (SSC) throughout the first month. Participants completed self-report depression scales at 1 week, and 1, 2, and 3 months postpartum. Women in the skin-to-skin contact group reported significantly lower scores on both the EPDS and CES-D scales at 1-week and 1-month visits. This benefit attenuated over time, such that there were not clinically or statistically significant differences at 2 or 3 months postpartum.

Treatment for Postpartum Depression

Identifying women with postpartum depression is only half the battle. Helping women to obtain appropriate treatment is also necessary to have the desired impact on the health of mothers and their families. Unfortunately, a minority of mothers who screened positive for depression are referred for treatment, or for follow-through with their care.[149] The primary care provider is in the best position to maintain ongoing contact with the mother to reinforce ongoing treatment. Such follow-up will decrease the chance that the mother falls through the cracks or discontinues treatment before attaining a satisfactory response.

Accumulated data from many studies indicate that pyschotherapy is helpful for women with mild-to-moderate PPD, and women who are breastfeeding may opt to begin treatment with psychotherapy.[115] The evidence most strongly supports the benefits of interpersonal psychotherapy and CBT.[115]

After excluding the diagnosis of bipolar disorder, a midwife may recommend or prescribe antidepressant therapy. The same considerations that make SSRIs the first-line antidepressants for MDD unrelated to childbirth also make them the mainstay for pharmacologic treatment for PPD. Any of the SSRIs listed in **Table 8-8** can be considered for use postpartum. **Figure 8-6** summarizes the guidelines for postpartum antidepressant management.[157] As a rule, women who were treated effectively with an SSRI or SNRI during pregnancy should continue on the same agent. Although the transfer rates for the different drugs vary, in all cases the infant is exposed to less drug during breastfeeding than during pregnancy.[158] When initiating pharmacotherapy for the first time, sertraline or paroxetine may be considered as first-line agents, based on studies of levels in breast milk and reports of side effects in infants.[159] Fluoxetine (Prozac), citalopram (Celexa), and escitalopram (Lexapro) have higher rates of transfer to the breast milk, and are more often associated with infant behavioral side effects.[159]

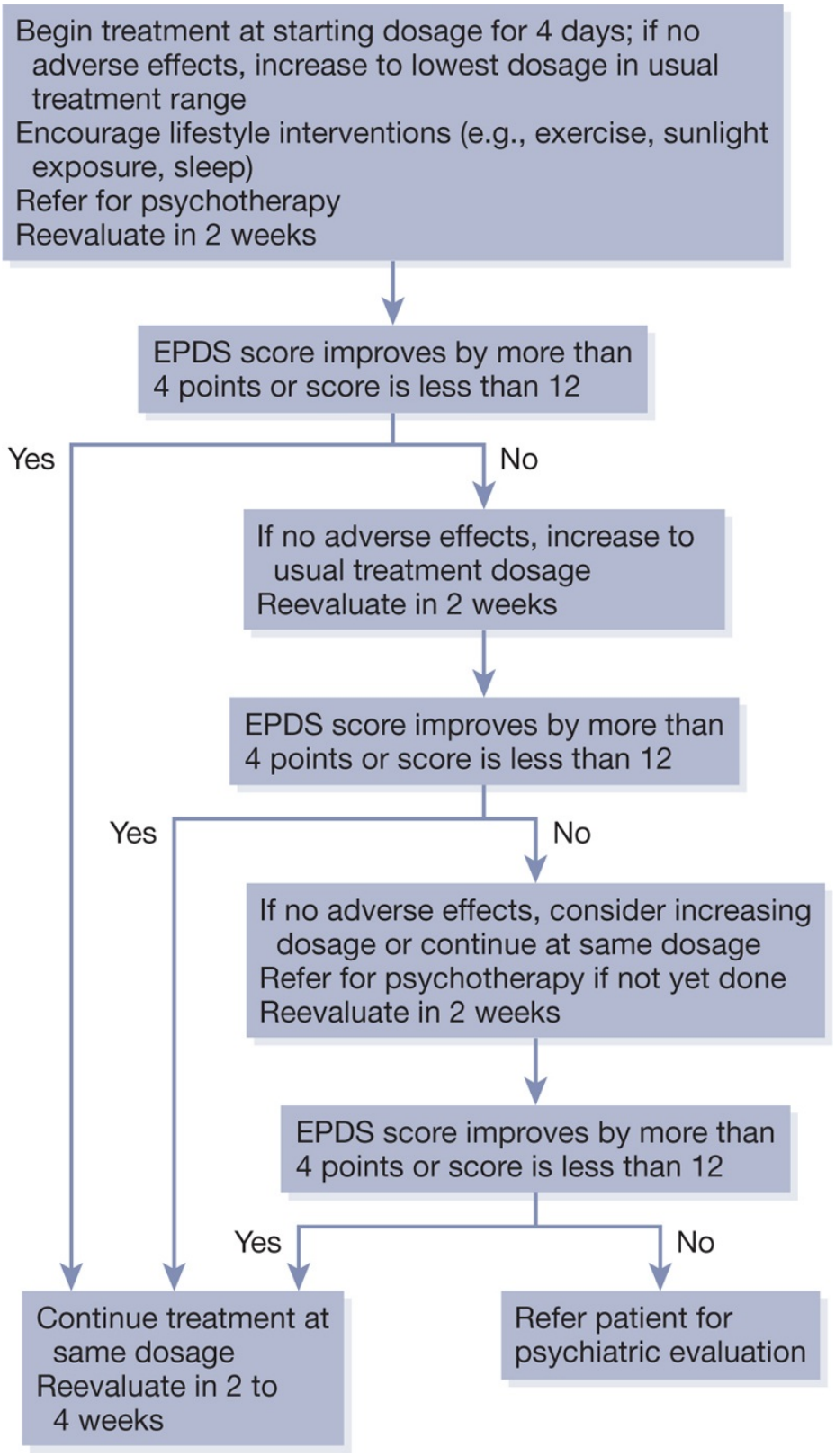


Figure 8-6 Pharmacologic treatment algorithm for postpartum major depression.

Abbreviation: EPDS, Edinburgh Postnatal Depression Scale.

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A decision to begin an antidepressant is made jointly by the mother and her care provider after a thorough discussion of risks and benefits, including the potential effects of postpartum depression on her infant and any other children. Women who perceive that a treatment decision has been imposed on them are less likely to follow through with the treatment regimen. In the postpartum setting, most women will need higher than the starting doses to achieve a therapeutic effect.

The approach when starting a medication is to begin at a low dose for 5 to 7 days, then titrate doses upward until remission is achieved. Whichever questionnaire was used to initially screen for PPD—whether the EPDS or the PHQ-9—should also be used to monitor response to treatment. Antidepressant therapy should be continued for 6 to 9 months after achieving remission. Counsel the woman that if the medication is stopped abruptly, she will probably experience unpleasant symptoms; instead, the psychotropic agent should be tapered over a period of at least 2 weeks.

Postpartum Bipolar Disorder

Although the general prevalence of bipolar disorder (BPD) is low,[3] there is a significant risk of relapse following birth. In addition, women who experience their first episode of depression in the postpartum period are more likely to have bipolar disorder, with rates varying between 15% and 50%, depending on the diagnostic criteria used.[160]

Women who were diagnosed with BPD prior to pregnancy are best cared for by a multidisciplinary team, including a psychiatrist to manage medications and decrease the chance of a relapse. Between 80% and 100% of women who discontinue their mood stabilizer medication during pregnancy experience a relapse. **Table 8-20** provides some of the essential information to be shared by a midwife with a psychiatrist in a situation of bipolar relapse or possible acute new onset of bipolar disorder postpartum.

Table 8-20 Sample Critical Elements for a Consulation/Transfer Note for a Woman with Bipolar Relapse

Past history and treatment for bipolar disease. Note the success or failure with treatments in the past, especially before pregnancy.
History of pregnancy and details of birth, including the date and the current status of the child.
Signs and symptoms indicating relapse.
Care provided before transfer: procedures and results; laboratory results received or pending; all medications (dosage, route, and time of last dose); response to treatment based on symptoms.
Pregnancy test results if appropriate, based on how many weeks postpartum and sexual activity.
Summary of the rationale for transferring the woman's care to the psychiatrists.
Request for information about the treatment plan for follow-up and/or collaboration.

While the discontinuation of medication is the most important risk factor for relapse, the dramatic hormonal and circadian rhythm alterations that occur after childbirth are also important factors. Women experiencing a bipolar episode postpartum often appear overly euphoric, talkative, and less in need for sleep. Conversely, they may present with the depressive symptoms associated with the disorder. BPD may also present as postpartum

psychosis. Any woman with a history of BPD, or exhibiting signs of either mania or hypomania, should be assessed by a psychiatric clinician.

Postpartum Psychosis

Postpartum psychosis is a rare and life-threatening emergency that requires immediate psychiatric evaluation. Postpartum psychosis occurs in 0.25 to 0.6 women per 1000 live births,[161] and may occur anytime in the first year postpartum. The peak incidence is during the first 4 weeks postpartum; in fact, this mental health disorder may be recognized in the first few days postpartum.[161]

Women with BPD are at a much higher risk for developing postpartum psychosis than other women, and many women who present with postpartum psychosis are ultimately diagnosed with BPD. Autoimmune thyroid disease may also contribute to the development of postpartum psychosis. Women with postpartum psychosis have a 4% to 5% risk of either suicide or infanticide. Treatment of postpartum psychosis often involves hospitalization, lithium therapy, and benzodiazepines to promote sleep.

Conclusion

Millions of women develop mental health disorders in the United States, and these conditions are likely to affect the well-being of both the woman and her family. For some women with mental health disorders, the midwife may be the first—and in some cases, the only—healthcare provider available. As primary care providers, midwives are, at a minimum, responsible for appropriate screening and referral for women at risk for mental health conditions. A system of universal screening can help identify those individuals with mental health concerns, and determine who can be treated safely within a primary care setting and who should be referred for more specialized treatment. With psychiatric consultation and collaboration midwives can provide first-line treatment for many women.

Psychopharmacology is an important component of treatment of mental health disorders. With careful screening, counseling, follow-up, and potential referral, this approach to treatment can be within the scope of midwifery practice. As when prescribing any medication regimen, it is essential that the midwife be familiar with psychotropic medications.

Women's susceptability to mental health disorders is influenced by their life stage and hormonal status. Pregnancy and the puerperium are times characterized by dramatic hormonal shifts, which when compounded with the stresses of becoming a parent can place a woman at increased risk for mood and anxiety disorders. Early identification and treatment can decrease long-term morbidity, but the potential effects of medications on the fetus and nursing newborn must also be considered.

Resources

Organization	Description	Webpage
Depression		
MacArthur Foundation Initiative on Depression and Primary Care	Depression Management Tool Kit	http://otgateway.com/articles/13macarthurtoolkit.pdf
Institute for Clinical Systems Improvement (ICSI)	Guidelines for Major Depression in Adults and Primary Care	https://www.icsi.org/guidelines__more/catalog_guidelines_and_more/catalog_guic
U.S. Department of Veterans Affairs (VA)	Practice Guidelines for the Management of Major Depression	http://www.healthquality.va.gov/guidelines/MH/mdd/
Psychotropic Medications in Pregnancy and Lactation		
Advancing Integrated Mental Health Solutions (AIMS) Center	University of Washington, Psychiatry and Behavioral Sciences, Commonly Prescribed Psychotropic Medications	https://aims.uw.edu/resource-library/commonly-prescribed-psychotropic-medicati
Center for Women's Mental Health at Massachusetts General Hospital (MGH)	Comprehensive resource for information about mental health and treatment in pregnancy	https://womensmentalhealth.org
Motherisk	Canadian-based clinical research and teaching program based at the University of Toronto, Ontario, which provides research, counseling, and recommendations to the public and to health providers on medication safety, chemicals, and maternal disease, and health topics	http://www.motherisk.org/women/pregnancyResources.jsp

	in pregnancy and lactation. This free resource includes helplines, updates, articles, and more.	
Organization of Teratology Specialists	MothertoBaby, an online and live chat resource for medications in pregnancy; also has an app available for iPhones and Android	https://mothertobaby.org
Suicide Screening		
Suicide Assessment Five-Step Evaluation and Triage (SAFE-T)	Pocket card for clinicians developed by the Suicide Prevention Resource Center, which is supported by the Substance Abuse and Mental Health Services Administration	http://stopasuicide.org/assets/docs/Safe_T_Card_Mental_Health_Professionals.p
Suicide Prevention Resource Center (SPRC)	Suicide prevention website and phone number	http://www.sprc.org 1-800-273-8255 (TALK)
Perinatal Mood Disorders		
Council on Patient Safety	Maternal Mental Health: Depression and Anxiety; patient safety bundle for helping clinical sites and healthcare providers screen, respond to, and refer women with perinatal mood disorders	http://safehealthcareforeverywoman.org/patient-safety-bundles/maternal-mental-h
Postpartum Support International	Multiple resources for providers and women	http://postpartum.net

References

1. Sansone RA, Sansone LA. Psychiatric disorders: a global look at facts and figures. *Psychiatry*. 2010;7(12):16-19.
2. Mayor E. Gender roles and traits in stress and health. *Front Psychol*. 2015;6:779.
3. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of *DSM-IV* disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005;62(6):593-602.
4. Seedat S, Scott KM, Angermeyer MC, et al. Cross-national associations between gender and mental disorders in the World Health Organization World Mental Health Surveys. *Arch Gen Psychiatry*. 2009;66(7):785-795.
5. Cross-Disorder Group of the Psychiatric Genomics C. Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis. *Lancet*. 2013;381(9875):1371-1379.
6. Provencal N, Binder EB. The effects of early life stress on the epigenome: from the womb to adulthood and even before. *Exper Neurol*. 2015;268:10-20.
7. Hettema JM, Kettenmann B, Ahluwalia V, et al. Pilot multimodal twin imaging study of generalized anxiety disorder. *Depress Anxiety*. 2012;29(3):202-209.
8. Kendler KS, Gardner CO. A longitudinal etiologic model for symptoms of anxiety and depression in women. *Psychol Med*. 2011;41(10):2035-2045.
9. Kendler KS, Gardner CO, Gatz M, Pedersen NL. The sources of co-morbidity between major depression and generalized anxiety disorder in a Swedish national twin sample. *Psychol Med*. 2007;37(3):453-462.
10. Tambs K, Kendler KS, Reichborn-Kjennerud T, et al. Genetic and environmental contributions to the relationship between education and anxiety disorders: a twin study. *Acta Psychiatr Scand*. 2012;125(3):203-212.
11. Etkin A, Prater KE, Schatzberg AF, Menon V, Greicius MD. Disrupted amygdalar subregion functional connectivity and evidence of a compensatory network in generalized anxiety disorder. *Arch Gen Psychiatry*. 2009;66(12):1361-1372.
12. Price JL, Drevets WC. Neurocircuitry of mood disorders. *Neuropsychopharmacol* 2010;35(1):192-216.
13. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
14. Siu AL, Bibbins-Domingo K, Grossman DC, et al. Screening for depression in adults: US Preventive Services Task Force recommendation statement. *JAMA*. 2016;315(4):380-387.
15. Gaynes BN, DeVeaugh-Geiss J, Weir S, et al. Feasibility and diagnostic validity of the M-3 Checklist: a brief, self-rated screen for depressive, bipolar, anxiety, and post-traumatic stress disorders in primary care. *Ann Fam Med*. 2010;8(2):160-169.
16. Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month *DSM-IV* disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005;62(6):617-627.
17. U.S. Department of Health and Human Services. *Depression in Primary Care: Volume 1. Detection and Diagnosis*. AHCPR Publication No. 93-0550. Rockville, MD: Agency for Health Care Policy and Research; 1993:20.
18. Wittayanukorn S, Qian J, Hansen RA. Prevalence of depressive symptoms and predictors of treatment among U.S. adults from 2005 to 2010. *Gen Hosp Psych*. 2014;36(3):330-336.
19. Merikangas KR, Ames M, Cui L, et al. The impact of comorbidity of mental and physical conditions on role disability in the US adult household population. *Arch Gen Psychiatry*. 2007;64(10):1180-1188.
20. Unützer J, Park M. Strategies to improve the management of depression in primary care. *Prim Care*. 2012;39(2):415-431.
21. Pence BW, O'Donnell JK, Gaynes BN. The depression treatment cascade in primary care: a public health perspective. *Current Psychiatry Rep*. 2012;14(4):328-335.
22. Hackley B, Sharma C, Kedzior A, Sreenivasan S. Managing mental health conditions in primary care settings. *J Midwifery Womens Health*. 2010;55(1):9-19.
23. Moussavi S, Chatterji S, Verdes E, Tandon A, Patel V, Ustun B. Depression, chronic diseases, and decrements in health: results from the World Health Surveys. *Lancet*. 2007;370(9590):851-858.
24. Arroll B, Goodyear-Smith F, Crengle S, et al. Validation of PHQ-2 and PHQ-9 to screen for major depression in the primary care population. *Ann Fam Med*. 2010;8(4):348-353.
25. Arroll B, Khin N, Kerse N. Screening for depression in primary care with two verbally asked questions: cross sectional study. *BMJ*. 2003;327(7424):1144-1146.
26. Kroenke K, Spitzer RL, Williams JB. The Patient Health Questionnaire-2: validity of a two-item depression screener. *Med Care*. 2003;41:1284-1292.
27. Lombardo P, Vaucher P, Haftgoli N, et al. The "help" question doesn't help when screening for major depression: external validation of the three-question screening test for primary care patients managed for physical complaints. *BMC Med*. 2011;9:114.
28. Pfizer, Inc. The Patient Health Questionnaire-9. Available at: https://integrationacademy.ahrq.gov/sites/default/files/PHQ-9_0.pdf. Accessed May 30, 2017.

29. Maurer DM. Screening for depression. *Am Fam Physician*. 2012;85(2):139-144.
30. Bergink V, Kooistra L, Lambregtse-van den Berg MP, et al. Validation of the Edinburgh Depression Scale during pregnancy. *J Psychosom Res*. 2011;70(4):385-389.
31. Claassen CA, Trivedi MH, Rush AJ, et al. Clinical differences among depressed patients with and without a history of suicide attempts: findings from the STAR*D trial. *J Affect Disord*. 2007;97(1-3):77-84.
32. Trangle M, Haight R, Hardwig J, et al. *Adult Depression in Primary Care*. Rockville, MD: Institute for Clinical Systems Improvement; 2016. Available at: https://www.guideline.gov/summaries/summary/50406/adult-depression-in-primary-care. Accessed May 30, 2017.
33. Gureje O. Dysthymia in a cross-cultural perspective. *Curr Opin Psychiatry*. 2011;24(1):67-71.
34. Dunner DL. Dysthymia and double depression. *Int Rev Psychiatyr*. 2005;17(1):3-8.
35. Spitzer RL, Williams JB, Kroenke K, et al. Utility of a new procedure for diagnosing mental disorders in primary care: the PRIME-MD 1000 study. *JAMA*. 1994;272(22):1749-1756.
36. Rhebergen D, Beekman ATF, de Graaf R, et al. Trajectories of recovery of social and physical functioning in major depression, dysthymic disorder and double depression: a 3-year follow-up. *J Affect Disord*. 2010;124(1-2):148-156.
37. Deligiannidis KM, Freeman MP. Complementary and alternative medicine therapies for perinatal depression. *Best Pract Res Clin Obstet Gynaecol*. 2014;28(1):85-95.
38. DeRubeis RJ, Siegle GJ, Hollon SD. Cognitive therapy versus medication for depression: treatment outcomes and neural mechanisms. *Nature Rev Neurosci*. 2008;9(10):788-796.
39. Cuijpers P, Weitz E, Twisk J, et al. Gender as predictor and moderator of outcome in cognitive behavior therapy and pharmacotherapy for adult depression: an "individual patient data" meta-analysis. *Depress Anxiety*. 2014;31(11):941-951.
40. Cuijpers P, Sijbrandij M, Koole SL, Andersson G, Beekman AT, Reynolds CF 3rd. Adding psychotherapy to antidepressant medication in depression and anxiety disorders: a meta-analysis. *World Psychiatry*. 2014;13(1):56-67.
41. Levkovitz Y, Tedeschini E, Papakostas GI. Efficacy of antidepressants for dysthymia: a meta-analysis of placebo-controlled randomized trials. *J Clin Psychiatry*. 2011;72(4):509-514.
42. Cuijpers P, van Straten A, Schuurmans J, van Oppen P, Hollon SD, Andersson G. Psychotherapy for chronic major depression and dysthymia: a meta-analysis. *Clin Psychol Rev*. 2010;30(1):51-62.
43. Hollon SD, Ponniah K. A review of empirically supported psychological therapies for mood disorders in adults. *Depress Anxiety*. 2010;27(10):891-932.
44. Markowitz JC, Kocsis JH, Bleiberg KL, Christos PJ, Sacks M. A comparative trial of psychotherapy and pharmacotherapy for "pure" dysthymic patients. *J Affect Disord*. 2005;89(1-3):167-175.
45. Grypma L, Haverkamp R, Little S, Unützer J. Taking an evidence-based model of depression care from research to practice: making lemonade out of depression. *Gen Hosp Psychiatry. 2006;*28(2):101-107.
46. Williams JW Jr, Gerrity M, Holsinger T, Dobscha S, Gaynes B, Dietrich A. Systematic review of multifaceted interventions to improve depression care. *Gen Hosp Psych*. 2007;29(2):91-116.
47. Schuch FB, Vasconcelos-Moreno MP, Borowsky C, Fleck MP. Exercise and severe depression: preliminary results of an add-on study. *J Affect Disord*. 2011;133(3):615-618.
48. Parikh SV, Quilty LC, Ravitz P, et al. Canadian Network for Mood and Anxiety Treatments (CANMAT) 2016 clinical guidelines for the management of adults with major depressive disorder. Section 2. Psychological treatments. *Can J Psychiat*. 2016;61(9):524-539.
49. Zhou T, Li X, Pei Y, Gao J, Kong J. Internet-based cognitive behavioural therapy for subthreshold depression: a systematic review and meta-analysis. *BMC Psychiatry*. 2016;16(1):356.
50. Culpepper L. The diagnosis and treatment of bipolar disorder: decision-making in primary care. *Prim Care Companion CNS Disord*. 2014;16(3). doi:10.4088/PCC.13r01609.
51. Early NK. Mental health. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:727-764.
52. Culpepper L. Misdiagnosis of bipolar depression in primary care practices. *J Clin Psychiatry*. 2014;75(3):e05. doi:10.4088/JCP.13019tx1c.
53. Kupfer D, Frank E, Phillips M. Major depressive disorder: new clinical, neurobiological, and treatment perspectives. *Psychopharmacology: Evidence and Treatments*. 2014;12(2):217-228.
54. Qaseem A, Snow V, Denberg TD, Forciea MA, Owens DK, Clinical Efficacy Assessment Subcommittee of American College of Physicians. Using second-generation antidepressants to treat depressive disorders: a clinical practice guideline from the American College of Physicians. *Ann Intern Med*. 2008;149(10):725-733.
55. MacArthur Initiative on Depression and Primary Care. Depression management tool kit. 2009. Available at: http://www.integration.samhsa.gov/clinical-practice/macarthur_depression_toolkit.pdf. Accessed July 22, 2017.
56. Department of Veterans Affairs, Department of Defense. The management of major depressive disorder: clinical guideline summary. 2016. Available at: https://www.healthquality.va.gov/guidelines/MH/mdd/. Accessed July 22, 2017.
57. von Wolff A, Holzel LP, Westphal A, Harter M, Kriston L. Selective serotonin reuptake inhibitors and tricyclic antidepressants in the acute treatment of chronic depression and dysthymia: a systematic review and meta-analysis. *J*

Affect Disord. 2013;144(1-2):7-15.
58. Uguz F, Sahingoz M, Gungor B, Aksoy F, Askin R. Weight gain and associated factors in patients using newer antidepressant drugs. *Gen Hosp Psychiatry*. 2015;37(1):46-48.
59. Lai MW, Klein-Schwartz W, Rodgers GC, et al. 2005 annual report of the American Association of Poison Control Centers' national poisoning and exposure database. *Clin Toxicol*. 2006;44(6-7):803-932.
60. Heitmiller DR. Serotonin syndrome: a concise review of a toxic state. *RI Med J*. 2014;97(6):33-35.
61. Wang RZ, Vashistha V, Kaur S, Houchens NW. Serotonin syndrome: preventing, recognizing, and treating it. *Cleve Clin J Med*. 2016;83(11):810-817.
62. Ables AZ, Nagubilli R. Prevention, recognition, and management of serotonin syndrome. *Am Fam Physician*. 2010;81(9):1139-1142.
63. Friedman RA. Antidepressants' black-box warning—10 years later. *N Engl J Med*. 2014;371:1666-1668.
64. Tamburrino MB, Nagel RW, Lynch DJ. Managing antidepressants in primary care: physicians' treatment modifications. *Psychol Rep*. 2011;108(3):799-804.
65. Sansone RA, Sansone LA. Dysthymic disorder: forlorn and overlooked? *Psychiatry*. 2009;6(5):46-51.
66. Kroenke K, Spitzer RL, Williams JB, Monahan PO, Löwe B. Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. *Ann Intern Med*. 2007;146(5):317-325.
67. Zolensky MJ, Garey L, Bakshaie J. Disparities in anxiety and its disorders. *J Anxiety Disord*. 2017;48:1-5.
68. Baldwin DS, Anderson IM, Nutt DJ, et al. Evidence-based pharmacological treatment of anxiety disorders, post-traumatic stress disorder and obsessive–compulsive disorder: a revision of the 2005 guidelines from the British Association for Psychopharmacology. *J Psychopharm*. 2014;28(5):403-439.
69. Bandelow B, Michaelis S. Epidemiology of anxiety disorders in the 21st century. *Dialog Clin Neurosci*. 2015;17(3):327-335.
70. Kesslert RCX, Petukhova M, Sampson NA, Zaslavsky AM, Wittchen HU. Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *Int J Methods Psychiatr Res*. 2012;21(3):169-184.
71. Simms LJ, Prisciandaro JJ, Krueger RF, Goldberg DP. The structure of depression, anxiety and somatic symptoms in primary care. *Psychol Med*. 2012;42(1):15-28.
72. Spitzer RL, Kroenke K, Williams JB, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch Intern Med*. 2006;166(10):1092-1097.
73. Robinson CM, Klenck SC, Norton PJ. Psychometric properties of the Generalized Anxiety Disorder Questionnaire for *DSM-IV* among four racial groups. *Cognitive Behav Ther*. 2010;39(4):251-261.
74. Karsnitz DB, Ward S. Spectrum of anxiety disorders: diagnosis and pharmacologic treatment. *J Midwifery Womens Health*. 2011;56(3):266-281.
75. Lukaschek K, Kruse J, Emeny RT, Lacruz ME, von Eisenhart Rothe A, Ladwig KH. Lifetime traumatic experiences and their impact on PTSD: a general population study. *Soc Psychiatry Psychiatr Epidemiol*. 2013;48(4):525-532.
76. Wiechelt SA, Miller BA, Smyth NJ, Maguin E. Associations between post-traumatic stress disorder symptoms and alcohol and other drug problems: implications for social work practice. *Practice*. 2011;23(4):183-199.
77. Chen L, Zhang G, Hu M, Liang X. Eye movement desensitization and reprocessing versus cognitive-behavioral therapy for adult posttraumatic stress disorder: systematic review and meta-analysis. *J Nerv Mental Dis*. 2015;203(6):443-451.
78. Baldwin DS, Ajel KI, Garner M. Pharmacological treatment of generalized anxiety disorder. *Curr Top Behav Neurosci*. 2010;2:453-467.
79. Walkup JT, Albano AM, Piacentini J, et al. Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *N Engl J Med*. 2008;359(26):2753-2766.
80. Hunot V, Churchill R, Teixeira V, Silva de Lima M. Psychological therapies for generalised anxiety disorder. *Cochrane Database Syst Rev*. 2007;1:CD001848. doi:10.1002/14651858.CD001848.pub4.
81. Olthuis JV, Watt MC, Bailey K, Hayden JA, Stewart SH. Therapist-supported Internet cognitive behavioural therapy for anxiety disorders in adults. Cochrane Database *Syst Rev*. 2016;3:CD011565. doi:10.1002/14651858.CD011565.pub2.
82. Roy-Byrne P, Craske MG, Sullivan G, et al. Delivery of evidence-based treatment for multiple anxiety disorders in primary care: a randomized controlled trial. *JAMA*. 2010;303(19):1921-1928.
83. Baldwin DS, Waldman S, Allgulander C. Evidence-based pharmacological treatment of generalized anxiety disorder. *Int J Neurophsychopharm*. 2011;14(5):697-710.
84. Ravindran LN, Stein MB. The pharmacologic treatment of anxiety disorders: a review of progress. *J Clin Psychiatry*. 2010;71(7):839-854.
85. Huh J, Goebert D, Takeshita J, Lu BY, Kang M. Treatment of generalized anxiety disorder: a comprehensive review of the literature for psychopharmacologic alternatives to newer antidepressants and benzodiazepines. *Prim Care Companion CNS Disord*. 2011;13(2). doi:10.4088/PCC.08r00709blu.
86. Baldwin DS, Anderson IM, Nutt DJ, et al. Evidence-based guidelines for the pharmacological treatment of anxiety disorders: recommendations from the British Association for Psychopharmacology. *J Psychopharm*. 2005;19(6):567-596.

87. Yonkers KA, Simoni MK. Premenstrual disorders. *Am J Obstet Gynecol*. 2017. [Epub ahead of print]. doi:10.1016/j.ajog.2017.05.045.
88. O'Brien PMS, Bäckström T, Brown C, et al. Towards a consensus on diagnostic criteria, measurement and trial design of the premenstrual disorders: the ISPMD Montreal consensus. *Arch Womens Mental Health*. 2011;14(1):13-21.
89. Lanze di Scalea T, Pearlstein T. Premenstrual dysphoric disorder. *Psychiatr Clin North Am.* 2017;40:201-216.
90. Ismaili E, Walsh S, O'Brien PMS, et al. Fourth consensus of the International Society for Premenstrual Disorders (ISPMD): auditable standard for diagnosis and management of premenstrual disorder. *Arch Womens Ment Health*. 2016;19:953-958.
91. Hofmesiter S, Bodden S. Premenstrual disorder and premenstrual dyphoric disorder. *Am Fam Physician*. 2016;94(3):236-240.
92. Nevatte T, O'Brien PMS, Bäckström T, et al. ISPMD consensus on the management of premenstrual disorders. *Arch Womens Ment Health*. 2013;16(4):279-291.
93. Rubinow DR, Schmidt PJ. Gonadal steroid regulation of mood: the lessons of premenstrual syndrome. *Front Neuroendocrinol*. 2006;27(2):210-216.
94. Eisenlohr-Moul TA, Girdler SS, Schmalenberger KM, et al. Toward the reliable diagnosis of *DSM-5* premenstrual dysphoric disorder: the Carolina Premenstrual Assessment Scoring System (C-PASS). *Am J Psychiatry*. 2017;174:51-59.
95. Endicott J, Nee J, Harrison W. Daily Record of Severity of Problems (DRSP): reliability and validity. *Arch Womens Ment Health*. 2006;9:41-49.
96. Epperson CN, Hantsoo LV. Making strides to simplify diagnosis of premenstrual dysphoric disorder. *Am J Psychiatry*. 2017;174(1):6-7.
97. Shah NR, Jones JB, Aperi J, Shemtov R, Karne A, Borenstein J. Selective serotonin reuptake inhibitors for premenstrual syndrome and premenstrual dysphoric disorder: a meta-analysis. *Obstet Gynecol*. 2008;111(5):1175-1182.
98. Pearlstein TB, Bachmann GA, Zacur HA, Yonkers KA. Treatment of premenstrual dysphoric disorder with a new drospirenone-containing oral contraceptive formulation. *Contraception*. 2005;72(6):414-421.
99. Coffee AL, Kuehl TJ, Willis S, Sulak PJ. Oral contraceptives and premenstrual symptoms: comparison of a 21/7 and extended regimen. *Am J Obstet Gynecol*. 2006;195(5):1311-1319.
100. Di Carlo C, Palomba S, Tommaselli GA, Guida M, Di Spiezio Sardo A, Nappi C. Use of leuprolide acetate plus tibolone in the treatment of severe premenstrual syndrome. *Fertil Steril*. 2001;75(2):380-384.
101. Joffe H, Petrillo LF, Viguera AC, et al. Treatment of premenstrual worsening of depression with adjunctive oral contraceptive pills: a preliminary report. *J Clin Psychol*. 2007;68(12):1954-1962.
102. Pinkerton JV, Guico-Pabia CJ, Taylor HS. Menstrual cycle-related exacerbation of disease. *Am J Obstet Gynecol*. 2010;202(3):221-231.
103. Bhat A, Reed SD, Unutzer J. The obstetrician-gynecologist's role in detecting, preventing, and treating depression. *Obstet Gynecol*. 2017;129(1):157-163.
104. Davalos DB, Yadon CA, Tregellas HC. Untreated prenatal maternal depression and the potential risks to offspring: a review. *Arch Womens Ment Health*. 2012;15(1):1-14.
105. Drury SS, Scaramella L, Zeanah CH. The neurobiological impact of postpartum maternal depression: prevention and intervention approaches. *Child Adolesc Psych Clin North Am.* 2016;25(2):179-200.
106. Cohen LS, Altshuler LL, Harlow BL, et al. Relapse of major depression during pregnancy in women who maintain or discontinue antidepressant treatment. *JAMA*. 2006;295(5):499-507.
107. Woody CA, Ferrari AJ, Sikind DJ, Whiteford HA, Harris MG. A systematic review and meta-regression of the prevalence and incidence of perinatal depression. *J Affect Disord*. 2017;219:86-92.
108. Fellmeth G, Fazel M, Plugge E. Migration and perinatal mental health in women from low- and middle-income countries: a systematic review and meta-analysis. *BJOG*. 2017;124(5):742-752.
109. Osborne LM, Payne J. Antidepressants, pregnancy, and stigma: how we are failing mothers and babies. *J Nerv Mental Dis*. 2015;203(3):164-166.
110. Stohl H, Kohm AD, Dossett E. A rock and a hard place: the selective serotonin reuptake inhibitor dilemmas in addressing perinatal mood and anxiety disorders. *J Neonatal Perinatal Med*. 2016;9(1):1-5.
111. Suri R, Lin AS, Cohen LS, Altshuler LL. Acute and long-term behavioral outcome of infants and children exposed in utero to either maternal depression or antidepressants: a review of the literature. *J Clin Psychiatry*. 2014;75(10):e1142-e1152.
112. Csaszar E, Melichercikova K, Dubovicky M. Neuroendocrine and behavioral consequences of untreated and treated depression in pregnancy and lactation. *Neurol Endocrinol Lett*. 2014;35(suppl 2):169.
113. American College of Obstetricians and Gynecologists. Committee Opinion No. 630: screening for perinatal depression. *Obstet Gynecol*. 2015;125:1268-1271.
114. O'Connor E, Rossom RC, Henninger M, Groom HC, Burda BU. Primary care screening for and treatment of depression in pregnant and postpartum women: evidence report and systematic review for the US Preventive Services Task Force. *JAMA*. 2016;315(4):388-406.

115. Kendig S, Keats JP, Hoffman MC, et al. Consensus bundle on maternal mental health. *J Midwifery Womens Health*. 2017;62(2):232-239.
116. Sylvén SM, Elenis E, Michelakos T, et al. Thyroid function tests at delivery and risk for postpartum depressive symptoms. *Psychoneuroendocrinology*. 2013 Jul;38(7):1007-1013.
117. Cox JL, Holden JM, Sagovsky R. Detection of postnatal depression: development of the 10-item Edinburgh Postnatal Depression Scale. *Br J Psychiatry*. 1987;150:782-786.
118. Gaynes BN, Gavin N, Meltzer-Brody S, Lohr KN, Swinson T, Gartlehner G, Brody S, Miller WC. *Perinatal Depression: Prevalence, Screening Accuracy, and Screening Outcomes. Evidence Report/Technology Assessment No. 119*. AHRQ Publication No. 05- E006-2. Rockville, MD: Agency for Healthcare Research and Quality; 2005.
119. Latendresse G, Deneris A. Selective serotonin reuptake inhibitors as first-line antidepressent therapy for perinatal depression. *J Midwifery Womens Health*. 2017; 62(3):317-328.
120. Yonkers KA, Gotman N, Smith MV, et al. Does antidepressant use attenuate the risk of a major depressive episode in pregnancy? *Epidemiology*. 2011;22(6):848-854.
121. Bergink V, Bouvy PF, Vervoort JS, Koorengevel KM, Steegers EA, Kushner SA. Prevention of postpartum psychosis and mania in women at high risk. *Am J Psychiatry*. 2012;169(6):609-615.
122. Buist A, Gotman N, Yonkers KA. Generalized anxiety disorder: course and risk factors in pregnancy. *J Affect Disord*. 2011;131(1-3):277-283.
123. Chaudron LH, Nirodi N. The obsessive–compulsive spectrum in the perinatal period: a prospective pilot study. *Arch Womens Ment Health*. 2010;13(5):403-410.
124. Chisolm MS, Payne JL. Management of psychotropic drugs during pregnancy. *BMJ*. 2016;532:h5918. doi:10.1136/bmj.h5918.
125. Tak CR, Job KM, Schoen-Gentry K, et al. The impact of exposure to antidepressant medications during pregnancy on neonatal outcomes: a review of retrospective database cohort studies. *Eur J Clin Pharmacol*. 73(9):1055-1069.
126. Smith KF, Huber LR, Issel LM, Warren-Findlow J. The association between maternal depression during pregnancy and adverse birth outcomes: a retrospective cohort study of PRAMS participants. *J Commun Health*. 2015;40(5):984-992.
127. Yonkers KA, Norwitz ER, Smith MV, et al. Depression and serotonin reuptake inhibitor treatment as risk factors for preterm birth. *Epidemiology*. 2012;23(5):677-685.
128. Cantarutti A, Merlino L, Monzani E, Giaquinto C, Corrao G. Is the risk of preterm birth and low birth weight affected by the use of antidepressant agents during pregnancy? A population-based investigation. *PloS One*. 2016;11(12):e0168115. doi:10.1371/journal.pone.0168115.
129. Meltzer-Brody S, Jones I. Optimizing the treatment of mood disorders in the perinatal period. *Dialog Clin Neurosci*. 2015;17(2):207-218.
130. Chambers CD, Hernandez-Diaz S, Van Marter LJ, et al. Selective serotonin-reuptake inhibitors and risk of persistent pulmonary hypertension of the newborn. *N Engl J Med*. 2006;354(6):579-587.
131. Becker M, Weinberger T, Chandy A, Schmukler S. Depression during pregnancy and postpartum. *Curr Psychiatry Rep*. 2016;18(3):32.
132. Huybrechts KF, Bateman BT, Palmsten K, et al. Antidepressant use late in pregnancy and risk of persistent pulmonary hypertension of the newborn. *JAMA*. 2015;313(21):2142-2151.
133. Hviid A, Melbye M, Pasternak B. Use of selective serotonin reuptake inhibitors during pregnancy and risk of autism. *N Engl J Med*. 2013;369(25):2406-2415.
134. Boukhris T, Sheehy O, Mottron L, Berard A. Antidepressant use during pregnancy and the risk of autism spectrum disorder in children. *JAMA Pediatr*. 2016;170(2):117-124.
135. Sit DK, Perel JM, Helsel JC, Wisner KL. Changes in antidepressant metabolism and dosing across pregnancy and early postpartum. *J Clin Psychiatry*. 2008;69(4):652-658.
136. Roder C, Schaefer M, Leucht S. Meta-analysis of effectiveness and tolerability of treatment of mild to moderate depression with St. John's wort [in German]. *Fortschr Neurol Psychiatr*. 2004;72(6):330-343.
137. Sperlich M, Seng JS, Li Y, Taylor J, Bradbury-Jones C. Integrating trauma-informed care into midwifery practice: conceptual and practical issues. *J Midwifery Womens Health*. 2017;62(6):661-672.
138. Seng TS, Sperlich M, Low LK, Ronis DL, Muzik M, Liberzon I. Childhood abuse history, posttraumatic stress disorder, postpartum mental health, and bonding: a prospective cohort study. *J Midwifery Womens Health*. 2013;58(1):57-68.
139. Olatunji BO, Fan Q. Anxiety sensitivity and post-traumatic stress reactions: evidence for intrusions and physiological arousal as mediating and moderating mechanisms. *J Anxiety Disord*. 2015;34:76-85.
140. Beck CT. Birth trauma: in the eye of the beholder. *Nurs Res*. 2004;53(1):28-35.
141. Breese McCoy SJ. Postpartum depression: an essential overview for the practitioner. *Southern Med J*. 2011;104(2):128-132.
142. Bernstein IH, Rush AJ, Yonkers K, et al. Symptom features of postpartum depression: are they distinct? *Depress Anxiety*. 2008;25(1):20-26.
143. NIHCM Foundation. *Identifying and Treating Maternal Depression: Strategies and Considerations for Health*

Plans. Issue Brief. Rockville, MD: U.S. Public Health Service, Health Resources and Services Administration, Maternal and Child Health Bureau; 2010. Available at: https://www.nihcm.org/pdf/FINAL_MaternalDepression6-7.pdf. Accessed June 21, 2017.
144. Beck CT. Postpartum depression: it isn't just the blues. *Am J Nurs*. 2006;106(5):40-50; quiz 50-51.
145. Stewart DE, Vigod S. Postpartum depression. *N Engl J Med*. 2016;375(22):2177-2186.
146. Gavin NI, Gaynes BN, Lohr KN, Meltzer-Brody S, Gartlehner G, Swinson T. Perinatal depression: a systematic review of prevalence and incidence. *Obstet Gynecol*. 2005;106(5 pt 1):1071-1083.
147. Clare CA, Yeh J. Postpartum depression in special populations: a review. *Obstet Gynecol Surv*. 2012;67(5):313-323.
148. Hall PL, Wittkowski A. An exploration of negative thoughts as a normal phenomenon after childbirth. *J Midwifery Womens Health*. 2006;51(5):321-330.
149. Beck CT, Gable RK, Sakala C, Declercq ER. Postpartum depressive symptomatology: results from a two-stage US national survey. *J Midwifery Womens Health*. 2011;56(5):427-435.
150. Cuijpers P, Brannmark JG, van Straten A. Psychological treatment of postpartum depression: a meta-analysis. *J Clin Psychol*. 2008;64(1):103-118.
151. Beck CT, Gable RK. Postpartum Depression Screening Scale. *Nurs Res. 2003;52*(5): 296-306.
152. Stagnaro-Green A. Approach to the patient with postpartum thyroiditis. *J Clin Endocrin Metabol*. 2012;97(2):334-342.
153. Bigelow A, Power M, MacLellan-Peters J, Alex M, McDonald C. Effect of mother/infant skin-to-skin contact on postpartum depressive symptoms and maternal physiological stress. *JOGNN*. 2012;41(3):369-382.
154. Miller LJ, LaRusso EM. Preventing postpartum depression. *Psychiat Clin North Am*. 2011;34(1):53-65.
155. Ickovics JR, Reed E, Magriples U, Westdahl C, Schindler Rising S, Kershaw TS. Effects of group prenatal care on psychosocial risk in pregnancy: results from a randomised controlled trial. *Psychol Health*. 2011;26(2):235-250.
156. McNeil DA, Vekved M, Dolan SM, Siever J, Horn S, Tough SC. Getting more than they realized they needed: a qualitative study of women's experience of group prenatal care. *BMC Pregnancy Childbirth*. 2012;12:17.
157. Hirst KP, Moutier CY. Postpartum major depression. *Am Fam Physician*. 2010;82(8):926-933.
158. Sriraman NK, Melvin K, Meltzer-Brody S. ABM Clinical Protocol #18: use of antidepressants in breastfeeding mothers. *Breastfeeding Med*. 2015;10:290-299.
159. Orsolini L, Bellantuono C. Serotonin reuptake inhibitors and breastfeeding: a systematic review. *Human Psychopharm*. 2015;30(1):4-20.
160. Sharma V, Doobay M, Baczynski C. Bipolar postpartum depression: an update and recommendations. *J Affect Disord*. 2017;219:105-111.
161. Bergink V, Rasgon N, Wisner KL. Postpartum psychosis: madness, mania, and melancholia in motherhood. *Am J Psychiatry*. 2016;173(12):1179-1188.

CHAPTER

9

Pharmacotherapeutics

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Introduction

Pharmacology is derived from the Greek word *pharmakon*, which means “medicine or poison.” Most sources define pharmacology more precisely as the study of how drugs interact with a living organism to produce a change in physiologic function. Any agent, substance, or medication that is used for medicinal purposes is considered a pharmaceutical. Although midwives may use a wide repertoire of nonpharmaceutical techniques, pharmacologic treatments are indicated for some conditions and situations. Certified nurse-midwives and certified midwives have varying degrees of prescriptive authority in most states within the United States.[1] One of the greatest challenges in clinical practice is maintaining current knowledge about drug indications, doses, side effects, and contraindications, and prescriptive authority.

In the United States, the U.S. Food and Drug Administration (FDA) is the regulatory agency charged with ensuring that drugs, biologic products, medical devices, the national food supply, cosmetics, and products that emit radiation are safe, effective, and secure for consumer use. Other governmental bodies that have some involvement with drugs include the Centers for Disease Control and Prevention (CDC), which publishes recommendations for treatments of disorders and diseases; the Drug Enforcement Administration (DEA), which regulates controlled substances; the Federal Communications Commission (FCC), which regulates advertising of over-the-counter drugs; and state governments, which control prescriptive authority for providers, including midwives.

Most adults in the United States take at least one medication, and many take multiple pharmaceuticals daily. The extensive use of drugs is related to several factors: the increase in their availability; the public belief that drugs are safe, especially over-the-counter products; and the plethora of healthcare providers, pharmacies, and Internet sites that supply drugs. These multiple options mean that an individual may receive drug prescriptions and recommendations from a variety of providers, many of whom may be unaware of the other prescribers’ actions. Direct-to-customer advertising exposes many more individuals to prescription drugs and proposed therapeutic indications of which they might have been previously unaware.[2,3] In addition to widespread public acceptance of drugs to treat illnesses, there is a growing appreciation of how agents may be used for prophylaxis against various diseases or for general health maintenance.

Even individuals who avoid taking specific drugs or agents may be exposed to pharmaceuticals through the food chain or in their daily environment.[4] The results of such exposure, including direct toxic reactions and reproductive toxicology, are beyond the scope of this chapter, although this area is likely to grow in importance over time.

This chapter presents a brief introduction and wide overview of the pharmacologic principles that form the foundation of pharmacotherapeutics in midwifery practice. These principles can be applied to specific pharmacotherapeutic therapies, which are addressed in other chapters. The examples included in this chapter are presented as illustrations to facilitate understanding of clinically relevant pharmacologic principles, effects, and properties; they are not a comprehensive summary of clinically relevant pharmacology for a particular topic. A list of resources is provided at the end of this chapter to assist the reader in obtaining a thorough

knowledge about when a specific drug is needed.

The terms “drugs,” “agents,” “medications,” and “pharmaceuticals” are used interchangeably throughout the chapter. The word “drug” does not connote an illicit substance or a licit substance being abused; rather, drugs with abuse potential are specifically designated as such. Drugs are listed by their generic names, followed by the most common brand name in parentheses.

The Lexicon of Pharmacology

As knowledge in pharmacology has expanded, so has the accompanying lexicon. A prerequisite to understanding drug actions and their effects is the midwife's ability to define the various terms used in pharmacology. A glossary presented in **Table 9-1**.

Table 9-1 Brief Glossary of Pharmacology

Term	Definition
Adverse drug reaction	Response to a drug that is noxious and unintended, and that occurs at doses normally used for prophylaxis, diagnosis, or therapy of disease or for the modification of physiologic function.
Agonist	A drug that binds to a receptor and activates it, producing a pharmacologic response.
Antagonist	A drug that attenuates the effects of an agonist. Antagonism can be competitive and reversible (i.e., the drug binds reversibly to a region of the receptor in common with the agonist) or competitive and irreversible (i.e., the antagonist binds covalently to the agonist binding site, and no amount of agonist can overcome the inhibition).
Behind-the-counter (BTC)	Descriptor for drugs that are sold without a prescription, but that are subject to restrictions such as proof of identity because of potential risks. For example, pseudoephedrine is BTC because it may be used as an ingredient in the production of methamphetamine.
Bioavailability	Percentage of an administered drug that is available to target tissues.
Bioequivalent	Pharmacologically equivalent.
Biovariability	Different expressions of a drug among individuals even when the agent is administered at a standard dose.
Black box warning	A notice from the U.S. Food and Drug Administration when unusual harm associated with an agent has been identified. A black box warning is added to package inserts after postmarketing studies identify unexpected risks linked to a drug.
Brand name	A trademarked name assigned to a drug by its manufacturer. Some brand names are similar to the generic name (e.g., pseudoephedrine/Sudafed); others suggest their indications for use (e.g., Tamiflu).
Chemical name	Although rarely used by prescribers and consumers, a name of a drug that describes its chemical composition.
Compounding	Mixing or combining ingredients to produce a pharmaceutical agent. Pharmacists may perform compounding to change the form from solid pill to liquid, or to create a unique dose and combination of products for a specific individual.
Controlled substance	One of the pharmaceuticals listed in schedules found in U.S. Law 21 U.S.C. §802(32)(A). These agents include opiates as well as non-opiates but generally have a high risk of addiction, and many lack a valid medicinal use. Examples include heroin.
Cosmeceutical	A cosmetic product that has medicinal benefits.
Counterfeit drugs	Pharmaceuticals that are produced and sold in a deceptive manner so that they appear to be authentic drugs.
Designer drug	A drug directed toward a specific biologic target that binds to and inhibits key molecules involved in a disease or pathologic event. Designer drugs often are older drugs whose composition is manipulated to minimize adverse effects. This term does not imply illicit drugs.
Direct-to-consumer (DTC)	Advertisements for selected drugs placed in popular media and directed to the general public, as opposed to being directed to providers.
Dispense (furnish)	The process of giving a drug to a consumer.

Drug	A chemical substance that brings about changes in a biologic system through its chemical action(s). Also called a *medication* or *pharmaceutical.*
Ecopharmacology	Derivation of drugs from plants, especially those found in the rainforest, as well as exploration of pharmacologic effects of pollutants in water that exert pharmaceutical-like effects, most often estrogenic in nature. The latter substances also have been called ecoestrogens or xenoestrogens.
Effectiveness	The ability of a drug or treatment to produce a positive effect in real-life circumstances or with typical use.
Efficacy	Ability of a drug or treatment to produce a positive effect in ideal circumstances such as a research trial.
Formulary	List of approved or available drugs. It is often used by insurers to identify agents that will be reimbursed or paid for by the insurer.
Generic name	A formulation that contains the same active ingredients found in the original brand formulation and is bioequivalent to that formulation.
Half-life	The period of time required for the concentration or amount of drug in the body to be reduced to exactly one-half of the initial concentration or amount.
High-alert drug	A pharmaceutical that is associated with significant harm if used in error.
Hypersensitivity reaction	A state of altered reactivity wherein the body reacts to a foreign substance with an exaggerated immune response; classified as Type I, II, III, or IV depending on the specific pathologic response.
Immunotherapy	Treatment of disease by inducing, enhancing, or suppressing an immune response.
Loading dose	A larger than normal dose administered as the first in a series of doses, with the other doses being equal to each other but smaller than the first. A loading dose is administered to achieve a therapeutic amount in the body more rapidly.
Nutraceutical (functional food)	A food or supplement that has specific health benefits.
Off-label use	Prescription or use of a drug for conditions other than those approved by the FDA.
Over-the-counter (OTC)	Pharmaceuticals sold without prescriptions.
Pharmacodynamics	How drugs produce their effects, such as interactions at a receptor site.
Pharmacogenetics	The study of how drugs interact with the genetic makeup of an individual or the genetic response to a drug; this may be one of the first clinical applications derived from the Human Genome Project.
Pharmacogenomics	Studies that illustrate similarities and differences in pharmacodynamic and pharmacokinetic mechanisms among various individuals and people of different ethnic backgrounds.
Pharmacokinetics	The movement of drugs in the body, specifically encompassing the study of factors that determine the amount of chemical agents at their sites of biologic effect at various times after the agent is administered. Pharmacokinetics encompasses four specific factors: absorption, distribution, metabolism (biotransformation), and excretion (clearance).
Pharmacotherapeutics	The field concentrating on the treatment effects of drugs. There are several subsections within pharmacotherapeutics.
Polypharmacy	The practice of treating individuals using multidrug regimens. This term generally is accepted to mean administration of five or more drugs.
Prescriptive authority	Legal ability to prescribe drugs, medical devices, or other regulated healthcare interventions.
Safety level	Plasma level or dose at which a drug's known adverse effects are not apparent.
Side effect	A physiologic response unrelated to the desired drug effects that occurs with therapeutic doses of the medication; it may be either beneficial or negative.
Therapeutic window	A point at which the plasma drug concentration is between the minimum effective

	concentration (MEC) in the plasma for obtaining the desired drug action and the mean toxic concentration (MTC).
Toxicology	The branch of pharmacology that deals with the nature, effects, and treatments of poisons.

Drug Categories and Naming Conventions

Drugs can be categorized in many different ways. For example, drugs can be described by the physiochemical property of the drug (e.g., an acid) or by the pharmacotherapeutic indication (e.g., sedative). Drugs also have chemical names, generic names, and brand names. The same drug could be identified by the chemical name ($C_{17}H_{21}NO$), the generic name (diphenhydramine), or the most common brand name (Benadryl).

Drug Development and Regulation

Drugs formulated as medications are approved for marketing by FDA. Pharmaceuticals for which the FDA has authority may be obtained by prescription, over the counter, or even behind the counter. The FDA also regulates medical devices such as intrauterine contraceptive systems (e.g., ParaGard, Mirena) and biologic agents such as vaccines. The FDA does not regulate dietary supplements, such as St. John's wort, although the agency does track adverse reactions that occur following use of dietary supplements. Similarly, the FDA may require labeling to designate that safety has not been established for cosmetics, although recall of cosmetics is voluntary by the manufacturer.

Drugs that are approved for specific use by the FDA must, in a series of preapproval studies that are submitted to the FDA for review, demonstrate both *safety* and *effectiveness* for a stated indication. Private-sector pharmaceutical companies conduct research, develop, manufacture, and market both prescription and *over-the-counter* (OTC) drugs. The FDA itself does not develop, manufacture, or test drugs. However, the Center for Drug Evaluation and Research (CDER), a subgroup within the FDA, oversees this process. Drug manufacturers submit full reports of clinical trials conducted on drugs, so that CDER can evaluate the data and determine whether the drug should be approved for a specific indication. This approval commonly is termed FDA labeling.

The preapproval clinical trials are intended to be appropriately large enough to determine safety and effectiveness. For a new drug to be introduced to the marketplace, several steps must occur. These steps are organized into four phases (**Figure 9-1**).[5] Phase 1 studies tend to be small and determine safety by exploring the basic pharmacology of the drug. Sometimes this phase is unnecessary because such studies have been conducted internationally and the information is already available. Phase 2 trials are designed to verify effectiveness. If the agent is intended to treat persons with a specific condition, the drug developer performs controlled clinical trials with members of that population. Phase 3 trials are randomized clinical trials with larger numbers of participants. Despite the larger size of the Phase 3 trials, many, if not most, adverse effects of drugs do not emerge until the postmarketing Phase 4. If risks or adverse reactions are identified after the drug is marketed, CDER takes action to inform the public, change a drug's label, or, when necessary, remove the product from the market.

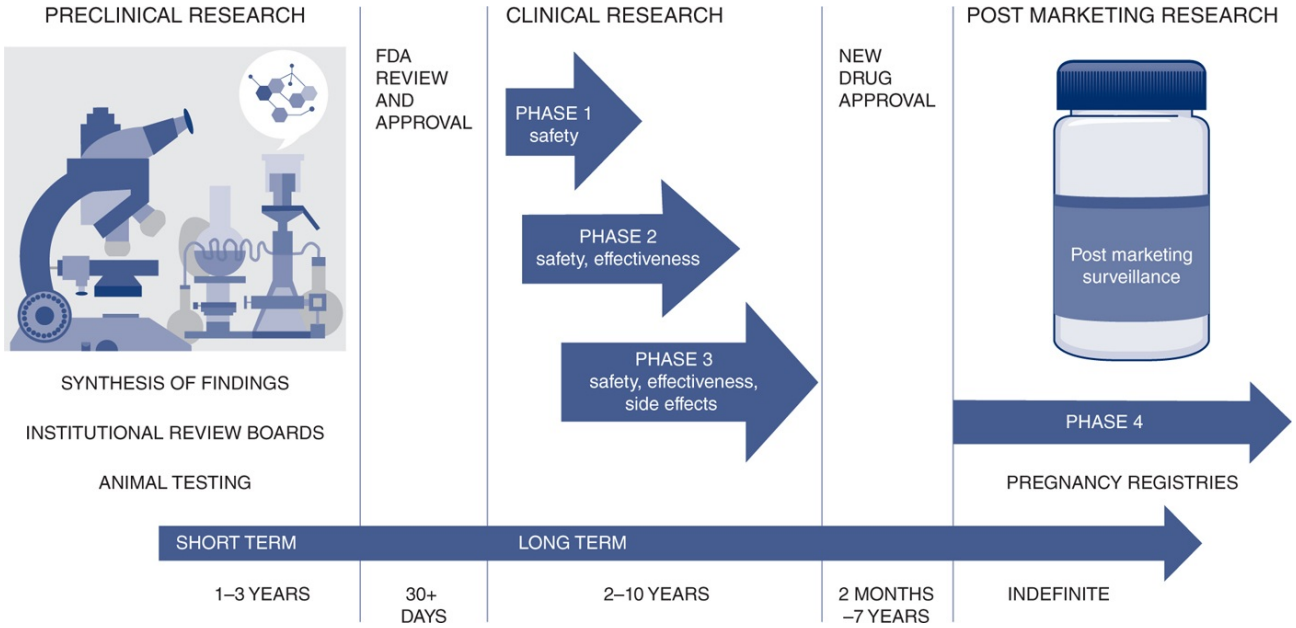


Figure 9-1 U.S. drug approval process.
Note: The lengths of time in the figure are approximate times.
Based on U.S. Food and Drug Administration. New drug approval process. Available at: https://www.drugs.com/fda-approval-process.html. Accessed August 17, 2017.[5]

Historically, children and women have not been participants in most drug development studies.[6] Although many reasons are cited to justify this exclusion, one factor is the changing environment in a woman's body over the course of the menstrual cycle, which could affect the pharmacokinetics and pharmacodynamics of a drug. Even when women do participate in clinical trials, analyses of gender differences in drug response are rarely published. Moreover, clinical trials for contraceptives generally do not include many adolescents, because women younger than 18 years are considered pediatric patients. Pediatrics itself is a difficult area in which to conduct clinical trials because of the inherent problems in obtaining informed consent and ethical concerns that drugs may adversely affect organs during growth and development.

After a drug receives FDA approval for treatment of a specific indication and becomes available for use, it may be prescribed for a different indication—a phenomenon called *off-label use*. Examples abound regarding off-label use.[7] For instance, the most commonly used tocolytic in the United States is magnesium sulfate, yet magnesium sulfate is not FDA approved for treating preterm labor. Methotrexate (Rheumatrex)—a folic acid antagonist used as a chemotherapeutic agent—has a marked predilection for destruction of trophoblastic tissue and is used off label for medical treatment of unruptured ectopic pregnancy. Although off-label use is common, it should not be undertaken capriciously. A legal liability potentially exists, especially if the drug is not widely used in practice for the off-label indication.

Controlled Substances

The U.S. Drug Enforcement Administration has a special role in the regulation of prescription drugs that have the potential for abuse, under the 1970 Controlled Substance Act (CSA). The CSA classifies pharmaceuticals that can be abused into one of five schedules based on the

substance's medicinal value, harmfulness, and potential for abuse or addiction. Schedule I is reserved for the most dangerous drugs that have no recognized medical use, such as heroin. Schedule V is the classification used for the least dangerous drugs, such as brand-name antitussives containing small amounts of codeine. Knowledge of these schedules is important for midwives with prescriptive authority, because prescriptive authority is often limited to specific schedules. **Table 9-2** lists the five schedules for controlled substances.

Table 9-2 U.S. Food and Drug Administration Classification of Controlled Substances

Schedule	Interpretation
I	High potential for abuse and no current accepted medical use. Examples are heroin and LSD.
II	High potential for abuse. Use may lead to severe physical or psychological dependence. Examples are opioids, amphetamines, short-acting barbiturates, and preparations containing codeine. Prescriptions must be written in ink or typewritten and signed by the practitioner. Verbal prescriptions must be confirmed in writing within 72 hours and may be given only in a genuine emergency. No renewals are permitted.
III	Some potential for abuse. Use may lead to low-to-moderate physical dependence or high psychological dependence. Examples are barbiturates and preparations containing small quantities of codeine. Prescriptions may be oral or written. Up to five renewals are permitted within 6 months.
IV	Low potential for abuse. Examples include chloral hydrate, phenobarbital, and benzodiazepines. Use may lead to limited physical or psychological dependence. Prescriptions may be oral or written. Up to five renewals are permitted within 6 months.
V	Subject to state and local regulations. Abuse potential is low; a prescription may not be required. Examples are antitussive and antidiarrheal medications containing limited quantities of opioids.

Modified with permission from U.S. Department of Justice, Drug and Enforcement Administration, Office of Diversion Control. Mid-level practitioners authorization by state. Available at: https://www.deadiversion.usdoj.gov/drugreg/practioners/. Accessed August 16, 2017.[8]

A registration number issued by the DEA is needed to prescribe a controlled substance. In 1993, the DEA published a regulation that established a new category under which healthcare providers other than physicians, dentists, veterinarians, or podiatrists could receive individual DEA registration numbers granting controlled substance prescribing privileges consistent with the authority granted them under state law.[8] Under this regulation, providers such as certified nurse-midwives and certified midwives were given authority to prescribe controlled substances if approved to do so by the state or jurisdiction in which they practice.

Regulation of Herbal Therapies

More than 2000 years ago, Hippocrates documented the use of more than 200 herbal remedies. Today, many herbs and botanicals are sold as dietary supplements, even though their actions and safety largely remain unknown. Under the Dietary Supplement Health and Education Act of 1994 (DSHEA), a manufacturer must ensure that its dietary supplement is safe before the product is marketed in the United States.[9] Effectiveness is not required for dietary supplements. The FDA is responsible for taking action against any unsafe dietary supplement

product after it reaches the market, but manufacturers are not required to register with the FDA or to seek FDA approval before producing or selling dietary supplements. The FDA's postmarketing responsibilities include monitoring safety concerns, through voluntary dietary supplement adverse event reporting. The FDA also monitors product information, such as labeling, claims, package inserts, and accompanying literature.

Much of the information available about the safety and effectiveness of herbs is anecdotal or reflects expert opinion. Some evidence-based information can be obtained, however. In 1978, a regulatory agency was established in Germany to evaluate the effectiveness of herbal remedies. Called the German Commission E, this group of 24 scientists evaluated studies (clinical, case, field) and prepared scientific monographs. By 2004, only herbs that the German Commission E found to be effective and to carry a low risk of adverse effects were expected to be available in Germany. Although the work of these scientists generally has been lauded, many common North American herbs are not included in the German registry.[10] An English translation of the German monographs has been published by the American Botanical Council.[11]

In the United States, the National Center for Complementary and Alternative Medicine (NCCAM) is currently conducting multiple studies that are comparing herbal remedies to conventional pharmaceutical treatments for specific conditions, such as endometriosis. Until such scientific evidence becomes available, claims involving therapeutic uses of herbs should be viewed with caution.

Prescriptive Authority

All 50 states grant some type of prescriptive authority to certified nurse-midwives.[1] Prescriptive authority remains less common for certified midwives, as their credential is newer and their numbers fewer. Prescriptive authority is controlled on the state level and ranges from very limited to relatively broad. Many states limit prescriptive authority according to the schedule of controlled substances. Some prescriptive authority laws are based on specific wording in state licensing laws or practice sites.

The Prescription

Midwives with prescriptive authority have the legal ability to write prescriptions. Although many prescriptions are phoned into a pharmacy or, increasingly, transmitted by electronic means, all prescriptions have some required components, most of which are established by state law and therefore vary from state to state. All handwritten prescriptions must be legibly written in indelible ink. Every prescription must include the midwife's name, address, and contact information. It is optimal to have the midwife's prescriptive authority number or other identifying information appear either on the top of the prescription order or adjacent to the midwife's signature. In some states, information about the midwife's collaborating physician must also be placed on the prescription. All prescriptions are dated, and information about the prescription is placed in the woman's record so that if she loses the prescription, changes pharmacies, or moves, it can be easily retrieved.

Only one drug can be written on each prescription blank. Refills, if any, are noted, as certain health plans limit coverage of drugs on a monthly basis. Permission to substitute a brand-name product for a generic drug is often included. Some authorities maintain lists of abbreviations that may be used on prescriptions, such as “tid” for “three times a day,” although use of any abbreviations is increasingly discouraged.

The section of the prescription specific to the medication consists of four parts: (1) superscription, (2) inscription, (3) subscription, and (4) signature. The superscription includes the symbol “Rx,” although according to World Health Organization guidelines, it is more proper to use the Latin “

”. In any case, this symbol is derived from the Latin word for “recipe” or “take.” The inscription specifies the ingredients and their quantities (e.g., nitrofurantoin 100 milligram capsules). In the inscription, it is important to avoid decimals and, where necessary, write words in full to avoid misunderstanding. For example, write “levothyroxine 50 micrograms,” not “0.050 milligram” or “50 mcg.” The subscription informs the pharmacist how to compound or dispense the medication (e.g., dispense 10 capsules). The signature, or “sig,” is not that of the prescriber, but rather represents the Latin “signa” or “mark” that provides the instructions that enable the woman to understand dosages and when to take a drug. The sig should include route, frequency, duration, and any other specific information—for example, “Take 1 capsule by mouth at bedtime for 10 days.”

In general, it is recommended that the generic or nonproprietary name be used when a prescription is written. Use of generic names enables the pharmacist to maintain a more limited stock of drugs and/or dispense the least expensive drug. However, if there is a particular reason to prescribe a brand-name drug, the trade name can be added. Some authorities allow generic substitution by the pharmacist and require the addition of “Do not substitute,” “Dispense as written,” or “Brand medically necessary” to the prescription if that brand, and no other, is to be dispensed. When a specific brand is required, the midwife should document that instruction in the woman’s chart, along with the rationale. This documentation is not only created for completeness, but also may be necessary for the prescription to be covered by a public insurance program such as Medicaid, various managed care groups, or other types of health insurance. **Figure 9-2** illustrates a sample prescription written appropriately by a midwife.

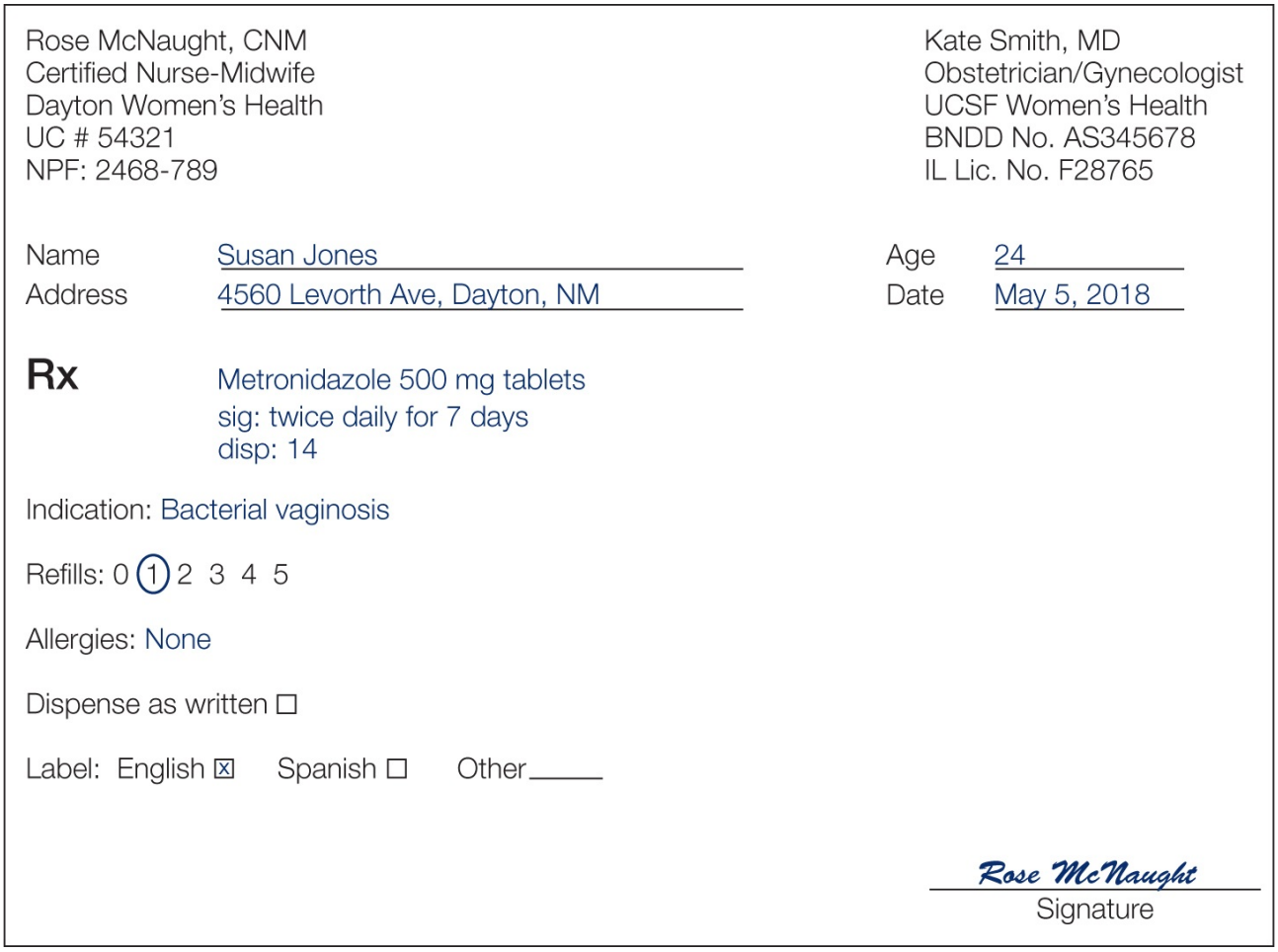


Figure 9-2 A sample prescription.

Principles of Clinical Pharmacology

Drugs undergo multiple biochemical transformations in the human body. Some are beneficial, some have no clinical effect, and some may be adverse effects. In addition, clinical situations such as pregnancy or renal disease can significantly alter an individual's response to a drug. To prescribe drugs safely, the clinician should be able to anticipate and recognize both the expected results and any adverse effects. A review of each drug's specific pharmacokinetics, pharmacodynamics, pharmacogenetics, and adverse effects supplies the knowledge that is needed to safely prescribe a drug in a manner that optimizes effectiveness.

Pharmacokinetics

Pharmacokinetics addresses the kinetics of drug absorption, distribution, metabolism, and excretion. Each of the four phases of pharmacokinetics effects how much of the drug reaches the target organ.

Absorption

Absorption is affected by the route of administration, pH of the drug, pH of the gastrointestinal tract, lipophilic or hydrophilic properties of the drug, entero-hepatic circulation (first-pass effect),[12] and the presence of other agents (drug–drug or drug–food interactions). The first-pass effect, which is also known as pre-systemic absorption, is most commonly associated with oral drugs. Pharmaceuticals administered orally must contain higher amounts of a drug to account for the loss that occurs through first-pass metabolism in the liver prior to the agent arriving in the circulation. When pharmaceuticals are administered using the transdermal route, such as hormone patches, the product may contain smaller amounts of the drug, as the active ingredient is directly absorbed into the circulation and avoids the initial metabolism by the liver.

Distribution

Distribution of the drug is affected by the size of the drug molecule as well as the drug's affinity for aqueous and lipid tissues, tissue permeability, systemic circulation, protein binding, and pH. There are two main phases of distribution: steady state and half-life. *Steady state* refers to the concentration of a drug in the systemic circulation when the rate of drug elimination equals the rate of drug availability. The *half-life* of a drug refers to the time it takes for the plasma concentration or the amount of drug in the body to be reduced by 50% (i.e., by half). Half-life determines the time required to reach steady state and, therefore, the time interval between doses. Steady state is usually reached after four to five half-lives, and elimination takes another four to five half-lives.

Another important pharmacokinetic concept when considering drug administration is *loading dose*. A loading dose is one or a series of doses that are given at the initiation of therapy so that the drug will reach the target plasma concentration rapidly. A loading dose is higher than the standard dose and is used to rapidly obtain a steady state when a quick therapeutic response is needed.

Protein binding can be one of the most important clinical factors in distribution of a drug. *Protein binding* refers to the fraction of total drug in the plasma that is bound to plasma proteins.[13] Albumin is the most common plasma protein that binds to drugs in plasma. Once bound to protein, the drug is altered or too large to diffuse out of the circulation and attach to a cell membrane receptor. Thus, only the unbound, or free, drug is distributed to its intended site of action in tissue. In turn, the drug's physiologic effect is related to the free drug concentration, rather than the total circulating plasma drug concentration.

Metabolism

Drug metabolism refers to the process by which a drug is chemically converted in the body so that it can be excreted via the kidney, feces, lung, or skin. The enzymes responsible for drug metabolism are mainly located in the liver, although some other tissues—such as kidney, lung, small intestine, and skin—also contain enzymes that metabolize drugs. The cytochrome P450 (CYP450) enzyme family is the group of enzymes that are responsible for the majority of drug metabolism reactions.[14]

For most drugs, metabolism results in changing the drug to a pharmacologically inactive compound; however, some drugs have no pharmacologic activity themselves, rather their metabolite is the active pharmacologic agents. These drugs are referred to as *prodrugs*. For example, valacyclovir (Valtrex) is not an effective antiviral drug, but its active metabolite, acyclovir, is active against herpes virus.[15]

Excretion or Clearance

Ultimately the amount of drugs removed from the body is the measurement of clearance. This calculation reflects the rate of drug excretion. Although most drugs are cleared through the renal system, some may be eliminated through other organs such as the lungs.

Pharmacodynamics

Pharmacodynamics refers to the relationship between the concentration of a drug in the systemic circulation and the biologic response that occurs, often termed the dose–response relationship. The minimum effective concentration is the lowest dose at which a desired response is noted.

Once at the target organ, a drug can act as an agonist, antagonist, partial agonist, or agonist–antagonist. *Agonists* elicit a pharmacologic response. Insulin and morphine are examples of drugs that act as receptor agonists. *Antagonists* do not activate the receptor when bound to it, so they prevent activation of the receptor by an agonist. Antihistamines are antagonists in that they block the histamine activated-receptor response to an allergen. A *partial agonist* elicits a partial pharmacologic response when bound to a specific receptor. *Agonist–antagonists* are similar to partial agonists but work via a different mechanism. These drugs affect receptors that have different forms, and they act as an agonist at one form and as an antagonist at another. For example, butorphanol (Stadol) blocks the effect of morphine at the mu opioid receptor but stimulates the kappa opioid receptor. The net result is analgesia that is not as potent as the

effect obtained with morphine.

Individual Variability in Drug Response: Pharmacogenetics, Race, and Gender

It has long been known that different individuals exhibit *biovariability*, or a different response to the same drug at a standard dose. Many drugs have wide variability in effectiveness for individuals.[16] This inter-individual variability in drug response may be due to extrinsic factors such as illness, smoking, diet, or drug–drug interactions; alternatively, it can be due to physiologic factors such as genetics, race, age, and gender.

Genetic differences account for approximately 15% to 30% of inter-individual differences in drug responses, and the field of pharmacogenetics is becoming more important clinically as knowledge about genetic influences on drug response proliferates. The two primary areas in which genetic differences affect drug response are metabolism by the CYP450 enzyme family and function of the cell receptors for drugs. Many variants, also known as polymorphisms, exist within the CYP450 enzyme family. CYP450 enzyme polymorphisms may result in a person being a poor (slow) metabolizer, an intermediate metabolizer, an extensive metabolizer, or an ultra-rapid metabolizer of a given medication.[17,18] When administered the same dose, persons who are poor metabolizers have elevated plasma concentrations of a drug and a greater risk of toxicity compared to persons who are extensive or ultra-metabolizers.[19] In contrast, a polymorphism that involves duplication of a specific CYP450 enzyme causes a person to be an ultra-rapid metabolizer of drugs that are metabolized by that enzyme, which can result in subtherapeutic responses to normal doses. Polymorphisms of the genes encoding the CYP enzyme family are distributed differently among racial and ethnic groups. For example, the prevalence of CYP2D6 ultra-rapid metabolizers is approximately 1% to 10% in Caucasians, 3% to 5% in African Americans, 16% to 28% in North Africans, and 21% in Asians.[18]

Differences in cell membrane receptors for drugs also lead to different pharmacodynamic responses. Polymorphic differences in drug receptors are differentially distributed among racial and ethnic groups. For example, persons of African American descent who have asthma are more likely to respond with exacerbations and increased morbidity when given long-acting beta-adrenergic agonists without concomitant therapy with an inhaled corticosteroid because the $beta_2$-adrenergic receptor downregulates (rather than upregulates) when those individuals are exposed to a $beta_2$-adrenergic agonist.[20]

The effect of gender is likely related to both drug metabolism and drug receptor differences. Sex hormones such as estrogen and progesterone modulate the activity of CYP450 enzymes. Thus, men and women may experience different drug effects despite adjustment of the dose for body size. In addition, estrogen may affect the function of drug receptors. A meta-analysis of gender differences in the response to opioids found morphine's effects are more potent among women than among men and the onset/offset of those effects is slower in women than in men.[21] Additional research is needed in this area before the evidence about gender differences in the response to pain medications can be applied clinically.

Clinical implications of pharmacogenetic differences are the current subject of research.

Genome-wide association studies have identified some populations who may benefit from genome testing to guide pharmacotherapy, and the FDA has added pharmacogenomic information to the drug label of such drugs.

Adverse Drug Reactions and Adverse Drug Events

Adverse drug reactions (ADRs) are unintended responses to a drug that occur when normal dosing is used. ADRs are quite common.[22,23] The term *adverse drug events* (ADEs) is a more inclusive category connoting any injury that results from the administration of a drug. ADEs includes adverse drug reactions, medication errors, overdoses, and known dose-related side effects.

After a drug has been determined to be safe and effective in preapproval clinical trials, it is prescribed for individuals in a broader population after approval—that is, for a more diverse group than the relatively select group who participated in the clinical trials. Thus, adverse effects of drugs are primarily identified during the postmarketing period. The Adverse Event Reporting Program of the FDA collects reports of ADRs via the FDA MedWatch program. Details of how to report an adverse drug reaction are listed in **Table 9-3**.

Table 9-3 How to Report an Adverse Drug Reaction

In 1993, the U.S. Food and Drug Administration (FDA) established *MedWatch*, a reporting system for adverse events or sentinel events. This system includes a computerized information database designed to support a postmarketing safety surveillance program for all FDA-approved drug and therapeutic biologic products.

Any suspected adverse events or reactions to medications, drug products, or medical devices (other than vaccines or investigational drugs) should be reported using FDA Form 3500 (available online at https://www.fda.gov/downloads/aboutfda/reportsmanualsforms/forms/ucm163919.pdf).

Information regarding the reporting of vaccine-related adverse event reports (VAERS) can be found at https://vaers.hhs.gov.

For individuals who prefer, the MedWatch program can be contacted by phone, fax, or mail, as well as online.

Phone: 1-800-332-1088

Fax: 1-800-FDA-0178

Mail: MedWatch
5600 Fishers Lane
Rockville, MD 20857

Adverse drug reactions are classified as either immune or non-immune reactions. Non-immune reactions are predictable. They include drug–drug interactions, drug overdose, and drug toxicity, and commonly occur secondary to known pharmacologic effects. Conversely, immune reactions, which are also called allergic reactions, are unpredictable. The four types of allergic or hypersensitivity reactions are listed in **Table 9-4**.

Table 9-4 Types of Hypersensitivity Reactions

Type	Timing of Reaction	Examples
Type I (immediate)	Minutes to hours after drug	Anaphylaxis from exposure to penicillin

	exposure	Hives following exposure to sulfa drugs
		Angioedema from ACE inhibitors
Type II (cytotoxic)	Variable	Transfusion reaction
		Hemolytic anemia from penicillin
Type III (immune complex formed)	1–3 weeks after drug exposure	Drug-induced lupus caused by minocycline (Minocin)
		Serum sickness caused by cefaclor (Ceclor)
Type IV (delayed or cell mediated)	2–7 days after drug exposure	Ampicillin rash
		Contact dermatitis caused by topical antihistamine

Abbreviation: ACE, angiotensin-converting enzyme.

Drug–Drug and Drug–Food Interactions

Adverse reactions may occur when certain drugs are used concomitantly, which is referred to as a drug–drug interaction. Adverse reactions may also occur when drugs are taken simultaneously with certain foods or botanical/herbal therapies. Assume drug A and drug B are both metabolized via the same cytochrome P450 enzyme. When these drugs are taken at the same time, drug A either inhibits or enhances the activity of the cytochrome P450 enzyme. In turn, metabolism of drug B is either inhibited, which results in higher plasma levels and perhaps overdose, or enhanced (i.e., it is metabolized more rapidly than usual), which results in a reduction of the plasma concentration of the drug, making it less effective. For example, the antifungal drug fluconazole (Diflucan) is an inhibitor of CYP2CP; when it is given to a person who is also taking tolbutamide (Micronase), the plasma concentration of tolbutamide increases and the individual becomes more likely to experience hypoglycemia. In another example, cardiac arrhythmias may occur when one of the macrolides erythromycin (E-base), azithromycin (Zithromax), or clarithromycin (Biaxin) is taken simultaneously with the antifungal ketoconazole (Nizoral).[24]

Pharmacotherapeutics in Primary Care

The drug categories most commonly used in primary care are vitamins, analgesics, antihistamines, antihypertensives, antimicrobials, hormonal formulations used for contraception, and immunizations. It is not possible to review these topics in depth in this chapter, and readers are referred to other texts for more detail. Instead, this section introduces pharmacologic considerations for analgesics, antimicrobials, and antihistamines—key drug categories that incorporate the pharmacologic principles and clinical implications that are inherent in all drug categories. References that have more in-depth information are provided where relevant.

Analgesics

Analgesic medications are categorized as non-opioid analgesics and opioid analgesics. Non-opioid analgesics include aspirin, acetaminophen (Tylenol), and nonsteroidal anti-inflammatory drugs (NSAIDs).[25] Opioid analgesics are a family of drugs with an action similar to morphine. For a variety of reasons, there is an epidemic of opioid overuse and overdoses in the United States; it is imperative for clinicians to appreciate and ensure the appropriate use of these agents.[26]

Analgesics have pharmacologic effects that are broad and affect many physiologic functions. Consequently, this category of drugs requires that the prescriber be especially aware of side effects and adverse effects. NSAIDs mitigate pain via their ability to inhibit cyclooxygenase (COX), the enzyme responsible for synthesizing prostaglandins.[27] Prostaglandins protect the gastric mucosa, inhibit platelet aggregation, and stimulate uterine contractions, in addition to mediating the inflammatory response and sensitizing pain receptors. Thus, these drugs can have multiple predictable side effects that can also be adverse effects.[28] Both gender differences and individual differences are noted in the pain response and in the response to many analgesics.[29-31] The various hormonal and genetic factors underlying these differences have yet to be fully elucidated.

Because analgesia may be potentiated when drugs that have different mechanisms of action are combined, it is common to combine an opiate with a non-opiate in one formulation. For example, the combination of acetaminophen and codeine comes in different strengths of acetaminophen (e.g., Tylenol #1, #2, or #3). Combination drugs have additional pharmacologic implications. For example, although the acetaminophen/codeine combinations are usually prescribed on the basis of the 30 mg of codeine in each tablet, the maximum dose is actually limited by the maximum dose of acetaminophen that is safe to take in a 24-hour period (i.e., 4 grams/24 hours). When choosing a specific formulation, the analgesic capability of a particular formulation should be assessed and matched to the pain control needs of the woman.[30]

Increasing concerns about treating pain have resulted in a marked increase in the use of and prescriptions for opioids over the last decade. Unfortunately, this trend has resulted in a significant rise in the prevalence of opioid misuse.[32,33] Strategies for treating pain without increasing the risk of opioid misuse are currently the subject of much research. In addition, several states have implemented policies or regulations that guide opioid prescribing.[32-35]

Data are constantly emerging on this topic.

Antimicrobials

Antimicrobial drugs include agents directed against bacteria, fungi, viruses, and parasites. Each type of microorganism targeted by these drugs has a different biochemical structure, and consequently each type of antimicrobial has a different chemical structure and biochemical action. In general, antifungal and antiviral drugs attack protein structures that are not present in human cells, so these drugs are less likely to have adverse side effects. At the other end of the spectrum are drugs directed at parasites, which are multicellular organisms that share many similarities with human cells. Safe and effective antiparasitic drugs are particularly difficult to create.

Antimicrobials and analgesics are the most common prescriptions written in primary care practice. Unfortunately, drug resistance is an increasingly problematic trend that is directly related to the overuse of antibiotics for conditions that do not actually require an antimicrobial agent.[36-39] Thus, the first step when using these agents is to make sure they are required. Eight principles describe the critical clinical considerations that must be taken into account prior to prescribing an antimicrobial drug:

1. Confirmation of the presence of an infection through history and physical examination
2. Identification of the pathogen when possible
3. Confirmation of the need for an antimicrobial agent rather than use of palliative therapies and infection control measures
4. Understanding of host factors that may influence pharmacodynamics as well as the individual's concerns and resources
5. Selection of an appropriate antimicrobial agent using the dual principles of narrowest spectrum and shortest effective duration possible
6. Knowledge of the pharmacokinetics and pharmacodynamics of selected medications
7. Education of the individual and family regarding appropriate use of the antimicrobial
8. Appropriate monitoring of the therapeutic response[38]

Antibacterial drugs are either bacteriostatic (i.e., they inhibit bacterial growth) or bacteriocidal (i.e., they kill bacteria) in nature. More generally, these agents are classified by their mechanism of action: (1) They attack the cell wall (penicillins, cephalosporins, and vancomycin); (2) they inhibit or alter protein synthesis (macrolides, tetracycline, and aminoglycosides); or (3) they interfere with bacterial DNA (fluoroquinolones, antiprotozoan drugs, and isoniazid [INH]).

Inappropriate prescribing of antibiotics for viral infections and use of third- and fourth-generation broad-spectrum agents when a narrow-spectrum agent is available are by far the most important problems facing midwives who may be prescribing these agents. There are two critical components to the solution to these problems. First, clinical guidelines are available from most professional associations as well as individual institutions; these guidelines can help the prescriber choose the correct therapy. Second, thorough health education that is both

culturally sensitive and provided at the correct health literacy level is essential. As a corollary to this general rule, when an antimicrobial agent is prescribed, detailed information about dose, timing, specific rules about taking the medication with food or drink, and possible side effects or adverse effects is also part of the required health education.

Drug Resistance to Antimicrobials and Antibiotic Stewardship

Shortly after drug manufacturers began mass production of penicillin in the 1940s, infections secondary to resistant *Staphylococcus aureus* began to appear. The subsequent spread of antibiotic resistance occurred rapidly.[40] This rise in antibiotic resistance has been directly correlated with increased use of antimicrobials.[41]

Several factors account for the development and rapid rise of drug-resistant strains of bacteria. One factor is the human complacency that developed in the 1980s, when many scientists and clinicians began to view bacterial infections as easily curable conditions. Antimicrobial agents were used liberally, even for conditions for which they were not indicated, such as the common cold. When susceptible bacteria were eradicated, the few survivors were those organisms that were resistant to the antimicrobial agent. Through natural selection, these microbes then became the predominant microorganism. Interestingly, use of antibacterial soaps and cleansers may contribute to this problem. The antibacterial agents added to a variety of substances, such as soaps, lotions, and cleaning supplies, can act like antibiotics in the selection of resistant strains of microbes; these products are no more effective than similar agents without the antibacterial additives, and controversy has arisen regarding whether they may contribute to drug resistance.[36]

Resistant microbes have several mechanisms of action. For example, penicillin resistance is the result of gene action. Because penicillin inhibits enzymes involved in cell wall synthesis, some organisms may alter their cell walls so that the antibiotic cannot bind to it. These organisms are called beta-lactamase inhibitors because they produce an enzyme called beta-lactamase that breaks open the beta-lactam ring of the penicillin molecule.[42] In contrast, resistance to quinolones emerges when organisms evolve in a way that alters the ability of the drug to penetrate its target.[43]

Bacteria can acquire genes that provide resistance to antimicrobial drugs in one of three ways: (1) spontaneous DNA mutation (e.g., drug-resistant tuberculosis); (2) transformation in which one bacterium acquires and then replicates DNA from another bacterium (e.g., penicillin-resistant gonorrhea); and (3) resistance acquired from a small circle of DNA called a plasmid that can move from one type of bacterium to another (e.g., *Shigella*). The third type is the most troublesome, because following plasmid acquisition, the bacteria may simultaneously become resistant to several types of drugs.

Antibiotic stewardship refers to a coordinated program that addresses “the optimal selection, dosage, and duration of antimicrobial treatment that results in the best clinical outcome for the treatment or prevention of infection, with minimal toxicity to the patient and minimal impact on subsequent resistance.”[39,44,45] Antibiotic stewardship programs focus on use of appropriate antimicrobials, decreasing overuse and misuse of these agents, and minimizing the development of antibiotic resistance. Many hospital settings now have antibiotic

stewardship teams. Examples of antibiotic stewardship interventions include limitations on formularies, computer-aided decision support programs, and feedback to clinicians about their prescribing practices. These techniques have been shown to be both effective and cost saving.[37-39] A useful mnemonic when writing a prescription for an antibiotic is to consider the following four items: Right Drug, Right Dose, De-escalation to pathogen-directed therapy, and Right Duration of therapy.[46] The Resources section at the end of this chapter includes resources related to antibiotic stewardship.

Antihistamines

Four different types of histamine receptors (H_1, H_2, H_3, H_4) are found in multiple tissue sites within the body. Antihistamine drugs that target these receptors have a wide range of actions and uses. Antihistamines are common OTC medications.

H_1 receptors are found in the muscles that line blood vessels (vascular smooth muscle) and in nervous tissue. These receptors are involved in the inflammatory response and allergic reactions. H_2 receptors are located in the gastric mucosa. Stimulation of these receptors causes secretion of gastric acid. H_3 and H_4 receptors are found in the brain, heart, eye, and breast tissues, as well as in immune cells. These receptors appear to be involved in circadian rhythms, allergic reactions, and perhaps carcinomas, although their exact functions are not yet well elucidated.

Antihistamines that act as antagonists at H_1 receptors are used to treat allergic reactions, nausea and vomiting, and motion sickness. First-generation H_1 antihistamines have a duration of action of approximately 4–6 hours and can affect different types of histamine receptors. These drugs cross the blood–brain barrier and cause sedation—the most common side effect associated with antihistamines. When sedation is the desired effect, it is important to note that tolerance to sedation develops within a few days. Thus, drugs such as diphenhydramine hydrochloride (Benadryl) and doxylamine succinate (Unisom Sleep Tabs) should be taken for a few days only.

The other side effect of antihistamines that is of clinical concern relates to their anticholinergic effects. This action causes a drying of mucous membranes, which is helpful in treating allergies, but can cause urinary retention and blurred vision. The primary contraindications to antihistamines are those wherein sedation or anticholinergic effects would cause adverse outcomes. For example, persons with glaucoma or hyperthyroidism should not use antihistamines.

The second-generation H_1 antihistamines have a longer duration of action, which can be as long as 24 hours. In addition, they are more selective for peripheral H_1 receptors and, therefore, do not cause sedation. The second-generation H_1 antihistamines, however, have more drug–drug interactions than do the first-generation drugs in this category.[47]

The H_2 antihistamines are used to treat gastric disorders in which hyperacidity is a problem. These antihistamines have very few side effects. Cimetidine (Tagamet) has many drug–drug interactions and, for this reason, is not considered the first H_2 antihistamine of choice.

Famotidine (Pepcid AC) is more potent than either cimetidine (Tagamet) or ranitidine (Zantac). The most common side effect of these drugs is headache, which occurs in 3% to 5% of persons who take these medications.

Use of Drugs During Pregnancy

Two key considerations must be taken into account when prescribing a drug for pregnant women: (1) physiologic changes of pregnancy that affect drug dosage and (2) effects of the drug on the fetus.

Pharmacokinetics in Pregnancy

The myriad physiologic changes that occur during pregnancy affect the pharmacokinetics of any drugs and herbs that the pregnant woman uses.[48] Pregnancy-related changes in pharmacokinetics, and some clinical examples of those changes, are summarized in **Table 9-5**.[48,49]

Table 9-5 **Pregnancy-Related Changes in Pharmacokinetics**

Pharmacokinetic Phase	Pregnancy Changes	Clinical Implications
Absorption	Increased progesterone production causes decreased intestinal motility and a 30–50% increase in gastric emptying time	Gestational nausea and vomiting may impair absorption
		Slower gastric emptying time may delay the onset of the drug response
	Gastric pH increases at midgestation	Increased exposure to bacteria in the intestine may decrease the bioavailability of some drugs
		Calcium and iron bind when ingested concurrently, thereby decreasing absorption of both minerals
Lung absorption	Respiratory minute volume increases approximately 50%	Dose requirements for inhaled drugs are decreased
Transdermal, subcutaneous absorption	Skin perfusion and skin hydration are both increased	Both lipophilic drugs and hydrophilic drugs are more rapidly absorbed transcutaneously
	Enhanced perfusion to muscles	Intramuscular absorption of drugs is more rapid and complete compared to absorption in nonpregnant individuals
Distribution	Plasma volume is expanded by approximately 50%	Hydrophilic drugs have reduced plasma concentration and need to be given in higher doses
	Body fat stores increase by 3–4 kg	Lipophilic drugs that concentrate in body fat may accumulate, such that prolonged effects could be seen following long-term use
Protein binding	Plasma albumin concentrations are reduced secondary to increased plasma volume	Increased free drug is available to exert pharmacologic effects
		Drugs that are highly protein bound will have more pharmacologic activity in a pregnant woman
Fetal–maternal distribution	The fetal compartment is available for distribution of drugs	The concentrations of most drugs in the fetal compartment tend to be 50–100% of the concentration in the maternal compartment
	The fetal circulation is more acidic than the maternal circulation	Highly lipophilic drugs of low molecular weight that have low protein binding can accumulate in the fetal compartment
	Fetal albumin concentration in plasma increases throughout pregnancy and is 20% higher than the maternal	Basic drugs such as meperidine (Demerol) can

	concentration at term	have higher concentrations in the fetal compartment than in the maternal circulation secondary to “ion trapping” Drugs that are highly bound to albumin can concentrate in the fetus at term
Metabolism	Changes in estrogen and progesterone affect cytochrome P450 enzyme activity CYP1A2 is inhibited CYP3A4 and CYP2C9 are increased	Metabolism of caffeine and theophylline (Theo-Dur) are inhibited or slower Metabolism of sertraline (Zoloft) and metoprolol (Lopressor) is enhanced or faster
Elimination	Glomerular filtration rate increases by 50% throughout pregnancy	More rapid clearance of most drugs that are eliminated renally

Some medications need dosage adjustments for pregnant women. For example, aminoglycosides such as gentamicin (Garamycin), as well as other antibiotics such as ampicillin (Omnipen, Polycillin, Principen) and cefazolin (Rocephin), achieve lower serum concentrations during pregnancy; therefore, to be effective, the doses of these medications need to be increased for pregnant women. Ampicillin, in particular, has serum concentrations in pregnant women that are 50% of the serum concentrations seen in nonpregnant women.[50,51]

Transport of Drugs Across the Placenta

Drug distribution is complex during pregnancy, as four separate compartments exist: (1) the fetus, (2) the amniotic fluid, (3) the placenta, and (4) the mother. Each of the compartments affects movement of drugs, and occasionally one compartment will have a higher concentration of drug than another.[52,53]

For a drug to move from the maternal circulation to the fetal circulation, it must transfer across the physical barrier between maternal and fetal circulation. The methods available for this transfer are diffusion, active transport, and movement via protein transporters. Most drugs move across the placental membranes by simple diffusion, although some take advantage of carrier-mediated mechanisms or use facilitated diffusion. Neither of these two methods requires energy or allows an agent to be transferred across the membrane to an area of higher drug concentration. Glucocorticoids and cephalexin (Keflex), for example, are transported via facilitated diffusion. This mechanism is utilized for transport of endogenous compounds such as hormones; likewise, it is used to transport drugs that are structurally similar to naturally occurring hormones.

Unlike passive transport, active transport requires energy. Pinocytosis is the most commonly used active transport method, a process during which a portion of the plasma membrane engulfs the drug molecule, creating a type of intracellular vesicle. Because of the energy and time required for pinocytosis, the small number of drugs that cross the placenta using active transport are also similar in structure to endogenous compounds that cross the placenta this way. Digoxin (Lanoxin) and valproic acid (Depakene), for example, cross the placenta via active transport.

The functions of protein transporters are quite interesting. These proteins extend from the intracellular space across the cell membrane into the extracellular space. Their function is to move substances from the placenta back into the maternal circulation. The protein transporters

(e.g., P-glycoprotein and multidrug resistance proteins 1, 2, and 3) protect the fetus from some drugs via this efflux mechanism.[13]

Drug transfer across the placenta is affected by various characteristics of the drug, including its size (molecular weight), lipid solubility, plasma protein binding, and acid versus basic properties.[48-54] Most drugs have a small molecular weight, usually less than 500 daltons (Da), so they are easily transferred across the placenta. Drugs with a molecular weight greater than 500 Da transfer incompletely; those that have a molecular weight greater than 1000 Da, such as heparin and insulin, transfer very poorly. Drugs that are more lipophilic cross the placenta more readily than those that are hydrophilic, as the placenta is lipoid in character. A non-ionized state facilitates transfer. When ionized basic drugs such as meperidine (Demerol) cross the placenta, they can accumulate in the fetal liver and adrenal glands and become ion-trapped in the fetus because the fetal circulation is more acidic than the maternal circulation.

Once a drug enters the fetal circulation, it is free to bind with proteins in the fetal plasma. Notably, albumin concentrations in the fetal compartment are higher than those in the maternal compartment. As a consequence, some highly protein-bound drugs such as diazepam (Valium) may have an increased fetal effect when compared with their maternal effects.

Teratogenic and Fetotoxic Effects of Drugs

Although most women do not plan to take drugs during pregnancy, almost 50% of all pregnancies are unintended and awareness of pregnancy typically occurs only after embryogenesis has begun. Thus, pregnant women may unknowingly be prescribed teratogenic medications.[55,56]

Teratology is essentially the study of congenital anomalies and teratogens or any agent that irreversibly alters growth, structure, or function in a developing embryo or fetus. Teratogens include viruses such as rubella, chemicals such as mercury, and drugs such as diethylstilbestrol (DES).[52,55-60] The term *fetotoxic* is used to describe agents, such as tobacco, that have toxic effects on the fetus and adversely affect growth or development.[57] The fetus is mostly likely to be exposed to teratogenic agents in the first trimester and to fetotoxic agents in the second and third trimesters.

Overall, birth defects occur in approximately 1% to 3% of all births.[58] This percentage is often called the “background risk” upon which additional risks are calculated based on family history, past history, and environmental exposure. Only 10% of birth defects can be associated with environmental factors, and the majority of environmental factors are not pharmaceutical. Drugs and chemical agents such as mercury and pesticides account for approximately 45% of the environmental teratogens involved in congenital anomalies. Drugs alone account for only 2% to 3% of all birth defects.[57-60]

The unique fact about teratogenic medications is that avoiding the teratogen can prevent the associated congenital anomaly. For this reason, knowledge of teratogenic drugs is essential for the practicing midwife. Fortunately, the number of teratogenic agents is relatively small, and even fewer are in common use.

The preimplantation period is considered the “all or nothing” period. If a small number of cells are damaged during this time period, the fetus usually compensates without any damage.

Conversely, if a large number of cells are damaged, the embryo will be lost and a spontaneous abortion will occur. The period of organogenesis—between 2 and 8 weeks post fertilization, or 4 to 10 gestational weeks—is the most critical period wherein teratogenic exposures can cause fetal malformations (**Figure 9-3**).[61] **Table 9-6** lists drugs that are known to have teratogenic or fetotoxic effects.

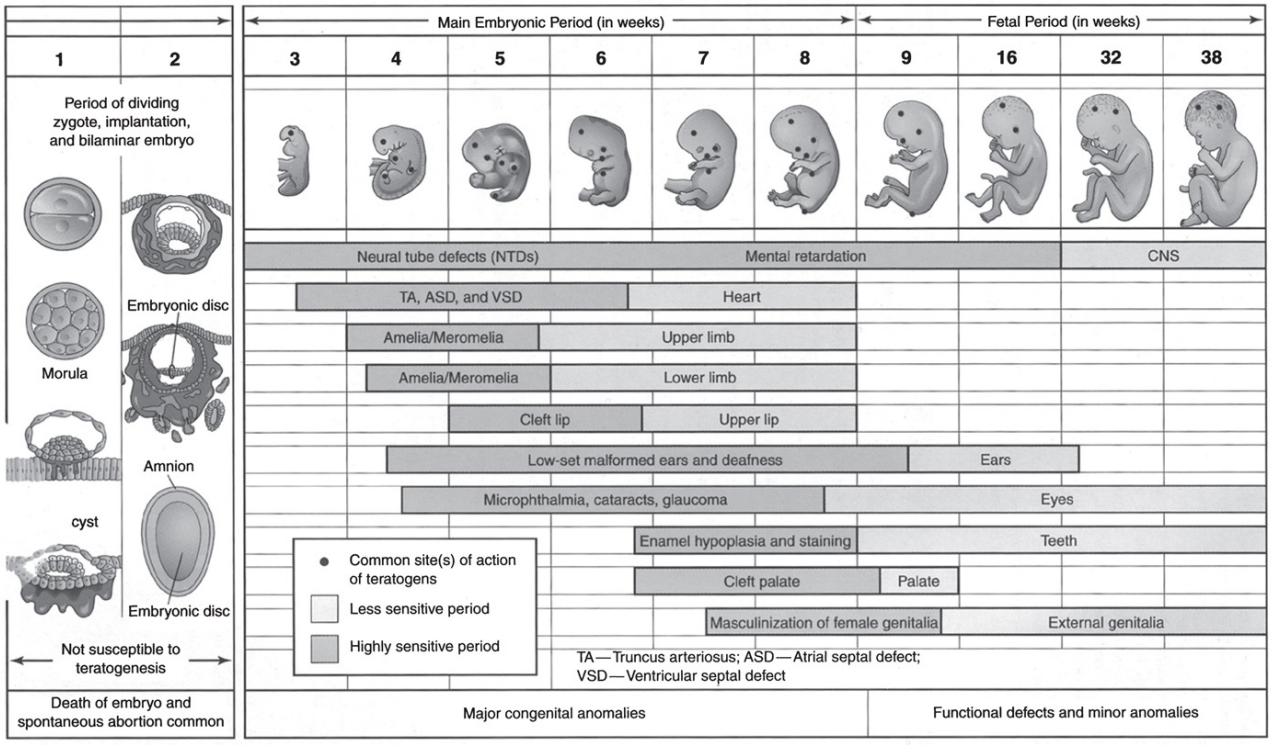


Figure 9-3 Early stages of development.
Reproduced with permission from Moore KL, Persaud TVN. *The Developing Human: Clinically Oriented Embryology.* 10th ed. Philadelphia, PA: W. B. Saunders; 2015.[61]

Table 9-6 Drugs That Have a Teratogenic or Fetotoxic Effect

Drug Category Name (Brand Name)	Teratogenic or Fetotoxic Effects	Clinical Implications
Androgens and testosterone derivatives: Danocrine (Danazol)	Virilization of females Advanced genital development of males	Dose dependent and based on critical period. Brief exposure rarely is significant. Before 9 weeks' gestation, labioscrotal fusion is common. Incidental, brief exposure usually has minimal risk.
Antibiotics: **Tetracyclines:** Tetracycline (Terramycin) Doxycycline (Adoxa)	Abnormalities of teeth discoloration	Critical period is after first trimester. Discoloration of permanent teeth is possible if exposure occurs after 24 weeks' gestation. No well-controlled studies of doxycycline have been performed, but case-controlled studies suggest it is not associated with teratogenic risk. Recommend that doxycycline be used only if it is the only effective agent.

Sulfonamides: Sulfamethoxazole (Bactrim, Septra)	Hyperbilirubinemia in neonates	Contraindicated in third trimester.
Chloramphenicol	Grey syndrome in neonates 2–9 days after therapy is administered	Oral chloramphenicol is contraindicated in pregnancy.
Aminoglycosides: Gentamycin Streptomycin Kanamycin	Neonatal ototoxicity	Streptomycin and kanamycin when administered in high doses to treat tuberculosis, has a clear risk of fetal ototoxicity. The risk associated with gentamycin may be smaller, but these antibiotics are not recommended for use in pregnancy.
Anticonvulsants: Carbamazepine (Tegretol) Phenytoin (Dilantin) Trimethadione (Tridione) Valproic acid (Depakene) Valproate (Depacon)	1% risk of neural tube defects, cardiovascular defects, developmental delays, and intrauterine growth restriction Reduced intelligence associated with use of valproic acid or valproate Phenytoin is specifically associated with cardiac defects and cleft palate	Critical period is all trimesters. Risk of neural tube defects is increased with use of anticonvulsants, especially when they are used with other antiepileptic drugs. Risk of neural tube defects is decreased with increased folic acid supplementation. Polytherapy with more than one anticonvulsant increases risk. Newer anticonvulsants such as gabapentin (Neurontin) do not appear to engender a risk of congenital anomaly.
ACE inhibitors: Captopril (Capoten) Enalapril (Vasotec) Lisinopril (Prinivil)	Intrauterine growth restriction Oligohydramnios Renal failure Decreased skull ossification Renal tubular dysgenesis	Risk of growth restriction is approximately 25%, with fetal morbidity of approximately 30%. Risk of adverse effects increases in the second and third trimesters (probably due to decreased uteroplacental flow). Beta blockers such as labetalol (Trandate) are safe in pregnancy.
Antidepressants: Fluoxetine (Prozac) Paroxetine (Paxil) Sertraline (Zoloft)	Paroxetine increases the risk of cardiac defects 1.5- to 2-fold; the other selective serotonin reuptake inhibitor–type antidepressants do not appear to have teratogenic effects Exposure in the third trimester is associated with neonatal withdrawal Persistent pulmonary hypertension noted in case reports	Overall, risks of teratogenic and fetotoxic effects are low. Antidepressants should not be discontinued during pregnancy. If starting antidepressant therapy during pregnancy, avoid the use of paroxetine.
Antineoplastic drugs: Cyclophosphamide (Cytoxan) Methotrexate (Rheumatrex)	Multiple birth defects and spontaneous abortion if used in the first trimester	If a woman needs one of these drugs, refer her for consultation with perinatologists and oncologists. Methotrexate is used to treat ectopic pregnancy.
Angiotensin II receptor blockers: Losartan (Cozaar)	Prolonged renal failure and hypotension in the newborn, decreased skull ossification, and renal tubular agenesis	Critical period is all trimesters.
Antithyroid drugs: Propylthiouracil (PTU) Methimazole (Tapazole)	Fetal and neonatal goiter, fetal hypothyroidism, and aplasia cutis are associated with methimazole but not propylthiouracil	Critical period is all trimesters. Women who need to take antithyroid drugs will generally be counseled to take propylthiouracil. The risk of fetal goiter is approximately 1–5%.

Aspirin	More than 150 mg/day is associated with prolonged gestation, prolonged labor, bleeding complications in the neonate, premature closure of the ductus arteriosus, and intrauterine growth restriction	Critical period is all trimesters. Although low-dose aspirin may be of benefit for women with antiphospholipid syndrome or lupus, normal adult doses should be avoided.
Benzodiazepines: Alprazolam (Xanax) Chlordiazepoxide (Librium) Diazepam (Valium)	Increased risk of neonatal withdrawal Some studies show increased risk of oral clefts with first-trimester exposure to diazepam	Early data on the association between benzodiazepines and oral cleft are controversial. Benzodiazepines are highly lipophilic and have a long half-life in the neonate.
Corticosteroids: Methyl prednisone (Medrol)	Possibly increased risk of oral and cleft defects	Critical period is first trimester. Because the risk is low, oral corticosteroids are used in the first trimester to treat hyperemesis gravidarum and other medical conditions. No risk associated with topical use.
Coumarin (Warfarin)	Bone defects Growth restriction CNS defects Developmental delays	15–25% risk when anticoagulants that impair vitamin K are used, especially between 6 and 9 weeks of gestation. Later use in pregnancy is associated with abruption, CNS defects, stillbirth, and hemorrhage of the fetus/newborn.
Ergot alkaloids: Ergotamine (Cafergot)	Spontaneous abortion Mobius syndrome Intestinal atresia Cerebral developmental abnormalities Low birth weight and preterm birth	Critical period is all trimesters. Sumatriptan (Imitrex) is an alternative to ergotamine for acute migraine treatment.
Folic acid antagonists: Methotrexate (Rheumatrex) Carbamazepine (Tegretol) Phenytoin (Dilantin) Phenobarbital (Solfoton) Primidone (Mysoline) Trimethoprim (Trimpex)	Spontaneous abortion Neural tube defects Cardiovascular defects Urinary tract defects	Drugs in many different drug categories are folic acid antagonists. Some sources suggest that the risks can be decreased with folic acid supplementation. Trimethoprim is a component of trimethoprim–sulfamethoxazole (Septra), which is commonly used to treat urinary tract infections. This drug should not be given in the first and third trimesters.
Lithium (Lithobid)	Cardiac defects Epstein's anomaly	Absolute risk is small, but alternative drugs should be recommended if possible.
Mifepristone (RU-486)	Antiprogestogen; used as an abortifacient Human data are limited	Primarily used for abortion in combination with misoprostol. Less effective when used alone.
Misoprostol (Cytotec)	Abortion Moebius sequence (brain stem ischemia, vascular disruption) for misoprostol	Potent uterostimulant capable of initiating uterine contractions at all gestational ages.
Nonsteroidal anti-inflammatory drugs:	Theorized premature closure of the ductus arteriosus Necrotizing enterocolitis	Contraindicated in general but especially in the third trimester.

Ibuprofen (Advil) Naproxen (Aleve)		
Retinoids: Isotretinoin (Accutane)	Multiple birth defects, including CNS, cardiac, and endocrine damage	Oral isotretinoin is contraindicated in pregnancy. Topical preparations are unlikely to have serious teratogenic effects but are still contraindicated because other agents can be used.
Statins: Atorvastatin (Lipitor) Lovastatin (Mevacor)	Interfere with cholesterol production Primarily have theoretical adverse effects on the fetus	Contraindicated in pregnancy.
Other: Thalidomide	Limb deficiencies Cardiac and gastrointestinal abnormalities	20–30% risk during critical period (very potent). Used for years before its teratogenic effects became obvious. Has caused the belief that all drugs have the potential to be a "new thalidomide." Back on market for treatment of oral lesions in HIV infection, Hansen's disease, tuberculosis, and multiple myeloma. STEPS is available online from the manufacturer, Celgene.

Abbreviations: ACE, angiotensin-converting enzyme; CNS, central nervous system; HIV, human immunodeficiency virus; STEPS, System for Thalidomide Education and Prescribing Safety.

U.S. Food and Drug Administration Pregnancy and Lactation Labeling System

In 1979, the FDA published a set of pregnancy risk categories for prescription and nonprescription drugs, including those with known teratogenic effects.[62] The letter-based categories A, B, C, D, and X were designed to indicate drug safety for the fetus. Although the FDA created the drug categories, the drug manufacturer determines the category to which a particular drug will be assigned.[6] Unfortunately, these FDA pregnancy categories oversimplified the complexity of what is known and what is not known about drug effects on human fetuses, in several different ways.[63]

In 2015, the FDA replaced the five letter categories with a more comprehensive narrative labeling system, the FDA Pregnancy/Lactation Labeling Rule (PLLR). The new labeling requirements address the safety of drugs in three settings: (1) pregnancy and intrapartum, (2) lactation, and (3) use of drugs by women and men with reproductive capability (**Table 9-7**).[62-64] Each section contains subsections that address risk information, clinical considerations, and background data.[62,64] In these sections, information from animal studies and human studies are listed separately.

Table 9-7 **Summary of the 2015 U.S. Food and Drug Administration Pregnancy/Lactation Labeling Rule**

General Information	Fetal Risk Summary	Clinical Considerations	Background Data
8.1 Pregnancy			
Inclusion of statement on background risk Contact information about scientifically, FDA acceptable pregnancy registries	Information from all relevant sources Risk conclusion regarding developmental abnormalities in humans and other relevant risks: whether it is likely that the drug increases the risk If increased risk is identified by human data, a narrative will be included If data demonstrate the drug is not systemically absorbed, a statement is included that maternal use is not expected to result in fetal exposure When the drug is systematically absorbed, statements of risk are divided based on type of data: human or animal, with findings from human studies presented first[a]	Statement on inadvertent exposure in early pregnancy: whether there is information or not Description of any known risks to the woman and fetus from the untreated disease Dosing adjustments during pregnancy Maternal adverse effects of the drug that are unique or increased during pregnancy Effects of dose, timing, and duration of treatment with the drug during pregnancy Potential neonatal complications and interventions needed If the drug may potentially be used on an intrapartum basis, even if not for an FDA-approved indication, information will be included about the effects on woman and the fetus/newborn, duration of labor and birth, risk of complications including the need for interventions, and long-term potential effects on the child	Include study type, dose, duration, timing, and results, including fetal abnormalities or other adverse effects Human data are presented first, including positive and negative effects, number of subjects, and study duration Animal study data include the species involved and recalculation of doses into human dose equivalents

8.2 Lactation			
General information is not mandated in the rule	Information from all relevant sources is included and identified Statement that the drug is compatible with breastfeeding if it does not have any effect on the quality or quantity of milk, if it is nondetectable in milk, or if no adverse effects are found in the child As applicable, a summary of the drug and its effects on milk production, its presence in milk, and any effects on the child[b]	Label will provide the following information, when available: • Strategies to minimize exposure to the child, including application of topical drugs to the nipple • Information about potential drug effects that could be useful to caregivers, such as monitoring for adverse effects, how to respond when they occur, and information about adjustments of maternal doses	Overview of the data that are the basis of the risk summary and clinical considerations Human data to be presented first
	Risks	**Clinical Considerations**	
8.3 Females and Males of Reproductive Potential			
	Risks are not specially noted as part of the information for females and males of reproductive potential, but the following address when it must be included and imply risks: • When pregnancy testing and/or contraception are required or recommended before, during, or after drug therapy • When there are human and/or animal data that suggest drug-associated fertility effects	Clinical considerations are not specifically noted as part of the information for females and males of reproductive potential, but the following list content of potential clinical importance must be included, in order: 1. Pregnancy testing 2. Contraception 3. Infertility	

Abbreviation: FDA, U.S. Food and Drug Administration.

[a] Several subsections follow, expanding upon the risk findings. In general, when human data are sufficient to assess the specific risk, standard language is required to indicate whether specific types of abnormalities are associated; when human data are available, but insufficient, the likelihood of the risk should be indicated; when animal data indicate the risk of a specific anomaly, a conclusion as to risk is required; and the last subsection includes a mandate for cross references to contraindications/warnings elsewhere on the label.

[b] Subsections that follow elucidate additional information to be contained about risk. In general, if no data are found of systemic absorption, a statement to that effect and assumption of no exposure to the nursling is made. If there is evidence of detection in milk, it must be so noted, as well as effects on child and milk production as well as the assay used, its limits, and a standard statement as to detection. When either the drug or active metabolites are detectable, the label also will include concentration in relationship to maternal/pediatric dose, an estimate of the maternal dose excreted, and an estimate of the consumption of a nursling based on average daily milk consumption. Another subsection must note pharmacologic and toxicologic information to identify known or predicted effects on the child, and pharmacokinetics related to amount and age will be included as they can affect the child. If only animal data are available, information regarding detection in animal milk and type of animal species is included. The last subsection includes a mandate for cross references to contraindications/warnings elsewhere on the label.

Based on U.S. Food and Drug Administration. Content and format of labeling for human prescription drug and biological products; requirements for pregnancy and lactation labeling. 79 FR 72063. *Federal Register.* 2014;79(9233 pt. 2):72064-72102. Available at: https://www.federalregister.gov/documents/2014/12/04/2014-28241/content-and-format-of-labeling-for-human-prescription-drug-and-biological-products-requirements-for#citation-3-p72066. Accessed September 11, 2016.[62]

The pregnancy and lactation section has two important additions. First, this section includes

a *Fetal Risk Summary* subsection for all drugs that are absorbed systemically. This text summarizes what is known about each medication's embryocidal, teratogenic, and/or fetotoxic risks. This section also has an initial subsection titled *Pregnancy Exposure Registry* that provides contact information for a pregnancy registry, if one exists. Pregnancy registries are observational studies of the effects of drugs on pregnant women. These studies are critically important for gathering postmarketing information about the effects of drugs.[63,65] Pregnancy exposure registries are created and monitored by the drug manufacturer. The FDA does not administer these groups, but it may recommend or even require establishment of such an observational study by the manufacturer. Midwives should contribute data about untoward drug effects identified in practice to these registries to facilitate early detection of adverse drug effects.[63]

Pharmaceuticals Commonly Used During Pregnancy

The drug classifications most commonly used during pregnancy include vitamins, analgesics, antimicrobials, and blood glucose regulators. The sections that follow present a brief review of these categories of pharmaceuticals.

Analgesics

The category of *nonsteroidal anti-inflammatory drugs* technically includes acetaminophen, ibuprofen, and aspirin. In general use, the term NSAIDs refers specifically to the family of drugs similar to ibuprofen (Advil), which is how the term is used in this chapter. Aspirin and NSAIDs have been implicated in disruption of the prostaglandin cascade involved in initiation of labor and, therefore, are linked to post-term pregnancy. NSAIDs can cause premature closure of the ductus arteriosus; indeed, they are administered to neonates for that specific therapeutic effect.[66] Aspirin has been associated with fetal vascular disruptions as well as competition with bilirubin for albumin binding sites. NSAIDs and aspirin should be avoided in pregnancy, especially in the first and third trimesters. An alternative agent without anti-inflammatory action is acetaminophen (Tylenol).[67] Acetaminophen is the analgesic of choice for minor pain during pregnancy.

Opiate analgesics have not been implicated as having teratogenic effects. Caution with their use in pregnancy is based on fear of addiction, not teratogenicity.[35]

Antimicrobials

Penicillins and *cephalosporins* are among the most commonly prescribed antimicrobials for treatment of various female reproductive infections. Few studies of cephalosporins have been conducted in the first trimester of pregnancy, but no adverse effects have been reported and cephalosporins, like penicillin, belong to the beta-lactam classification of antimicrobial agents. However, because the penicillins and erythromycin have been studied extensively and not found to be associated with teratogenic effects, many experts advocate using them as first-line therapies whenever indicated.[68]

Metronidazole (Flagyl) is a well-established antiprotozoal agent and the treatment of choice for *Trichomonas vaginalis* infection and bacterial vaginosis. No teratogenic effect secondary to use of metronidazole has been confirmed. However, its mechanism of action involves bacterial mutation and the drug is carcinogenic in rodents, so many providers consider use of metronidazole contraindicated in the first trimester. Although evidence does not support withholding the drug,[69] the conditions for which it is used generally are not life threatening. Because metronidazole is the most effective drug for the treatment of trichomoniasis, it should be prescribed for women who are symptomatic. Metronidazole is not recommended for treatment of asymptomatic trichomoniasis or bacterial vaginosis for the purpose of decreasing the risk of preterm labor.

Quinolones are rarely used in pregnancy because animal studies have demonstrated drug accumulation in the joints with subsequent damage.[70] These drugs may not pose the same risk

to humans but until more data are available, they are not recommended for use during pregnancy.

Sulfonamides are not teratogenic but require attention to timing. These agents are highly protein bound and can displace bilirubin from binding sites. The most commonly used agents in this category are a combination of sulfamethoxazole and trimethoprim (Bactrim, Septa). Administration of sulfonamides becomes problematic when these agents are administered near the time of birth, because the free bilirubin that is displaced will be in the newborn's circulation. The immature liver is unable to compensate for this effect, and kernicterus may result from these high plasma levels. Thus, sulfonamides should be avoided in the third trimester. Sulfasalazine (Azulfidine) appears to be an exception; this sulfonamide is used primarily for the treatment of ulcerative colitis and Crohn's disease and has a weak effect (if any) on bilirubin.

Tetracyclines are contraindicated in pregnancy because they concentrate in teeth. Children exposed in utero to tetracyclines may have yellowed teeth with poor enamel and vulnerability to caries, as well as potential problems with bone growth.[68]

Aminoglycosides include streptomycin, gentamycin (Garamycin), and kanamycin (Kantrex). All are ototoxic drugs that can cause permanent eighth cranial nerve damage. These agents are reserved for grave situations in which their benefits outweigh their risks.

Drugs Used to Treat Medical Conditions During Pregnancy

Although midwives care independently for women without medical or obstetric complications, they often consult and collaborate in the care of women who experience these complications. Thus, knowledge of drugs that have teratogenic or fetotoxic effects and that are used in the care of pregnant women who have medical complications is necessary.

Anticonvulsants

Antiepileptic agents pose a distinct challenge to the healthcare provider. Seizure disorders themselves may contribute to teratogenic outcomes. Several older antiepileptic drugs (AEDs) are teratogenic, although the absolute risk of congenital malformations following prenatal exposure is low.[71] Phenytoin (Dilantin) is associated with midline cardiac defects, hypospadias, neonatal coagulopathy, and neurobehavioral impairment. Carbamazepine (Tegretol), valproate (Depacon), and valproic acid (Depakene) can cause a dysmorphic pattern of minor anomalies, including neural tube defects and midline cardiac defects. Valproate and valproic acid are associated with intellectual impairment, and the congenital anomalies related to these drugs appear to be dose dependent.[72] Phenobarbital (Solfoton) has been associated with neurobehavioral impairment and newborn withdrawal syndrome. Newborns exposed in utero to phenytoin (Dilantin), carbamazepine (Tegretol), or phenobarbital (Luminal) are at risk of coagulation problems secondary to drug-induced vitamin K deficiency, and need prompt supplemental vitamin K after birth. Newer-generation antiepileptic drugs such as gabapentin (Neurontin) and lamotrigine (Lamictal) do not appear to have teratogenic effects.[73]

Many mechanisms have been proposed to explain the teratogenicity of anticonvulsant agents. Several of these drugs appear to have antifolate characteristics, and some authorities recommend that women on AEDs should be treated with 4 mg of folic acid both prior to conception and in early pregnancy.[74] Working closely with maternal–fetal specialists when caring for a woman with a seizure disorder is necessary, as treatment requires the choice of the least risky drug, at the lowest dose that controls seizures.

Antidepressants

Antidepressants represent another drug category used by many women during pregnancy. The most widely used class of medications to treat depression, bipolar disorder, and anxiety disorders are the selective serotonin reuptake inhibitors (SSRIs), which include agents such as fluoxetine (Prozac), paroxetine (Paxil), and sertraline (Zoloft).[75,76] Untreated depression is associated with several adverse pregnancy outcomes, including preterm birth, postpartum depression, and poor behavioral and mental health outcomes in children.[77] For this reason, treatment of depression during pregnancy is recommended.

Although knowledge about the teratogenic and fetotoxic effects of SSRIs and serotonin–norepinephrine reuptake inhibitors (SNRIs) is still being elucidated, midwives need to educate women on some effects linked to these drugs prior to initiating or continuing therapy during pregnancy. For example, paroxetine generally is avoided in pregnancy because of its potential for inducing fetal cardiac conditions.[76] Conversely, paroxetine and sertraline appear to have the best safety profile for women who are breastfeeding.[76,78] Midwives who choose to add these drugs to their individual formulary or who manage women who are taking these drugs must know the required screening and diagnostic criteria for depression and other related mental disorders, understand the side effects and adverse effects associated with these agents, appreciate the FDA's "black box" warnings for these drugs, and practice within a setting that provides access to psychiatric services. Because information related to these agents' use is rapidly increasing, professionals must keep abreast of any changes related to their recommended usage.

Antihypertensive Drugs

Antihypertensives are prescribed for women with chronic hypertension, a condition that may coexist with pregnancy. Chronic hypertension is defined as a systolic blood pressure of at least 140 mm Hg or a diastolic blood pressure of 90 mm Hg before pregnancy or in pregnant women prior to 20 weeks' gestation.[79,80]

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) are contraindicated in pregnancy secondary to an increased risk for teratogenic effects. Use of these drugs in the second and third trimesters is associated with low birth weight, oligohydramnios, and hypoplastic fetal lung development.[79] Atenolol (Tenormin) has been associated with intrauterine growth restriction (IUGR) secondary to reduced placental function. This problem has not been noted following use of the other beta blockers; thus, with the exception of atenolol, beta blockers are the first-line drugs used to treat chronic

hypertension in pregnancy. Labetalol (Trandate) is the most commonly used antihypertensive prescribed in the United States today.[79]

The other category of antihypertensive drugs that appear safe in pregnancy is the calcium-channel blockers, which include agents such as nifedipine (Procardia). Nifedipine is also used as a tocolytic.

Hypoglycemic Agents

Hypoglycemic agents are another drug category of interest to midwives, given that the number of women with type 2 diabetes is increasing in the United States and these women typically use oral hypoglycemic agents when not pregnant. Untreated diabetes is associated with multiple adverse outcomes for both the pregnant woman and her fetus, most of which occur secondary to hyperglycemia. Therefore, close glucose control is essential during pregnancy.[81,82] Traditionally, women with preexisting type 2 diabetes who are pregnant are treated as though they have gestational diabetes and managed with diet, exercise, and injectable insulin. In contrast to oral hypoglycemic agents, insulin is a large molecule and has difficulty crossing the placenta. Therefore, for many years it has been the drug of choice for management of diabetes mellitus during pregnancy.[83]

Glyburide (Micronase, DiaBeta) and metformin (Glucophage) have recently been the subject of research for treatment of diabetes during pregnancy.[84,85] Glyburide is extensively protein bound and does not cross the placenta in sufficient amounts to affect the fetus. Both glyburide and metformin appear to be safe and effective when used to treat diabetes in pregnancy, but neither is fully endorsed or established in clinical practice as yet.[84-86]

Thyroid Drugs

Thyroid drugs are frequently used during pregnancy. The most common cause of hyperthyroidism is Graves' disease, which occurs secondary to the development of autoantibodies. These autoantibodies bind to the thyroid-stimulating hormone (TSH) receptor on the thyroid gland, which results in excessive production of thyroid hormone. Untreated hyperthyroidism has been linked to spontaneous abortion. The most popular treatment for hyperthyroidism is radioiodine (RAI), which is radioactive iodine that concentrates in the thyroid and destroys thyroid tissue. Radioiodine is contraindicated in pregnancy because it can cross the placenta and destroy the fetal thyroid. Traditionally, propylthiouracil (PTU) has been the drug of choice for hyperthyroidism in pregnancy, as methimazole (Tapazole) has been linked to aplasia cutis in the newborn. More recently, this neonatal association has been called into question.[87] Moreover, PTU may result in fetal hypothyroidism when the drug is used in high doses, so close monitoring is required when it is prescribed to a pregnant woman. Women who are taking PTU should be seen by an obstetrician to establish a plan for ultrasound monitoring to assess the fetal thyroid.

The most common etiology of hypothyroidism is Hashimoto's disease, also known as autoimmune thyroiditis. Untreated hypothyroidism can cause fetal mental retardation, preeclampsia, stillbirth, and other adverse obstetric outcomes.[88,89] Hypothyroidism is treated

with levothyroxine (Synthroid), which is synthetic thyroid hormone. Levothyroxine has no adverse effects on the fetus, but the maternal dose may need to be increased by as much as 45% during pregnancy; to confirm that the appropriate dose is being administered, TSH and free thyroxine (T_4) levels are typically assessed once each trimester. The goal is to maintain a euthyroid state during pregnancy.

Many practices are adopting universal screening for thyroid disorders in pregnancy, although questions have been raised about whether such screening makes a difference in perinatal outcomes. It has been associated with increased use of thyroid drugs.[89]

Conclusion

Pharmaceutical agents are part of modern life. Pharmacology is an essential area in women's health care, and one that continues to expand and challenge providers and women alike. Like any other intervention in a midwife's repertoire, drugs and herbs should be used appropriately and monitored for both therapeutic and adverse effects. Various publications, both in print and in electronic media, have emerged to help clinicians access current pharmacologic information. Prominent among these innovations are applications for smartphones that can download information about all FDA-approved medications, including doses, contraindications, cautions, interactions, adverse reactions, cost, and pregnancy categorization. Of course, such resources are useful only when they are updated and consulted regularly.

A single agent is rarely a magic cure for a woman independent of the larger context of her health, social, educational, economic, and other factors. No professional should simply prescribe or recommend any pharmaceutical product without taking such considerations into account. Viewing the woman and her needs holistically is one of the skills of a great midwife. As an ancient Chinese saying states, "It is easy to get a thousand prescriptions, but hard to get one single remedy."

Resources

Textbooks with Information on the Care of Woman		
Briggs GG, Freeman RK, Towers CV, Forinash AB. *Drugs in Pregnancy and Lactation: A Reference Guide to Fetal a* 11th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2017.		
Brucker MC, King TL. *Pharmacology for Women's Health.* 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017.		
Organization	**Description**	**Webpage**
Apps for Drug Prescribing		
Epocrates	Mobile app to look up information on drugs, including drug interactions, news, articles, and diagnosis codes. Basic app is free.	http://www.epocrates.com
Drugs.com	Mobile app similar to Epocrates, including pill identifier	https://www.drugs.com
Resources for Prescribing Antibiotics and Other Pharmaceuticals		
Association for Professionals in Infection Control and Epidemiology (APIC)	Resources for antibiotic stewardship programs and guidelines	https://apic.org/Professional-Practice/Practice-Resources/Antimicrobial-Ste
Indiana University School of Medicine	Drug interaction table of substrates, inhibitors, and inducers with links from the drug name to a PubMed list of citations that is continuously updated	http://medicine.iupui.edu/flockhart/table.htm
Clinical Pharmacogenetics Implementation Consortium (CPIC)	Shared project of PharmGKB and the Pharmacogenomics Research Institute, which is funded by the National Institutes of Health. The CPIC publishes peer-reviewed dosing guidelines for drugs.	https://www.pharmgkb.org/guidelines#
U.S. Food and Drug Administration (FDA)	List of drugs that have genomic information on the drug label. Some of these drugs require genomic testing prior to initiating therapy; others	https://www.fda.gov/drugs/scienceresearch/researchareas/pharmacogeneti

	include genomic information in the precautions, warnings, or drug pharmacology.	
	FDA Adverse Event Reporting System (FAERS) is a computerized database that contains postmarketing reports of adverse events and medication errors submitted to the FDA. The FAERS is part of the FDA's safety surveillance program for all approved drug and therapeutic biologic products. The goal of FAERS is to improve public health through effective collection and analysis of safety reports.	https://www.accessdata.fda.gov/scripts/medwatch/index.cfm?action=report
	Drug manufacturers are required by regulation to submit all reports of adverse drug events to FAERS. Healthcare providers and consumers may voluntarily submit reports through the MedWatch website.	
	Information about drug alerts, drug safety communications, and drug recalls can be found on the FDA website.	https://www.fda.gov/Drugs/DrugSafety/default.htm
Resources for Drugs in Pregnancy		
Centers for Disease Control and Prevention (CDC)	"Treating for Two: Safer Medication Use in Pregnancy Initiative." This effort aims to prevent birth defects and improve maternal health by	http://www.cdc.gov/ncbddd/birthdefects/documents/ncbddd_birth-defects_medicationuseonepager_cdcrole.pdf

	providing clinicians with the necessary tools and information on medication use in pregnancy to make informed clinical decisions regarding safe medication use for both pregnant and reproductive-age women. The initiative includes a formal review process and evaluation of the evidence regarding medication-associated embryonic, fetal, perinatal risks (i.e., preterm birth, fetal death, structural birth defects, poor fetal growth, and severe adverse maternal events), and other adverse outcomes (e.g., developmental disabilities, neurocognitive effects, behavioral effects). Participants include experts from academia, professional organizations, and federal agencies.	
Motherisk	Canadian-based clinical research and teaching program based at the University of Toronto, Ontario, which provides research, counseling, and recommendations to the public and to health providers on medication safety, chemicals, and maternal disease, and health topics in pregnancy and lactation. This free	http://www.motherisk.org

	resource includes helplines, updates, articles, and more.	
MotherToBaby (Organization of Teratology Information Specialists)	MotherToBaby is a nonprofit, evidence-based organization that provides information for healthcare professionals, mothers, and general public on medication exposures during pregnancy and lactation. 1-866-626-6847	www.mothertobaby.org
Teratogen Information System (TERIS)	Fee-based subscription online database for providers and healthcare professionals. Summaries are based on published clinical and experimental data. Housed in the University of Washington Department of Pediatrics.	http://depts.washington.edu/terisdb/
Toxnet and Lactnet	Online databases supported by the U.S. federal government. Toxnet provides information on a wide variety of agents; Lactnet is specific for drugs and lactation.	https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm

References

1. Osborne K. Regulation of prescriptive authority for certified nurse-midwives and certified midwives: a 2015 national overview. *J Midwifery Womens Health*. 2016;60(3):519-533.
2. Aikin KJ, Sullivan HW, Dolina S, Lynch M, Squiers LB. Direct-to-consumer promotion of prescription drugs on mobile devices: content analysis. *J Med Internet Res*. 2017;19(7):e225.
3. Frosch DL, Grande D, Tarn DM, Kravitz RL. A decade of controversy: balancing policy with evidence in the regulation of prescription drug advertising. *Am J Public Health*. 2010;100(1):24-32.
4. Quackenbush R, Hackley B, Dixon J. Screening for pesticide exposure: a case study. *J Midwifery Womens Health*. 2006;51(1):3-11.
5. U.S. Food and Drug Administration. New drug approval process. Available at: https://www.drugs.com/fda-approval-process.html. Accessed August 17, 2017.
6. Parekh A, Fadiran EO, Uhl K, Throckmorton DC. Adverse effects in women: implications for drug development and regulatory policies. *Expert Rev Clin Pharmacol*. 2011;4(4):453-466.
7. Allen R, O'Brien BM. Uses of misoprostol in obstetrics and gynecology. *Rev Obstet Gynecol*. 2009;2(3):159-168.
8. U.S. Department of Justice, Drug and Enforcement Administration, Office of Diversion Control. Mid-level practitioners authorization by state. Available at: https://www.deadiversion.usdoj.gov/drugreg/practioners/. Accessed August 16, 2017.
9. Hoffman FA. Regulation of dietary supplements in the United States: understanding the Dietary Supplement Health and Education Act. *Clin Obstet Gynecol*. 2001;44(4):780-788.
10. Marty AT. The complete German Commission E Monographs. *JAMA*. 1999;281(19):1952.
11. Blumenthal M, Busse W, Goldberg A, et al., eds.; Klein S, Rister R, trans. *Complete German Commission E Monographs: Therapeutic Guide to Herbal Medicines*. Newton, MA: Integrative Medicine Communications; 1998.
12. Holford NHG. Pharmacokinetics and pharmacodynamics. In: Katzung BG, Trevor AJ, eds. *Basic and Clinical Pharmacology*. 13th ed. New York, NY: McGraw-Hill; 2015: 41-55.
13. Ye M, Nagar S, Korzekwa K. A physiologically based pharmacokinetic model to predict the pharmacokinetics of highly protein-bound drugs and impact of errors in plasma protein binding. *Biopharmaceut Drug Disp*. 2016;37(3):123-141.
14. Krau SD. Cytochrome p450, Part 1: what nurses really need to know. *Nurs Clin North Am*. 2013;48:671-680.
15. Wilkinson G. Drug metabolism and variability among patients in drug response. *N Engl J Med*.2005;352(21):2211-2221.
16. Evans WE, McLeod HL. Pharmacogenomics: drug disposition, drug targets and side effects. *N Engl J Med*. 2003;348(6):538-539.
17. Samer CF, Lorenzini KI, Rollason V, Daali Y, Desmeules JA. Applications of CYP450 testing in the clinical setting. *Mol Diagn Ther*. 2013;17(3):165-184.
18. Belle DJ, Singh H. Genetic factors in drug metabolism. *Am Fam Physician*. 2008;77(11):1553-1560.
19. Weinshilboum R. Inheritance and drug response. *N Engl J Med*. 2003;348(6):529-537.
20. Ortega VE, Meyers DA. Pharmacogenetics: implications of race and ethnicity on defining genetic profiles for personalized medicine. Mechanisms of allergic diseases. *J Allergy Clin Immunol*. 2014;133:16-26.
21. Niesters M, Dahan A, Kest B, et al. Do sex differences exist in opioid analgesia? A systematic review and meta-analysis of human experimental and clinical studies. *Pain*. 2010;151(1):61-68.
22. Tharpe N. Adverse drug reactions in women's health care. *J Midwifery Womens Health*. 2011;56(3):205-214.
23. Tache SV, Sonnichsen A, Ashcrofft DM. Prevalence of adverse drug events in ambulatory care: a systematic review. *Ann Pharmacother*. 2011;45(7-8):977-989.
24. Brüggemann RJM, Alffenaar JW, Blijlevens NMA, et al. Clinical relevance of the pharmacokinetic interactions with azol antifungal drugs and other coadministered agents. *Rev Anti-infect Agents*. 2009;48:1441-1446.
25. Munir M, Enany N, Zhang JM. Nonopioid analgesics. *Med Clin North Am*. 2007;91:97-111.
26. Voon P. Karamouzian M, Kerr T. Chronic pain and opioid misuse: a review of reviews. *Subst Abuse Treat Prev Policy*. 2017;12(1):36-44.
27. van der Bijl P, van der Bijl P Jr. Efficacy, safety and potential clinical roles of the COX-2-specific inhibitors. *Int J Immunopathol Pharmacol*. 2003;16(2 suppl):17-22.
28. Wolfe MM, Lichtenstein DR, Singh G. Gastrointestinal toxicity of nonsteroidal antiinflammatory drugs. *N Engl J Med*. 1999;340:1888-1900.
29. Khan MI, Walsh D, Brito-Dellan N. Opioid and adjuvant analgesics: compared and contrasted. *Am J Hosp Palliat Care*. 2011;28(5):378-383.
30. Craft RM. Sex differences in opioid analgesia: "from mouse to man." *Clin J Pain*. 2003;19:175-186.
31. Sandner-Kiesling A, Eisenach JC. Estrogen reduces efficacy of mu but not kappa-opioid agonist inhibition in response to uterine cervical distention. *Anesthesiology*. 2000;96:375-380.
32. Centers for Disease Control and Prevention. Prescription opioid overdose data. December 16, 2016. Available at: https://www.cdc.gov/drugoverdose/data/overdose.html. Accessed August 1, 2017.
33. Dowell D, Haegerich TM, Chou R. CDC guideline for prescribing opioids for chronic pain—United States, 2016. *MMWR*

Recomm Rep. 2016;65(1):1-49.
34. Manchikanti L, Kaye AM, Knezevic NN, et al. Responsible, safe, and effective prescription of opioids for chronic non-cancer pain: American Society of Interventional Pain Physicians (ASIPP) guidelines. *Pain Physician*. 2017;20(2S):S3-S92.
35. Roper V, Cox KJ. Opioid disorder in pregnancy. *J Midwifery Womens Health*. 2017;62:329-340.
36. Roe V. Antibiotic resistance: a guide for effective prescribing in women's health. *J Midwifery Womens Health*. 2008;53:216-226.
37. Arnold SR, Straus SE. Interventions to improve antibiotic prescribing practices in ambulatory care. *Cochrane Database Syst Rev*. 2005;4:CD003539. doi:10.1002/14651858.CD003539.pub2.
38. Waller DG. Rational prescribing: the principles of drug selection and assessment of efficacy. *Clin Med*. 2005:1(5):26-28.
39. Sanchez GV, Fleming-Dutra KE, Roberts RM, Hicks LA. Core elements of outpatient antibiotic stewardship. *MMWR Recomm Rep*. 2016;65(6):1-12.
40. Harkins CP, Pichon B, Doumith M, et al. Methicillin-resistant *Staphylococcus aureus* emerged long before the introduction of methicillin into clinical practice. *Genome Biol*. 2017;18:130. doi:10.1186/s13059-017-1252-9.
41. Doron S, Davidson LE. Antimicrobial stewardship. *Mayo Clin Proc*. 2011;86(11):1113-1123.
42. Drawz SM, Bonomo RA. Three decades of beta-lactamase inhibitors. *Clin Microbiol Rev*. 2010;23(1):160-201.
43. Correia S, Poeta P, Hébraud M, Capelo JL, Igrejas G. Mechanisms of quinolone action and resistance: where do we stand? *J Med Microbiol*. 2017;66(5):551-559.
44. Dellit TH, Owens RC, McGowan Jr JE, et al. Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America guidelines for developing an institutional program to enhance antimicrobial stewardship. *Clin Infect Dis*. 2007;44:159e77.
45. Ramasethu J, Kawakita T. Antibiotic stewardship in perinatal and neonatal care. *Semin Fetal Neonatal Med*. 2017:22(5):278-283.
46. Dryden M, Johnson APA, Ashiru-Oredope D, Sharland M. Using antibiotics responsibly: right drug, right time, right dose, right duration. *J Antimicrob Chemother*. 2011;66(11):2441-2443.
47. Simons FE, Simons KJ. Histamine and H(1)-anti-histamines: celebrating a century of progress. *J Allergy Clin Immunol*. 2011;128(6):1139-1150.
48. Gedeon C, Koren G. Designing pregnancy centered medications: drugs which do not cross the human placenta. *Placenta*. 2006;27:861-868.
49. Tasnif Y, Morado J, Herbert MF. Pregnancy-related pharmacokinetic changes. *Clin Pharm Therap*. 2016;100(1):53-62.
50. Frederiksen MC. Physiologic changes in pregnancy and their effect on drug disposition. *Semin Perinatol*. 2001;25:120-123.
51. Chamberlain A, White S, Bawdon R, Thomas S, Larsen B. Pharmacokinetics of ampicillin and sulbactam in pregnancy. *Am J Obstet Gynecol*. 1993;168(2):667-673.
52. Lassiter NT, Manns-James LE. Pregnancy. In: Brucker MC, King TL. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:1025-1064.
53. Syme MR, Paxton JW, Keelan JA. Drug transfer and metabolism by the human placenta. *Clin Pharmacokin*. 2004;43(8):487-514.
54. Zhao Y, Hebert MF, Venkataramanan R. Basic obstetric pharmacology. *Semin Perinatol*. 2014;38(8):475-486.
55. Mitchell AA, Gilboa SM, Werler MM, et al.; National Birth Defects Prevention Study. Medication use during pregnancy, with particular focus on prescription drugs: 1976–2008. *Am J Obstet Gynecol*. 2011;205:51.e1-51.e8.
56. Shroff S, McNeil M, Borrero S. An innovative framework to improve teratogenic medication risk counseling. *J Midwifery Womens Health*. 2017;62:353-357.
57. Hansen WF, Yankowitz J. Pharmacologic therapy for medical disorders during pregnancy. *Clin Obstet Gynecol*. 2002;45(1):13-52.
58. Buhimschi CS, Weiner CP. Medications in pregnancy and lactation: part 1. Teratology [erratum, *Obstet Gynecol*. 2009;113(6):1377]. *Obstet Gynecol*. 2009;113(1):166-188. Review.
59. Mitchell AA. Research challenges for drug-induced birth defects. *Clin Pharmacol Ther*. 2016;100(1):26-28.
60. Koren G. Fetal risks of maternal pharmacotherapy: identifying signals. *Handb Exp Pharmacol*. 2011;205:285-294.
61. Moore KL, Persaud TVN. *The Developing Human: Clinically Oriented Embryology*. 10th ed. Philadelphia, PA: W. B. Saunders; 2015.
62. U.S. Food and Drug Administration. Content and format of labeling for human prescription drug and biological products; requirements for pregnancy and lactation labeling. 79 FR 72063. *Federal Register*. 2014;79(9233 pt. 2):72064-72102. Available at: https://www.federalregister.gov/documents/2014/12/04/2014-28241/content-and-format-of-labeling-for-human-prescription-drug-and-biological-products-requirements-for#citation-3-p72066. Accessed September 11, 2016.
63. Brucker MC, King TL. The 2015 US Food and Drug Administration Pregnancy Labeling Rule. *J Midwifery Womens Health* 2017;62(3):308-316.
64. U.S. Food and Drug Administration. Pregnancy and lactation labeling final rule. Updated March 23, 2016. Available at:

http://www.fda.gov/Drugs/DevelopmentApprovalProcess/DevelopmentResources/Labeling/ucm093307.htm. Accessed September 12, 2016.
65. Sinclair SM, Miller RK, Chambers C, Cooper EM. Medication safety during pregnancy: improving evidence-based practice. *J Midwifery Womens Health* 2016;61(1):52-67.
66. Buhimschi CS, Weiner CP. Medications in pregnancy and lactation: part 2. Drugs with minimal or unknown human teratogenic effect. *Obstet Gynecol.* 2009;113(2 pt 1):417-432.
67. Ostensen ME, Skomsvoll JF. Anti-inflammatory pharmacotherapy during pregnancy. *Expert Opin Pharmacother*. 2004;5(3):571-580.
68. Bookstaver PB, Bland CM, Griffin B, Stover KR, Eiland LS, McLaughlin M. A review of antibiotic use in pregnancy. *Pharmacotherapy.* 2015;35(11):1052-1062.
69. Koss CA, Baras DC, Lane SD, et al. Investigation of metronidazole use during pregnancy and adverse birth outcomes. *Antimicrob Agents Chemother.* 2012;56(9):4800-4805.
70. Ozyüncü O, Beksac MH, Nemutlu E, Katlan D, Kir S. Maternal blood and amniotic fluid levels of moxifloxacin, levofloxacin, and cefixime. *J Obstet Gynecol Res.* 2010;36(3):484-487.
71. Lowe SA. Anticonvulsants and drugs for neurological disease. *Best Pract Res Clin Obstet Gynecol.* 2001;15(6):863-876.
72. Banach R, Boskovic R, Einarson T, Koren G. Long-term developmental outcome of children of women with epilepsy, unexposed or exposed prenatally to antiepileptic drugs: a meta-analysis of cohort studies. *Drug Safety.* 2010;33(1):73-79.
73. Molgaard-Nielsen D, Hviid A. Newer-generation antiepileptic drugs and the risk of major birth defects. *JAMA.* 2011;305(19):1996-2002.
74. Hernandez-Diaz S, Werler M, Walker A, Mitchell A. Folic acid antagonists during pregnancy and risk of birth defects. *N Engl J Med*. 2000;343(22):1608-1614.
75. Hackley B. Antidepressant medication use in pregnancy. *J Midwifery Womens Health*. 2010;55:90-100.
76. Latendresse G, Elmore C, Deneris A. Selective serotonin reuptake inhibitors as first-line antidepressant therapy for perinatal depression. *J Midwifery Womens Health*. 2010;62:317-328.
77. Yonkers KA, Wisner KL, Stewart DE, et al. The management of depression during pregnancy: a report from the American Psychiatric Association and the American College of Obstetricians and Gynecologists. *Obstet Gynecol.* 2009;114(3):703-713.
78. Orsolini L, Bellantuono C. Serotonin reuptake inhibitors and breastfeeding: a systematic review. *Hun Psychopharmacol.* 2015;30(1):4-20.
79. Seely EW, Ecker J. Chronic hypertension in pregnancy. *N Engl J Med*. 2011;365:439-446.
80. American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy: report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122(5):1122-1131.
81. Reedy NJ. Addressing the epidemic: pharmacotherapeutic management of diabetes in women. *J Midwifery Womens Health*. 2002;47(6):471-486.
82. Langer O. Management of gestational diabetes: pharmacologic treatment options and glycemic control. *Endocrin Metab Clin North Am.* 2006;35:53-78.
83. Refuerzo JS. Oral hypoglycemic agents in pregnancy. *Obstet Clin North Am.* 2011;38:227-234.
84. Kimber-Trojnar Z., Marciniak B, Patro-Malysza J, et al. Is glyburide safe in pregnancy? *Curr Pharm Biotechnol.* 2014;15(1):100-112.
85. Langer O, Conway D, Berkus M, Xenakis EM, Gonzales O. A comparison of glyburide and insulin in women with gestation diabetes mellitus. *N Engl J Med*. 2000;343(16):1134-1138.
86. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 60: pregestational diabetes mellitus. *Obstet Gynecol.* 2005;105:675-685.
87. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 37: thyroid disease in pregnancy. *Obstet Gynecol.* 2002;100:387-396.
88. Negro R, Mestman JH. Thyroid disease in pregnancy. *Best Pract Res Clin Endocrinol Metab*. 2011;25(6):927-943.
89. Spencer L, Bubner T, Bain E, Middleton P. Screening and subsequent management for thyroid dysfunction pre-pregnancy and during pregnancy for improving maternal and infant health. *Cochrane Database Syst Rev.* 2015;9:CD011263. doi:10.1002/14651858.CD011263.pub2.

PART III

Gynecology

PATRICIA AIKINS MURPHY AND FRANCES E. LIKIS

Patricia Aikins Murphy and Frances E. Likis

Today, the average life expectancy at birth for a woman who resides in the United States is 81.2 years.[1] Within this lifespan, the average woman wants two children, and thus spends about 3 years pregnant, postpartum, or trying to become pregnant. She will spend approximately 30 years—the vast majority of her reproductive life—trying to avoid pregnancy.[2] Moreover, given modern life expectancy, this woman will enjoy 3 or more decades of life after her reproductive years have ended.

Ideally, most of this woman's life will be spent in good health, where health, as defined by the World Health Organization (WHO), is a "state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity."[3] Ideally, she will also enjoy reproductive health at all stages of her life, defined by WHO as the ability "*to have a responsible, satisfying, and safe sex life and . . . the capability to reproduce; and the freedom to decide if, when, and how often to do so.*"[4]

Making these hopes for good health a reality requires health promotion, disease prevention, health education, and empowering women to make the choices that are meaningful to them and the choices that will optimize their health.

Fortunately, these are skills in which midwives excel. Add the recognition that life stages, normal physiology, and developmental processes are not diseases; that continuity of care is a value to be promoted; that informed choice, shared decision making, and the right to self-determination are important to health; and that evidence-based decision making should underlie the health care of women, and we have several of the hallmarks of midwifery important for providing gynecologic, sexual, and reproductive health care.[5] While midwifery is often associated with pregnancy and birth, women have healthcare needs at all stages of their lives that are not related to pregnancy. Thus, gynecologic, sexual, and reproductive care are, and

should be, essential aspects of midwifery practice that are well established in the American College of Nurse-Midwives (ACNM) Core Competencies,[5] where specific knowledge and skills of midwifery responsibilities beyond perinatal care are detailed in several sections.

Caring for women in the area of gynecologic, sexual, and reproductive health gives midwives the opportunity to truly be with women for a lifetime, from adolescence through old age. Most women spend only a few years of their lives needing maternity care services, but they need other healthcare services that midwives can provide for many more years. Although the face of U.S. health care can change with political imperatives and economic demands, the Health Resources and Services Administration has published Women's Preventive Services guidelines, the most recent iteration of which was released in 2016.[6] Under the prevailing insurance guidelines, women can currently receive annual well-woman visits, human papillomavirus testing, counseling for sexually transmitted infections, HIV screening, and contraceptive methods and counseling without paying a copayment, coinsurance, or a deductible (note that there are some exemptions for organizations that object to contraceptive counseling or provision).[6] Midwives must be poised and prepared to provide these services. The knowledge found in the chapters in this section provides the foundation for being "with women for a lifetime."[7]

References

1. National Center for Health Statistics. *Health, United States, 2016: With Chartbook on Long-Term Trends in Health*. Hyattsville, MD: National Center for Health Statistics; 2017. Available at: https://www.cdc.gov/nchs/data/hus/hus16.pdf. Accessed November 16, 2017.
2. Guttmacher Institute. *Unintended Pregnancy in the United States: September 2016 Fact Sheet*. New York, NY: Guttmacher Institute; 2017. Available at: https://www.guttmacher.org/fact-sheet/unintended-pregnancy-united-states. Accessed November 16, 2017.
3. World Health Organization. *WHO Definition of Health*. Geneva, Switzerland: World Health Organization; 1948. Available at: http://www.who.int/suggestions/faq/en/. Accessed November 16, 2017.
4. World Health Organization. Reproductive health. Available at: http://www.who.int/topics/reproductive_health/en/. Accessed November 16, 2017.
5. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
6. Health Resources and Services Administration. Women's preventive services guidelines. Available at: https://www.hrsa.gov/womens-guidelines/index.html. Accessed November 16, 2017.
7. Williams DR. Unveiling an evidence-based image for ACNM: "with women for a lifetime." *J Midwifery Womens Health*. 2001;46(3):vi.

CHAPTER

10

Anatomy and Physiology of the Female Reproductive System

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Introduction

Midwifery practice begins with an understanding of reproductive anatomy and physiology. Four chapters in this text specifically focus on the anatomy and physiology underpinning clinical practice. This chapter provides a general overview of the anatomy of the female reproductive system, including the breast, and describes basic reproductive physiology, including menstrual physiology. The *Anatomy and Physiology of Pregnancy* chapter reviews maternal anatomic and physiologic adaptations to pregnancy and fetal and placental development and function. The *Anatomy and Physiology During Labor and Birth* chapter focuses on the processes involved in labor and birth. The *Anatomy and Physiology of Postpartum* chapter describes the physiology of the puerperium. Other chapters also include information on important physiologic processes. The physiology of lactation, for example, is described in the *Breastfeeding and the Mother–Newborn Dyad* chapter, while the transition of the newborn to extrauterine life is covered in the *Anatomy and Physiology of the Newborn* chapter.

The Breast

The breast undergoes dramatic changes in size, shape, and function during a woman's lifetime. Growth, differentiation, and lactogenesis are the result of complex hormonal stimuli. Breast development starts during embryonic life. Full development of breast tissue takes place during pregnancy and lactation.

Breast Anatomy

The adult female breast extends vertically from approximately the second rib to the sixth rib, and horizontally from the edge of the sternum to the mid-axillary line with an extension of tissue into the axilla known as the tail of Spence (**Figure 10-1**).[1] Breast tissue is composed of skin, subcutaneous tissue, and breast tissue. The nipple, which is located slightly below the center of each breast, is surrounded by a circular area of pigmented skin known as the areola. The shape of the breast is maintained primarily by suspensory ligaments, known as Cooper's ligaments, that connect the dermis of the breast to the deep pectoral fascia, which overlays the pectoralis major and serratus anterior muscles of the chest.

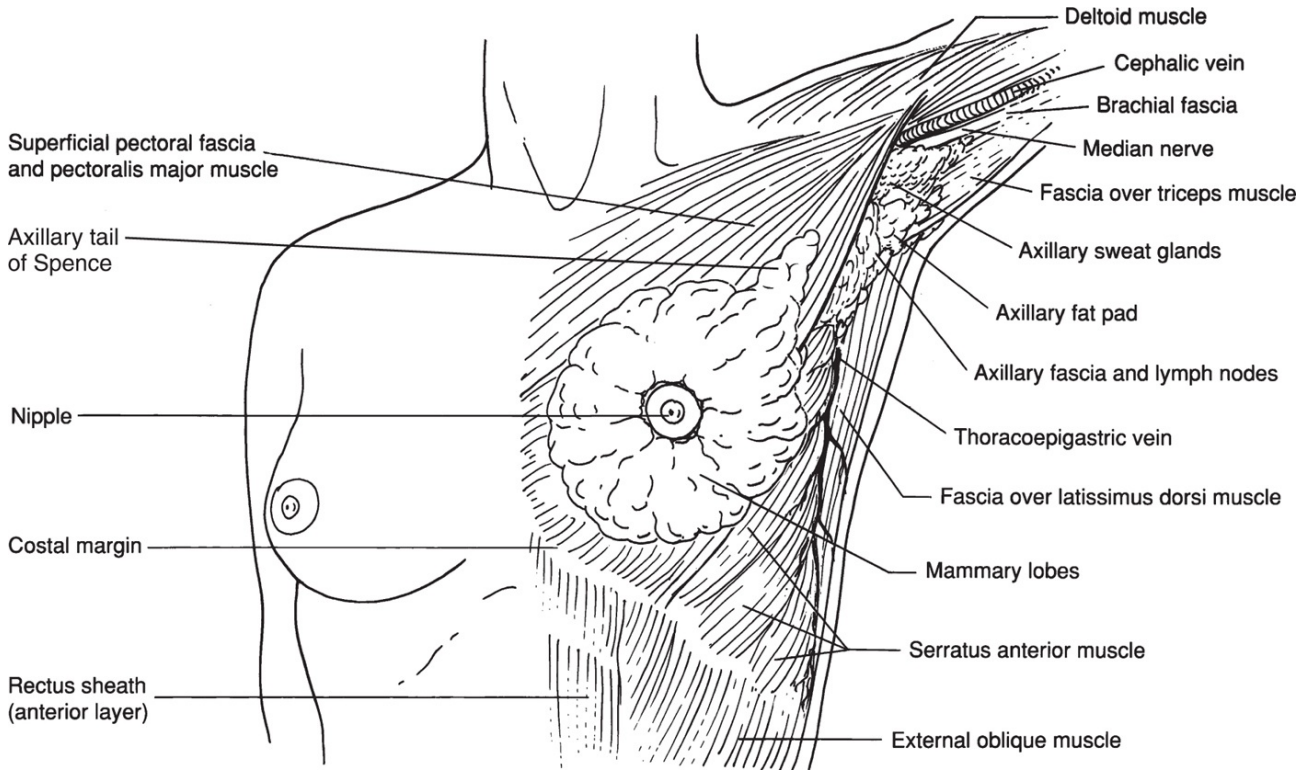


Figure 10-1 Anterior pectoral dissection.

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The skin of the breast includes the nipple and the areola, the area that surrounds the nipple. The nipple's elastic tissue contains smooth muscle fibers and is innervated by both sensory and autonomic nerve endings. The nipple becomes smaller and firmer in response to cold, touch, and sexual stimulation under the influence of oxytocin. The darker circular areola surrounding the nipple is somewhat elastic and varies in diameter.

The nipple has approximately 5 to 9 mammary ductal orifices and is abundantly innervated with sensory nerve endings. The nipple/areola complex has many sebaceous and apocrine sweat glands. For example, the Montgomery glands are large sebaceous glands located under the areola. The openings to these glands form round elevations just under the skin of the areola, called Montgomery tubercles. The Montgomery glands become enlarged in pregnancy; they produce secretions that keep the nipple/areola complex lubricated and may assist in the initiation of lactation by providing olfactory cues to the newborn. A more detailed description of lactogenesis can be found in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

The breast tissue is composed of epithelial (glandular or secretory) tissue, between which is interspersed stromal (adipose and connective) tissue. These two types of tissue are present in approximately equal amounts in the breasts of women who are not pregnant or lactating.[2] During pregnancy and lactation, however, the glandular tissue proliferates and becomes the predominant breast tissue.[3] Postmenopausally, the glandular tissue tends to shrink and is generally replaced by fat, resulting in changes in shape and size.

The glandular tissue in each breast of a reproductive-age woman is organized into several lobes that are made up of clusters of 10 to 100 alveoli, referred to as lobules (**Figure 10-2**). Traditionally, the lobes have been depicted as separate, distinct entities that terminate in a lactiferous sinus just under the nipple.[2] More recently, some controversy has arisen regarding the structure of the breast ductal system. First, the lactiferous sinus may not be an anatomic structure per se, but rather a transient dilation of the terminal duct that occurs during lactation.[2] A few studies have found anastomoses between lobes, while others have not found anastomoses; the majority, however, have shown that each ductal system is independent.[4] Because breast cancer forms in ductal systems, a thorough understanding of this anatomy has important clinical implications.

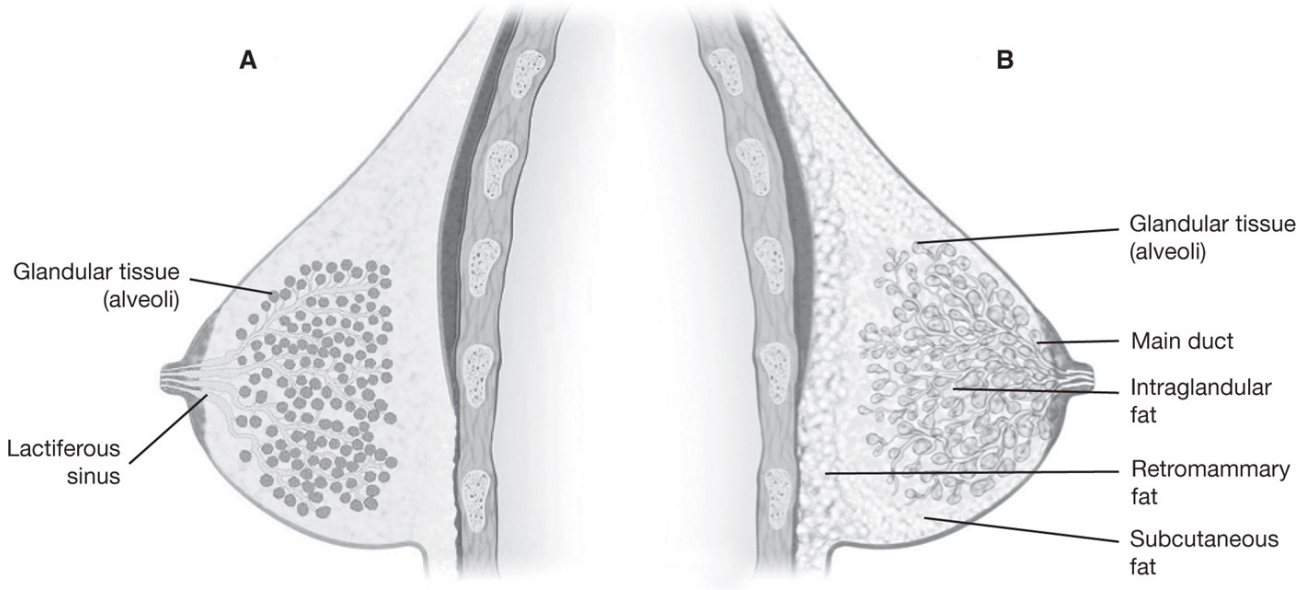


Figure 10-2 **A.** Traditional schematic diagram of the anatomy of the breast. The main milk ducts below the nipple are depicted as dilated portions or lactiferous sinuses and the glandular tissue is deeper within the breast. **B.** Schematic diagram of the ductal anatomy of the breast based on the findings of Ramsay et al.[3] Milk ducts are small and branch a short distance from the base of the nipple. The ductal

system is erratic and glandular tissue is situated directly beneath the nipple.

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The alveoli are dilated sac-like structures composed of two layers of cells. The inner luminal cells are responsible for synthesis and secretion of breast milk. They are surrounded by myoepithelial cells that eject milk during lactation (**Figure 10-2**). During pregnancy, the alveoli gain the capacity to produce and excrete milk. These differentiated alveoli are referred to as acini. The secretory cells in these acinar units are stimulated by prolactin to secrete milk into the lumen of the alveolus, whereas the myoepithelial cells are stimulated by oxytocin to contract, which compresses the alveolar sacs so that milk is ejected into the milk ductules. The ductules are connected to larger lactiferous ducts, which merge to form a smaller number of ducts leading to openings at the nipple, through which milk exits the breast. The exact number of these openings is not known, but has been reported to vary from 4 to 18; however, the most recent evidence seems to suggest that there are, on average, 5 to 9 patent ducts in each nipple of a lactating woman.[3]

Blood is supplied to the breast primarily by the mammary artery and by the lateral thoracic arteries. Venous flow from the breast drains into the internal thoracic, axillary, and cephalic veins. The lymphatic vessels of the breast serve an important function during lactation by draining milk molecules that are too large to move into blood vessels.

Lymphatic flow to and from the breast is significant because it determines the location of metastasis of cancer cells from the breast to other parts of the body. The majority of breast lymph flows to the axillary lymph nodes, which is, therefore, the main route of cancer metastasis from the breast. The intermammary lymph nodes are the other main group of lymph nodes that drain the breast, but they receive only a small fraction of the lymph flow from the breast.

The breast is innervated by the lateral and anterior cutaneous branches of the second to the sixth intercostal nerves and by the supraclavicular nerves. The nipple and areola are innervated primarily by the cutaneous branch of the fourth intercostal nerve.

Breast Development

Human breast development begins during the embryologic period and is believed to originate with the appearance of the primitive milk streaks bilaterally in the embryo from the axilla to the groin. This ridge regresses except for the region that eventually becomes the mammary gland. On occasion, additional areas of this ridge may not regress; these areas then develop into accessory nipples and sometimes even into glandular mammary tissue capable of milk production and excretion. Such auxiliary nipples may be noted on a physical examination, but have no clinical significance.

At birth, newborns have an opening of a primitive ductal system to the surface, a protruding nipple, and a circular area of skin that has proliferated into the areola. Varying degrees of glandular breast tissue development occur during the fetal period. At birth, the structures present can range from simple blunt-ended tubular structures to well-developed branching ductal systems with lobular–alveolar structures.[5] Under the influence of maternal and neonatal pro-lactation hormones, these structures are capable of producing milk shortly after birth. In

some cases, this milk is excreted and exits via the neonate's nipple (sometimes referred to as "witch's milk"). Once the maternal hormones are removed, however, the infant breast tissue undergoes a period of involution. By the time the child reaches 2 years of age, only a small ductal system remains. Some growth in this tissue is noted during childhood, but no development occurs until puberty, when the female breast undergoes a period of extensive development.

With the onset of puberty, a process of elongation and branching of the ductal system begins, along with formation of lobules through development of some of the ductal endings into clusters of ductules and alveolar buds. This gradual process of branching and lobule formation occurs primarily under the influence of estrogen, but actually involves multiple hormones, including growth hormone. In the adult woman, some of these lobules will undergo further branching and glandular development with each menstrual cycle. However, full glandular differentiation and formation of the secretory acini capable of producing and secreting milk do not occur until pregnancy. The changes in breast structure that take place in pregnancy and lactation are referred to as mammogenesis and lactogenesis. Both processes are detailed in the *Anatomy and Physiology of Pregnancy* and *Breastfeeding and the Mother–Newborn Dyad* chapters, respectively. Following lactation, the breast glandular tissue undergoes regression and atrophy with tissue remodeling. The breast tissue atrophies further after menopause, with the number of lobes decreasing and fibrous connective tissue and adipose tissue accumulating during this phase of the life span.

The External Female Genitalia

The external female genitalia, also referred to as the vulva, are those structures located between the pubis and the perineum. They include the mons pubis, the labia minora, the labia majora, the clitoris, the hymen, the vestibule, and the urinary meatus or urethral opening. Although not externally visible, the Bartholin glands and Skene's glands and ducts as well as the vestibular bulbs are also considered part of the external female genitalia.

The mons pubis is the layer of fatty tissue that overlays the pubic bone. In the postpubertal woman, the skin of the mons is covered with coarse, curly hair. The labia majora are folds of connective and adipose tissue that extend inferiorly from the mons and merge posteriorly into the perineal body to form the posterior commissure. Medial to the labia majora are the labia minora, which are two thin folds of connective tissue. The area between the labia minora that extends from the clitoris to the fourchette is referred to as the vestibule and is the area into which the urethra, the ducts of the Bartholin glands, the vagina, and, sometimes, the Skene's ducts open. The labia minora merge superiorly to form the prepuce and frenulum of the clitoris and inferiorly to form the fourchette. Prior to a vaginal birth, both the labia majora and the labia minora assist in keeping the vaginal introitus closed and in protecting the urethral opening. In multiparous women, the labia minora may project beyond the labia majora; in contrast, in nulliparous women, the labia minora are usually not visible unless the labia majora are separated.

After menopause, pubic hair tends to thin and gray. The vulva also may thin and flatten, losing some of its fullness.

Clitoris

The clitoris is a highly innervated, erectile organ located in the superior portion of the vestibule where the labia minora fuse. It is often described as the homologue of the penis. Unlike the penis, however, the function of the clitoris is purely erogenous. The clitoris has both internal and external portions that include a glans, prepuce/hood, corpus, suspensory ligament, root, and vestibular bulbs (**Figure 10-3**).[6] The glans, which is approximately 1.5 to 2.0 centimeters in length, is the visible portion of the clitoris. The two crura are composed of erectile tissue that creates an inverted V structure attached to the pubic arch and extending along the ischiopubic rami. Adjacent to the crura and flanking the vaginal orifice are the vestibular bulbs, which are also composed of vascular, erectile tissue. These structures become engorged with blood during sexual arousal. The root of the clitoris is located just behind the glans; it is where all the erectile tissue merges.

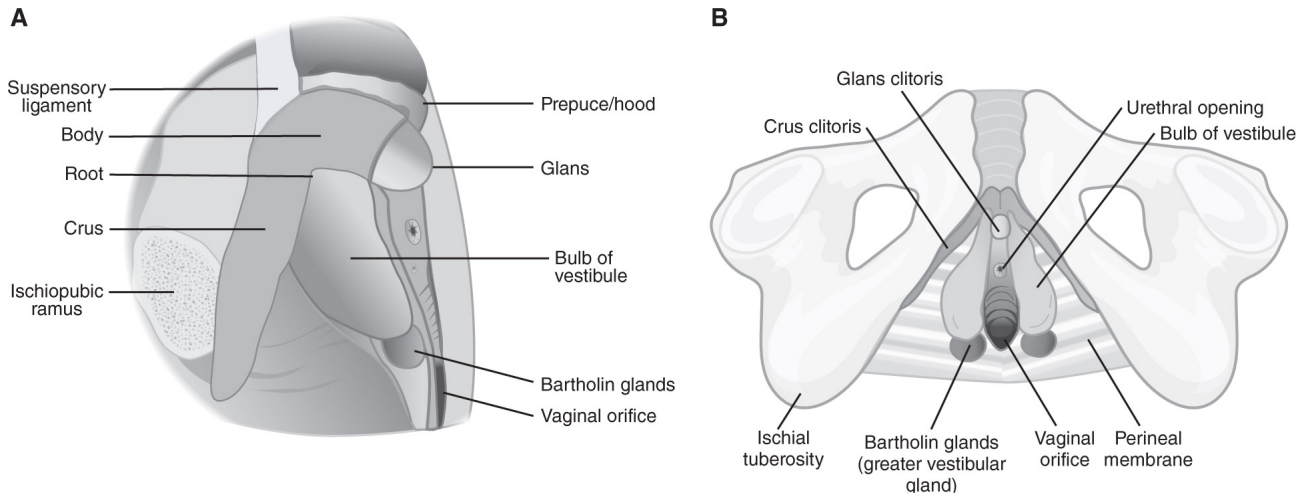


Figure 10-3 **A.** Lateral view of the clitoris and adjacent anatomy. **B.** Inferior view of the clitoris in relation to the urethra, vaginal orifice, and pelvic bones.

The clitoris is attached to the pubic symphysis by the suspensory ligaments. This organ is innervated by the dorsal nerve of the clitoris, branches of the pudendal nerve, and cavernous nerves that supply the erectile tissue arteries.

Although female sexual response may not be exclusively associated with clitoral stimulation, the clitoris is considered the center for orgasmic response.

Bartholin Glands and Skene's Glands

The Bartholin glands, which are also known as the major or greater vestibular glands, are located beneath the fascia of the vestibule on either side of the vaginal opening at about the 4 o'clock and 8 o'clock positions. Each gland has a duct that opens into the inferior part of the vestibule between the labia minora and the hymen. These glands secrete mucus during sexual arousal.

The Skene's glands, which are also known as the lesser vestibular glands or the periurethral glands, usually open onto the vestibule on either side of the urethra, but sometimes open on the posterior wall of the urethra. These glands also secrete mucus during sexual stimulation and/or arousal. Blockage of the Bartholin or Skene's glands can cause abscesses or cysts.

The Internal Female Genitalia

Figure 10-4 shows a midsagittal view of the internal organs and associated structures that make up the female reproductive system.[7] These organs and structures include the vagina, uterus, cervix, fallopian tubes, and ovaries. A detailed description of each component follows.

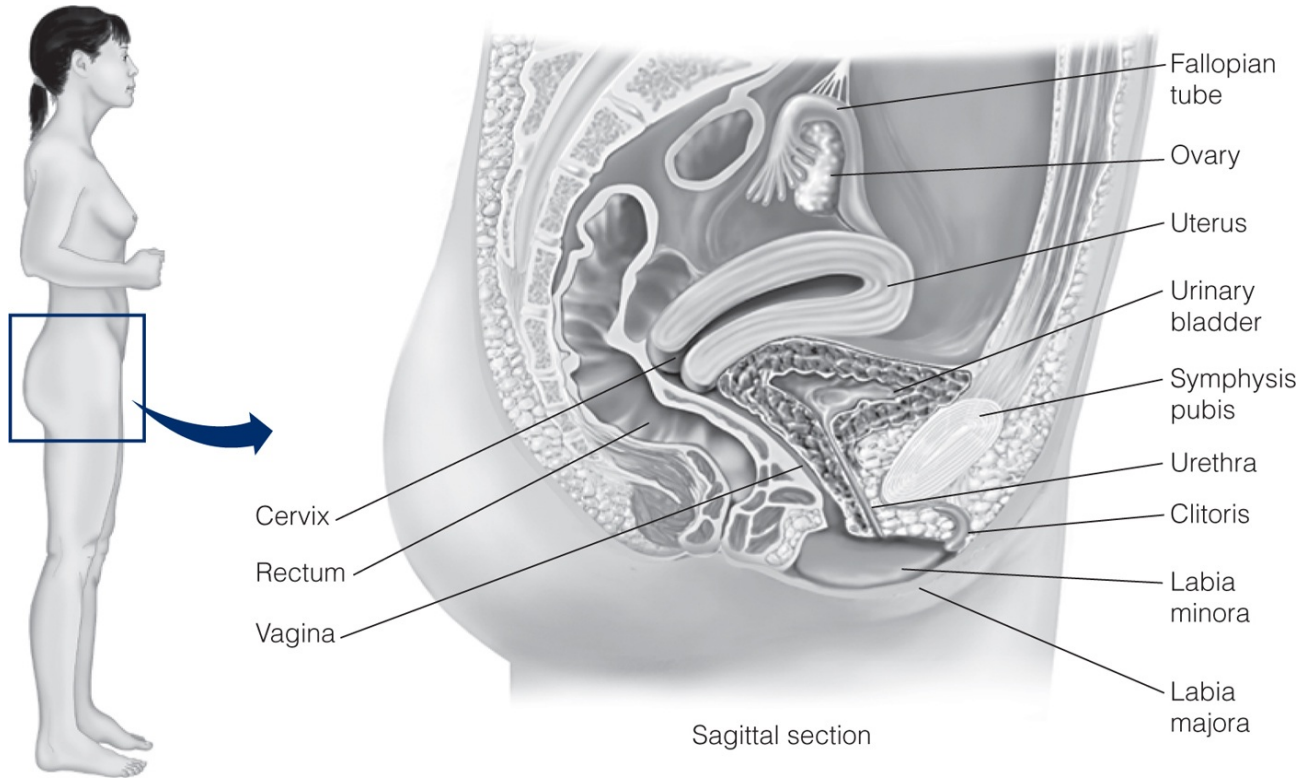


Figure 10-4 Midsagittal view of a woman's pelvis.

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The Vagina

The vagina is a muscular structure extending from the vulva to the cervix. It is a potential space, in that the walls are usually in opposition, but can be separated to form a tube during intercourse or childbirth. Anteriorly, the vagina is separated from the bladder by connective tissue known as the vesicovaginal septum. Posteriorly, the vagina is separated from the rectum by the rectovaginal septum (in the lower segment) and by the rectouterine pouch or cul-de-sac of Douglas (in the upper segment). The walls of the vagina, which are referred to as the anterior, posterior, and lateral vaginal walls, run from the vaginal opening at the vestibule to the cervix posteriorly, where they create a "dead end." The spaces created between the cervix and the ends of the vaginal walls are called the fornices. There are four fornices: the anterior fornix, the posterior fornix, and two lateral fornices.

The vagina has three layers: mucosa, muscularis, and adventitia. The vaginal mucosa is lined with a layer of stratified squamous epithelium, which in premenopausal women is folded

into ridges known as rugae that can stretch and provide distensibility to the vagina. Beneath this lining is a layer of smooth muscle. The outermost layer of connective tissue, referred to as the adventitia, is contiguous with the visceral endopelvic fascia. These layers maintain vaginal tone. Postmenopausally, the vaginal walls become thinner, the rugae less apparent, and elasticity and moisture lessen.

The blood supply to the vagina emerges from the vaginal artery, which branches from the uterine artery, or directly from the internal iliac artery, the inferior vesical arteries, and the middle rectal and internal pudendal arteries. Blood is drained from the vaginal area by a venous plexus that follows the arteries, and lymph is drained primarily via the inguinal lymph nodes.

The vagina is innervated primarily by the uterovaginal plexus, which arises from the inferior hypogastric or pelvic plexus. This latter structure contains sympathetic efferent fibers from the second, third, and fourth sacral nerves. In addition, the vagina is innervated by a few filaments from the first two sacral ganglia. The lower two-thirds of the vagina is primarily innervated by the pudendal nerve.

The Uterus

The uterus is a pear-shaped, muscular organ that, in the nonpregnant state, is situated in the pelvic cavity superior to the urinary bladder. The uterus, a potential space for the nonpregnant woman, is anchored in place, usually—but not always—in an anteverted position, by the uterine ligaments. The adult nulliparous uterus measures approximately 6 to 8 centimeters in length, 5 centimeters across, and 4 centimeters in thickness; it weighs approximately 50 grams. The functions of the uterus include receiving a fertilized ovum, providing the environment for the embryo and the fetus, and contracting to help in the expulsion of the fetus and placenta.

The uterus has two main parts: the corpus (also called the body) and the cervix. During pregnancy, the body of the uterus differentiates into an upper segment called the fundus and a lower segment called the isthmus (**Figure 10-5**).

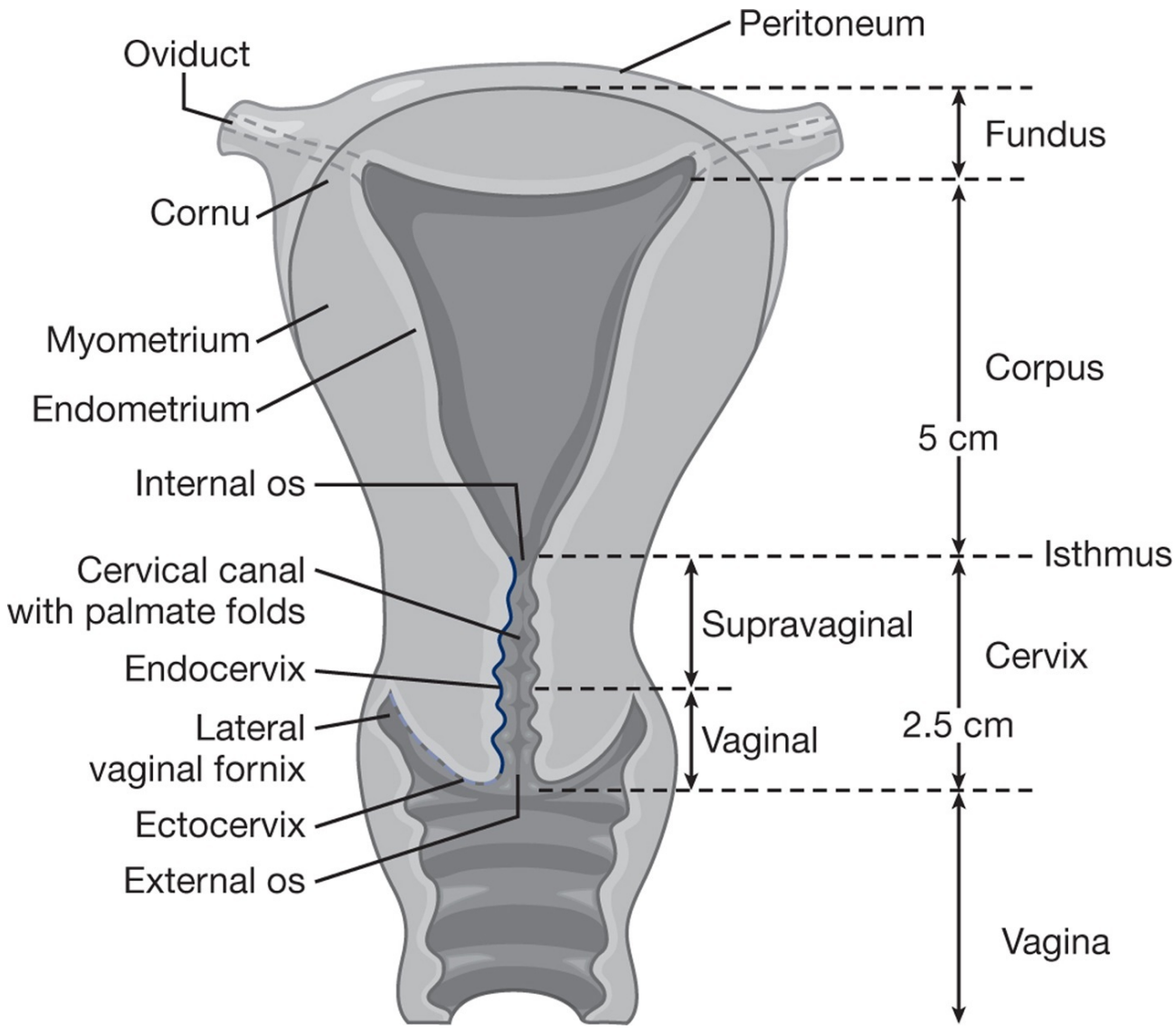


Figure 10-5 Frontal view of uterus.

The Uterine Body

The body of the uterus is made up of an external serosal layer formed by the peritoneum, a muscular (myometrial) layer, and a mucosal (endometrial) layer (**Figure 10-5**).

Endometrium

The endometrium is the mucosal lining of the uterine cavity. It is composed of (1) ciliated columnar epithelial cells; (2) glands that secrete a thin alkaline mucus rich in proteins, sugars, and secretions that allows survival of the zygote and blastocyst before implantation; and (3) the mesenchymal stroma, a layer of connective and vascular tissue that lies between the epithelial layer and the myometrium. This stroma can be subdivided into two layers: (1) the stratum basalis, which is closest to the myometrium and is not shed during menstruation, and (2) the stratum functionalis, the layer of the endometrium that proliferates and degenerates cyclically in menstruating women.

The endometrium receives blood from two different sets of arteries: a “straight” set of arteries that supply the stratum basalis and a set of “coiled” spiral arterioles that supply the stratum functionalis (**Figure 10-6**).[8] During the menstrual cycle, these vessels and the rest of the endometrium undergo marked changes. As part of the proliferative phase of the menstrual cycle, under the effects of rising levels of estrogen and progesterone, the endometrium becomes increasingly vascularized and the glands of the endometrium become longer and increasingly convoluted and fill with secretions. As a result, there is a 10-fold increase in thickness of the myometrium during the proliferative phase, from 0.5 to 5 millimeters.

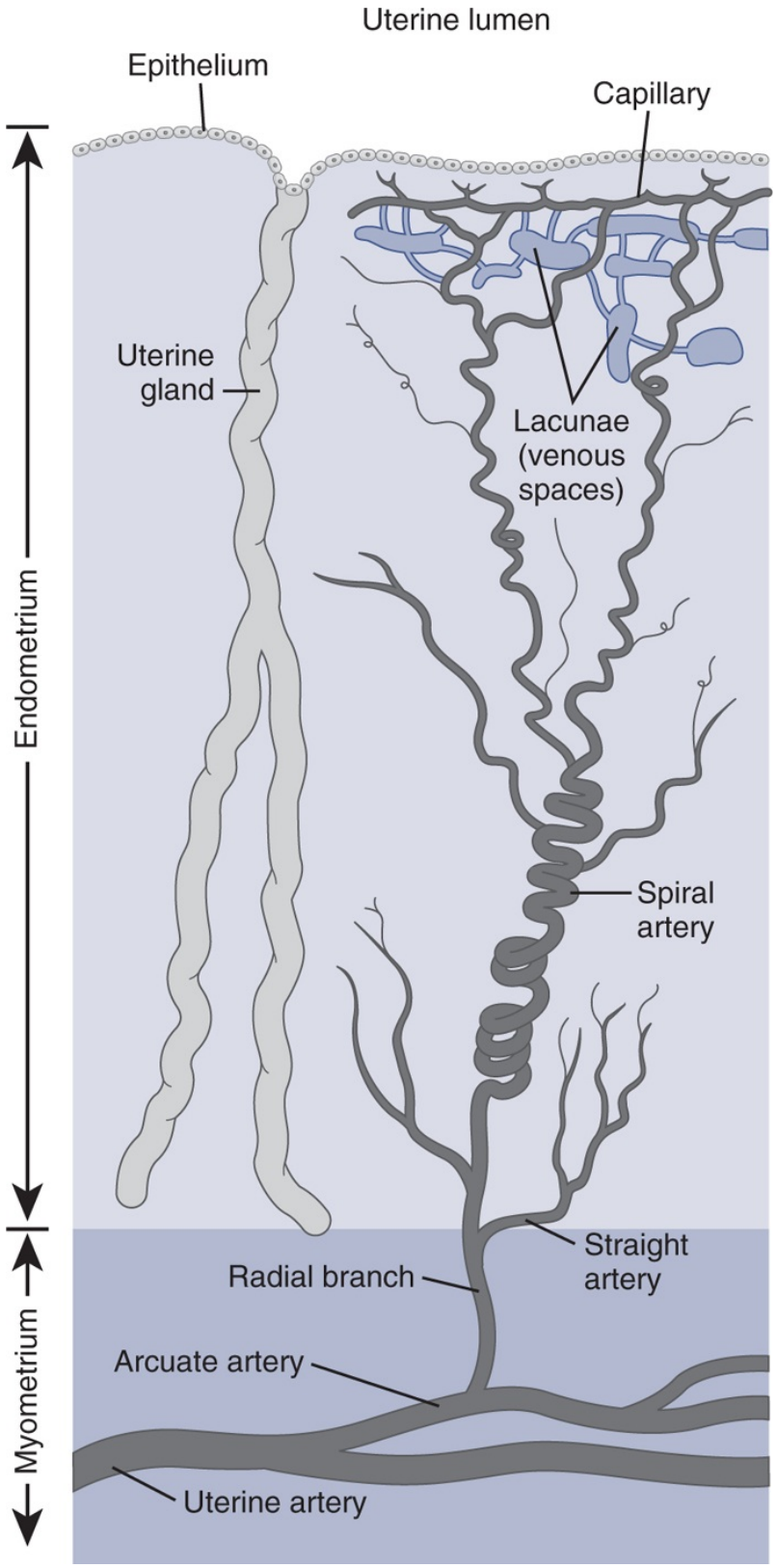


Figure 10-6 Anatomy and circulation of the endometrium.

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If fertilization and implantation do not occur, the subsequent drop in estrogen and

progesterone levels leads to atrophy of the functional layer of the endometrium, which in turn leads to increased coiling of the spiral arterioles and a regression of the glandular development. The excessive coiling of the spiral arterioles diminishes the blood flow to the endometrial layer, which then produces tissue ischemia, necrosis, and endometrial bleeding. Menstrual flow is the result of this bleeding and shedding of necrotic endometrial tissue. A more detailed description of the menstrual cycle appears later in this chapter.

Once a woman has reached menopause, the endometrium becomes atrophic. The epithelium flattens, the glands gradually disappear, and the endometrial stroma becomes increasingly fibrous. In some cases, however, the endometrium may retain a weak proliferative capacity, which may contribute to the development of endometrial hyperplasia and perhaps ultimately endometrial cancer.[9]

Myometrium

The bulk of the uterus is made up of the myometrium, which is composed of bundles of smooth muscle fibers separated by connective tissue made primarily of collagen and elastin. The muscle fibers of the myometrium are arranged in three distinct patterns that contribute to this tissue's ability to contract effectively. In the inner layer of the myometrium, the fibers run in a circular pattern that spirals in a perpendicular orientation to the long axis of the uterus. In the middle layer, the muscle fibers are interlaced and form figure-eight patterns running diagonally along the long axis of the uterus.[10] The outermost layer of the myometrium contains an arrangement of fibers in both longitudinal and spiral patterns. These layers become more differentiated in pregnancy with the hypertrophy of the myometrial cells that occurs in pregnancy. Changes in the uterus during pregnancy are discussed in the *Anatomy and Physiology of Pregnancy* chapter, and the *Anatomy and Physiology During Labor and Birth* chapter contains a detailed description of the physiology of uterine contractions.

A gradient in the amount of muscle tissue found throughout the uterus is apparent, such that at the fundus, the myometrium contains primarily muscle fibers, whereas only 10% to 15% of the tissue mass in the cervix is composed of muscle fibers. The proportion of muscle fibers present determines the contractile strength of the uterine muscle, so this distribution of muscle creates a contraction strength gradient along the length of the uterus.

Uterine Ligaments, Blood Supply, and Innervation

The broad ligaments of the uterus are composed of folds in the peritoneum. These two wing-like structures extend from the sides of the uterus to the pelvic sidewalls. The superior part of the broad ligaments forms the suspensory ligament of the ovary and the mesosalpinx (to which the fallopian tubes are attached). The round ligaments attach on either side of the uterus just below and in front of the insertion of the fallopian tubes; they then cross the broad ligament in a fold of peritoneum, pass through the inguinal canal, and insert in the anterior (upper) portion of the labia majora on either side of the perineum. The ligaments are composed largely of smooth muscle that is continuous with the smooth muscle of the uterus. The round ligaments hypertrophy during pregnancy and stretch as the uterus enlarges. The peritoneal ligaments

(uterosacral, cardinal, and pubocervical) extend from the cervix to different parts of the pelvis and help to stabilize the uterus.

The vessels that supply the uterus are derived primarily from the uterine and ovarian arteries. The uterine artery is a branch of the anterior branch of the internal iliac arteries (also referred to as the hypogastric arteries). The uterine artery enters the side of the uterus through the broad ligament and divides into several branches supplying the lower cervix and upper vagina, the upper portion of the cervix, and the body of the uterus. One large branch of the uterine artery travels along the margin of the uterus toward the fundus. At approximately the height of the round ligament, this branch of the uterine artery furcates into three vessels. One vessel joins the terminal branch of the ovarian artery, one supplies part of the fallopian tube, and one supplies the uterine fundus. The vessels that supply and drain blood from the uterus are collectively referred to as the arcuate vessels of the uterus.

The ovarian artery is a direct branch of the aorta that enters the broad ligament. The main stem of the ovarian artery travels near the mesosalpinx (the part of the broad ligament that encloses the fallopian tubes) all the way to the upper, lateral portion of the uterus to join with the ovarian branch of the uterine artery. Branches from this main stem perfuse the ovaries and fallopian tubes.

The arcuate uterine veins that compose the uterine venous plexus join to form the uterine vein, which empties into the internal iliac vein. The blood from the upper and outer parts of the uterus and ovaries is collected by multiple veins that form the pampiniform plexus, draining into the ovarian vein. The right ovarian vein empties into the vena cava, while the left ovarian vein empties into the left renal vein. Lymph from the body of the uterus is drained by lymph vessels that carry their contents to either the internal iliac nodes or the periaortic nodes. The periaortic nodes also receive lymphatic drainage from the ovaries.

The parasympathetic system innervates the uterus via branches arising from the second, third, and fourth sacral nerves. In addition, the uterus receives some innervation from the sympathetic nervous system by the presacral nerve and lumbar sympathetic chain.

The Cervix

The cervix is the cylindrical, fibromuscular lower portion of the uterus that is sometimes referred to as the neck of the uterus. It has two areas of constriction at each end: the internal os, which is found at the junction of the cervix and uterine body, and the external os, which opens into the vagina. The role of the cervix and the cervical ora in pregnancy and birth are additionally reviewed in the *Anatomy and Physiology of Pregnancy* and *Anatomy and Physiology During Labor and Birth* chapters.

The cervix is composed primarily of subepithelial stroma, along with a smaller proportion of smooth muscle fibers.[11] The subepithelial stroma is approximately 80% collagen and ground substance, which contains blood vessels, nerves, and glands that secrete thick mucus. Elastin (a different protein than collagen) is present in smaller amounts but appears to play an important role in cervical remodeling during pregnancy, in labor, and after birth.[11] Recent studies have found that smooth muscle lies in a circular pattern around the internal os.[12] This tissue is not a classic sphincter, but may play a role in the cervical remodeling that occurs

during early labor.

The tunnel-like area between the internal and external os is called the cervical canal. The external surface of the cervix, known as the ectocervix, is lined with squamous epithelial cells like those lining the vagina. In contrast, the inner canal of the cervix, known as the endocervix, is lined with glandular epithelial cells, also termed columnar epithelial cells.

The area where the endocervix and exocervix meet is known as the transformation zone or the squamocolumnar junction; metaplasia begins here. It is in this region where most cervical cancers originate—which explains why obtaining a sample of these cells during a Pap test is important. Based on several factors such as age, pregnancy status, or hormonal contraceptive use, the squamocolumnar junction may be more or less apparent. When the columnar cells are visible, this benign condition usually is known as ectopy or eversion. In years past, eversion was sometimes termed erosion or ulceration, but this is a misnomer. A prepubescent female has a pink, smooth cervix without eversion. Women using hormonal contraception or who are pregnant often have marked eversion. As a woman ages, the cervical canal tends to recede and the squamocolumnar junction often is not visible.

The Fallopian Tubes

The two fallopian tubes, also referred to as oviducts and salpinges, are the 8- to 14-centimeter-long, narrow, muscular tubes that extend from the uterine horns or cornua. The oviducts, as their name would imply, transport the ovum from the ovary to the uterus. It is also in the fallopian tubes that fertilization normally takes place. The fallopian tubes have three layers: (1) the mucosa, a single-layer lining of ciliated or secretory columnar cells; (2) the muscularis layer; and (3) the serosa, the outer covering of the tubes, which is derived from the visceral peritoneum.

The fallopian tubes can be divided into four sections:

- The pars interstitialis is the portion of the tube that penetrates the muscular wall of the uterus and connects the cavity of the oviduct with the uterine cavity.
- The isthmus is the narrow segment that extends from the uterus.
- The ampulla is a wider segment of the oviduct and is where fertilization of the ovum most commonly occurs.
- The infundibulum is the fimbriated, open distal end of the fallopian tube.

The fimbriae are fine, fingerlike mucosal projections at the end of the fallopian tube that sweep near the ovaries, but are not connected to the ovaries. As a consequence, the oviducts open directly into the abdominal cavity. The fallopian tubes vary in diameter at the different segments, with the isthmus measuring approximately 2 to 3 millimeters in thickness while the ampulla reaches a diameter of 5 to 8 millimeters.

The structure of the oviducts promotes movement of the ovum from the ovary into the tubes and down toward the uterine cavity. The cilia of the mucosa move in waves to assist with transport of the ovum, and the fibers of the muscularis layer are arranged so that the oviducts may move and contract in a way that promotes transport of the ovum.

The Ovaries

The ovaries are the organs of gamete production in the female. In addition to producing ova, the ovaries manufacture the steroid hormones estrogen and progesterone and, therefore, are also part of the endocrine system. The two ovaries, which are almond shaped, vary in size from each other, from woman to woman, and in the same woman in different stages of her life. During the reproductive years, the ovaries measure, on average, 2.5 to 5 centimeters in length, 1.5 to 3 centimeters in width, and 0.6 to 1.5 centimeters in thickness. These organs are located in the upper part of the pelvic cavity. Each is attached in three ways: (1) to the broad ligament by the mesovarium; (2) to the uterus by the utero-ovarian ligaments, which extend from below the insertion points of the fallopian tubes in the posterior part of the uterus; and (3) to the pelvic wall by the suspensory ligament of the ovary (also referred to as the infundibulopelvic ligament). The suspensory ligament of the uterus is a fold of the peritoneum that extends from the ovary to the pelvic wall. This fold also contains blood vessels and nerves that supply the ovary.

Each ovary is made up of two main parts: the cortex and the medulla. The cortex—the outer layer of the ovary—is composed of connective tissue and contains follicles at different stages of development. It is lined with germinal epithelium, and its dull and whitish outer layer is referred to as the tunica albuginea. The medulla of the ovary also consists of connective tissue and contains arteries, veins, and smooth muscle fibers.

Two primary glandular cells within the ovarian follicles are involved in the synthesis of steroids: the thecal cells and the luteal (or granulosa) cells. Theca cells differentiate from the interfollicular stroma of ripening follicles in response to proteins secreted by growing follicles.[13] These cells produce the androgen substrate required for ovarian estrogen biosynthesis.

Ovarian Development

The human gonads are derived from three types of embryonic tissues: (1) coelomic epithelium, (2) mesenchyme, and (3) primordial germ cells. The primordial germ cells, which eventually will produce the gametes, originate in the yolk sac of the developing embryo. Until approximately 4 weeks' gestation, no sexual differentiation occurs in the development of the gonads. However, once the sex-determining region of the Y gene becomes activated, the process of sex differentiation begins.[14] Whereas development of the male reproductive system relies on activation of this gene and the effects of specific hormones, the development of the ovaries and female genitals follows the "default" process that happens in the absence of these hormones. For example, in the absence of testosterone, the Wolffian structures (which in the male embryo will become the male internal genitalia) regress. Similarly, the Müllerian ducts, without suppression from Müllerian-inhibiting hormone from the fetal testes, continue to develop and become the uterus, the oviducts, and the upper part of the vagina.

The ovaries develop from the coelomic epithelium, which proliferates to form the medulla and the cortex by the third month of gestation. The primordial germ cells in these fetal ovaries undergo rapid division and then differentiate into primary oocytes. By the fourth month of gestation, each ovary contains approximately 10 million primary oocytes and some primordial

follicles (oocytes surrounded by follicular cells). Most of the oocytes degenerate, so that by birth the neonate has approximately 250,000 to 500,000 oocytes; by puberty, only about 200,000 oocytes are left.[15] The oocytes stop developing and will remain in a state of arrested meiosis until puberty. Information regarding ovulation is found later in this chapter in the section discussing the menstrual cycle.

The Bony Pelvis

The bony pelvis has multiple functions. First, it provides an attachment site for the muscles and connective tissue of the pelvis, thereby providing support and stability to the pelvic organs. Second, it provides the site of attachment and articulation for the lower limbs. Third, it supports the weight of the upper trunk and distributes it to the lower extremities. Finally, in the female, the fetus passes through the bony pelvis during childbirth.

Before puberty, males and females possess similar pelvises. Proximate to puberty, however, the female pelvis changes in a manner conducive to pregnancy and birth. When a woman reaches approximately 40 years, the pelvis gradually changes so that it becomes more similar to the pelvis of her male counterpart.[16]

Pelvic Bones

The pelvis comprises four bones: two innominate bones, the sacrum, and the coccyx (**Figure 10-7**).

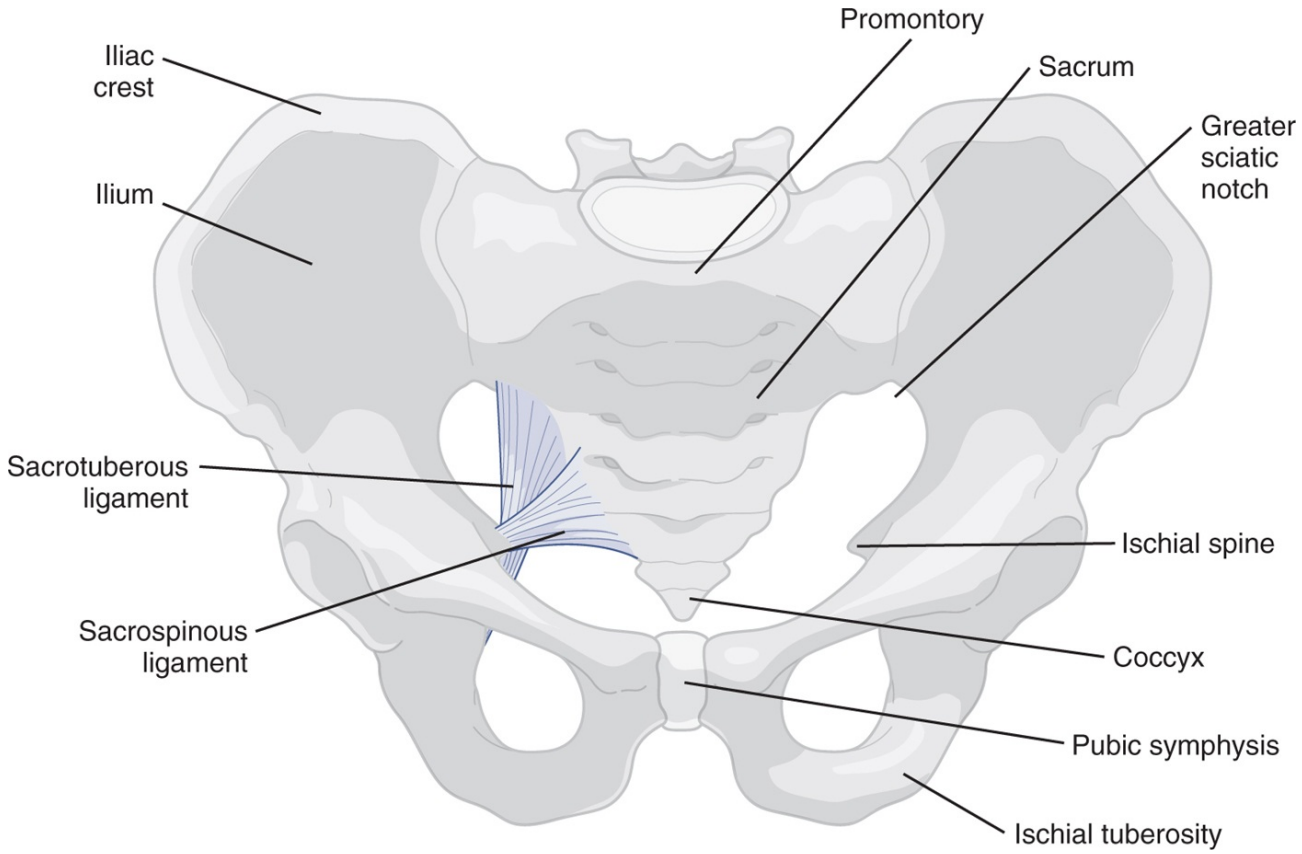


Figure 10-7 Front view of the bony pelvis with ligaments.

Each innominate bone has three parts: the ilium, the ischium, and the pubis. The ilium is the posterior and upper portion of the innominate bone; the two ilia join the sacrum along the sides at the sacroiliac synchondroses. The ischium is the medial and lower portion of the innominate bone. Important bony landmarks of the ischium include the ischial spine, the ischial tuberosity, and the pelvic sidewall. The pubis is the anterior portion of the innominate bone. The two

pubic bones join at the symphysis pubis, where they are attached to an avascular fibrocartilage disc. The inferior margin of the symphysis pubis and the descending rami create the pubic arch, another important bony landmark of the pelvis.

The longitudinal axis of the symphysis pubis is normally parallel to the longitudinal axis of the sacrum. If the symphysis pubis is not at least approximately parallel to the sacrum, the anteroposterior diameters of two of the three pelvic planes that the fetus passes through during birth—namely, the pelvic inlet and the pelvic outlet—can be changed significantly. Tilting of the superior margin of the symphysis pubis toward the sacral promontory and of the inferior margin away from the sacrum is called anterior inclination. Tilting of the inferior margin of the symphysis pubis toward the sacrum and the superior margin away from the sacral promontory is called posterior inclination (**Figure 10-8**).

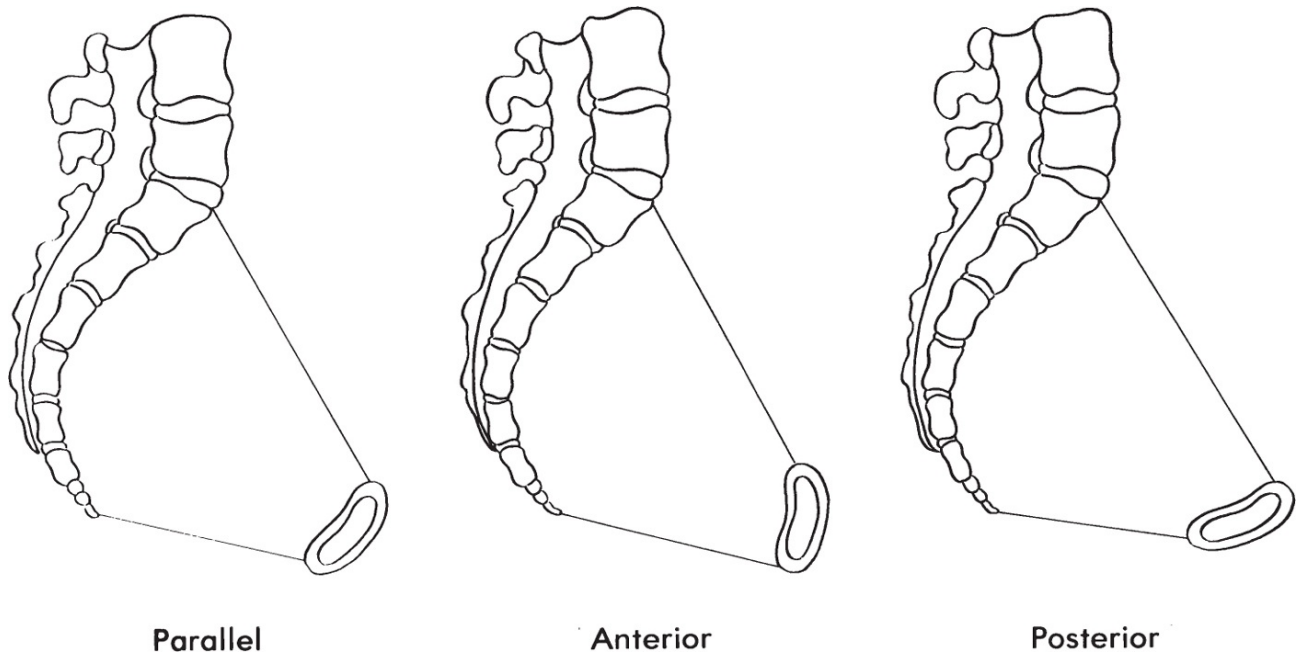


Figure 10-8 Inclination of symphysis pubis.

The sacrum and the coccyx make up the posterior portion of the pelvis. The sacrum is formed by the fusion of the five sacral vertebrae. The superior part of the sacrum, the sacral promontory, is an important bony landmark of the pelvis. The coccyx is formed by the fusion of usually four, or occasionally three or five, rudimentary vertebrae and is itself an important bony landmark. The sacrum and the coccyx join at the sacrococcygeal symphysis.

Pelvic Joints

There are four joints within the bony pelvis: the symphysis pubis, the two sacroiliac synchondroses (or sacroiliac articulations), and the sacrococcygeal symphysis. These joints are made of a network of cartilage and/or ligaments that join the bones of the pelvis. During pregnancy, the joints of the pelvis soften under the influence of the hormones of pregnancy, including relaxin. This softening allows for movement and widening of the pelvis, which increases its potential size to facilitate birth.

The pubic symphysis is composed of fibrocartilaginous tissue. During pregnancy, it widens slightly. Although this widening usually does not exceed 10 millimeters, it may cause tenderness, particularly with motion.[16,17] Rarely, the symphysis pubis may separate spontaneously (or surgically) during labor.

The sacroiliac joints are primarily weight-bearing joints that take the weight of the upper body and distribute it to the lower limbs. They are synovial joints, meaning that the surfaces of the articulating bones are covered by a thin membrane of cartilage and are separated by a joint cavity lined by a synovial membrane that produces a lubricating fluid. The resulting hinge-type movement gives the sacrum the ability to rotate slightly, enlarging the measurements of two of the three pelvic planes that the fetus passes through during birth.

The sacrococcygeal joint is also a synovial hinge joint that allows flexion and extension of the coccyx. Extension occurs during relaxation of the levator ani and rectal sphincter muscles —an event that occurs normally during defecation and during the birth process. Extension of the coccyx increases the anteroposterior diameter of the plane at the pelvic outlet.

Pelvic Ligaments

The term “ligament” is generally used to describe connective tissue that joins two bones. However, in the pelvis, in addition to indicating a connection between bones, the term “ligament” is used to describe a variety of tissues that connect pelvic organs to bones. Nevertheless, the sacrospinous and sacrotuberous ligaments of the sacrum are “true” ligaments in the bony pelvis that help provide stability to the pelvis in part by supporting the sacrum and restricting its ability to tilt.

The sacrospinous ligaments are triangular, with a broad base that attaches to the lateral margins of the sacrum and coccyx and the insertion at the apex of the ischial spine. The coccygeus muscle lies along the pelvic aspect of this ligament. The pudendal nerve, which innervates the skin and muscles of the perineum, lies just posterior to the sacrospinous ligaments where it attaches to the ischial spine. The sacrotuberous ligaments attach to the sacrum at the level of the S3 through S5 vertebrae and extend to the inferior spine of the ilium, where it becomes the ischial tuberosity extending inferior and lateral to the sacrospinous ligaments.

The Pelvic Floor

The pelvic floor is made up of major muscle groups as well as ligaments and fascia that together support the abdominal and pelvic organs, maintain continence, control urination and elimination, and facilitate passage of the fetus during birth. From the most superior to the most inferior, the major components of the pelvic floor are (1) the endopelvic fascia, (2) the pelvic diaphragm, (3) the urogenital diaphragm, and (4) the superficial perineal muscles. The muscles of the pelvic floor are described in **Table 10-1**.

Table 10-1	**Muscles of the Perineum**

Muscle	Description	Function
Pubococcygeus	Primary portion of the levator ani. It originates at posterior border of the symphysis pubis and sweeps back to insert on lateral margins of the coccyx. It is divided into three bands.	
	Pubovaginalis: origin is the posterior aspect of the pubis and insertion is the fascia of the vagina and perineal body. The muscle is in a U shape around the vagina.	Acts as a sling for the vagina and vaginal sphincter (muscle that causes spasm during vaginismus).
	Puborectalis: intermediate fibers that form a U loop around the anal rectal junction and insert into the posterior wall of the rectum, blending with the anal sphincter.	Sling and accessory sphincter for the rectum.
	Pubococcygeus proper: lateral fibers that go directly back to form a Y and insert on the lateral margins of the coccyx.	Flexes the coccyx to increase anal–rectal flexure and control defecation.
Iliococcygeus	Arises from a facial line on the obturator internus muscle along the pelvic wall of the obturator foramen and extends to insert on the lateral margins of the coccyx and anococcygeal raphe.	Pelvic organ support and urinary continence.
Bulbocavernosus	Two bulbocavernosus muscles. Posteriorly, they attach to the central tendinous point of the perineum; anteriorly, they insert into the corpus cavernosus of the clitoris; laterally, they surround the orifice of the vagina, covering the vestibular bulbs and Bartholin glands on either side.	Known as the sphincter vaginae, contraction of these muscles reduces the size of the vaginal orifice. Contraction of the anterior muscle fibers contributes to clitoral erection.
Ischiocavernosus	Two ischiocavernosus muscles, one on either lateral boundary of the perineum. Posteriorly, they arise from the inner surface of the ischial tuberosities; anteriorly, they cover and insert into the sides and posterior surface of the crus clitoris; laterally, they extend from the clitoris to the ischial tuberosities along the ischial ramus, from which they derive some of their fibers.	Maintain clitoral erection.
Superficial transverse perineal (transversus perinei superficialis)	Two superficial transverse perineal muscles arise from the inner and anterior surfaces of the ischial tuberosity of the superior ramus of the ischium by a small tendon; they insert into the central tendinous point of the perineum.	Fix the location of the central tendinous point of the perineum.
Perineal membrane	Arises from the inferior ramus of the ischium, spans the anterior pelvic outlet, and is attached to the lateral vaginal walls. The urethra passes through this membrane.	Helps to stabilize the vagina.
Central tendinous point of the perineum	A fibromuscular structure in the midline between the vagina and the anus, and at the base of the urogenital diaphragm. The tissue is fibrous because it is the point of fusion for both the superior and inferior fascia of the urogenital diaphragm and the external perineal and Colles' fascia; it has muscular fibers because it is a common point of attachment for a number of muscles whose fibers blend together into the central tendinous point of the perineum, among them the bulbocavernosus, superficial transverse perineal, some fibers of the deep transverse perineal, external anal sphincter, and the levator ani–pubococcygeus.	Common point of attachment for a number of layers of fascia and muscles.

The endopelvic fascia is a layer of connective tissue that surrounds the vagina and provides support to it and to the visceral organs. This fascia also plays a role in urinary continence by providing support to the urethra and neck of the bladder.[18,19]

The pelvic diaphragm separates the pelvic cavity from the perineal space and can be envisioned as a diamond-shaped hammock that supports the viscera, abdominal organs, and pelvic organs. The pelvic diaphragm consists of the levator ani muscles, composed of the puborectalis, pubococcygeus, and iliococcygeus; the coccygeus muscle; and their associated fascia (**Figure 10-9**). Insertions of the muscles of the levator ani group around the vagina, urethra, and rectum allow passage of these openings and create functional sphincters that contribute to fecal and urinary continence. The levator ani maintains the position of the pelvic organs. This muscle originates at the posterior surface of the superior ramus of the pubis, then runs along the arcus tendineus, a line of fascia on the internal surface of the ischium. The levator ani inserts into the last segments of the coccyx laterally and into tendons on the rectum, vagina, and urethra medially, so that they make up part of the sphincters for these organs.

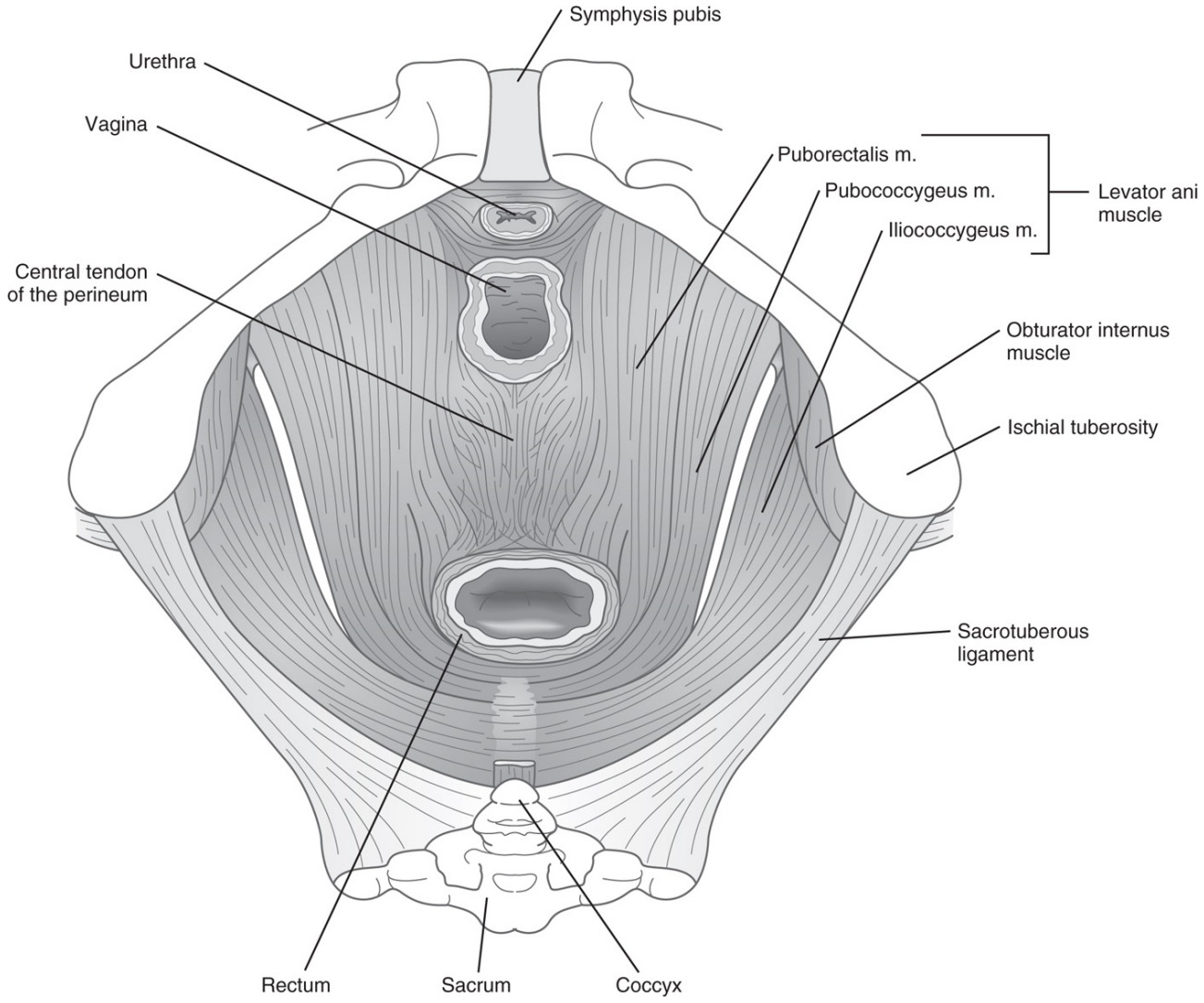


Figure 10-9 Superior view of the structures of the pelvic diaphragm.

When the muscles of the pelvic floor become damaged, which can occur during childbirth, support of the urethra and other pelvic structures becomes increasingly dependent on the ligaments and fascia. Under the strain of continuous or prolonged stretching, these ligaments and fascia can fail and become unable to prevent organ prolapse and/or stress incontinence.

Perineal Membrane

The perineal membrane is a fibrous sheet that spans the urogenital triangle just inferior to the pelvic diaphragm. The urogenital triangle is an equilateral triangle defined by the pubic symphysis and ischial tuberosities. The perineal membrane has historically been called the urogenital diaphragm. However, newer studies suggest that the urogenital diaphragm is really a single layer of muscle and fascial tissue that should be referred to as the perineal membrane.[20] The urethra and the vagina pass through this membrane.

Superficial to the perineal membrane are the bulbocavernosus, ischiocavernosus, and superficial transverse perineal muscles (**Figure 10-10**). The perineal body and central tendinous point of the perineum, which crosses the midline of the perineum between the vaginal and anal openings, is the point of convergence of the superficial transverse perineal muscle, the urogenital sphincter, the bulbocavernosus muscles, and the external anal sphincter. This perineal body is also the point of attachment of the two sides of the perineal membrane, provides support to the posterior vaginal wall, and helps prevent rectal prolapse. Damage to the perineal body, in addition to stretching of the levator ani muscles that may occur during the second stage of labor, can result in rectal prolapse.

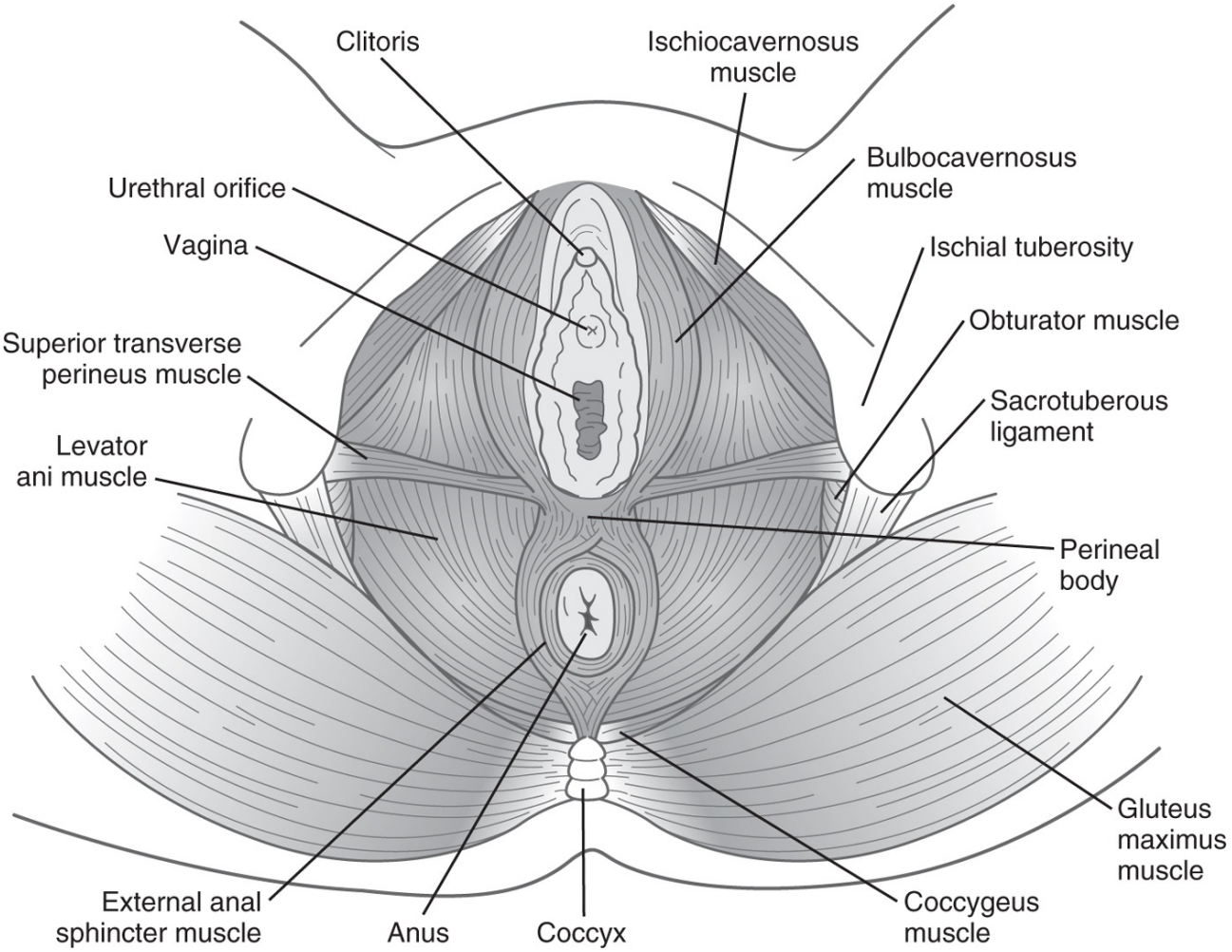


Figure 10-10 Inferior superficial view of the structures of the perineum.

An understanding of how pelvic floor anatomy promotes urinary and fecal continence is necessary to understand how changes related to pregnancy, childbirth, and aging result in some women developing pelvic floor dysfunction, including stress urinary incontinence.

Bladder and Urethra

In a female, the urethra is approximately 4 centimeters long and runs posteriorly along the pubic symphysis from the external opening (urethral meatus) to the bladder. The urethra is composed of an inner layer of epithelium surrounded by a layer of vascular tissue and a layer of muscular tissue, which together help maintain the urethra in a constricted position. The urethra is stabilized by the pubourethral ligaments, which attach the anterior portion of the urethra to the symphysis pubis. To maintain urinary continence, urethral constricting pressure must be greater than bladder pressure. This pressure balance is achieved in part by the constriction provided by the urethral muscles and sphincters and in part by the support/compression provided by the pelvic floor muscles and their associated structures.

Two sphincter structures are involved in urinary continence: the inner sphincter at the neck of the bladder and the external urethral rhabdosphincter/urethrovaginal sphincter complex. The inner sphincter is an extension of the detrusor smooth muscle of the bladder that is under

autonomic control, whereas the urethral rhabdosphincter/urethrovaginal sphincter complex is composed of skeletal muscle and is under voluntary control. Support by the pelvic floor musculature is also critical to maintaining urinary continence. The major components of this supportive structure are the vaginal wall, the endopelvic fascia, the arcus tendineus fasciae pelvis, and the levator ani muscles. The endopelvic fascia surrounds the vagina and attaches to the arcus tendineus fascia, which serves to suspend and support the urethra.

The urethra exits the pelvis via the urogenital hiatus of the levator ani, through which the vagina also passes. The baseline muscle tone of the levator ani, therefore, is critical in maintaining urinary continence by keeping the urethra compressed.

Anorectal Anatomy

The anal canal is approximately 2.5 to 5 centimeters in length. The anal sphincter surrounds the anal canal and is a multilayer structure that includes the anal mucosal lining, the internal sphincter, and the external sphincter. The internal anal sphincter comprises a layer of muscle that is a continuation of the muscular layer of the rectum, ending approximately 1 centimeter from the anal opening.[21] Similar to the internal sphincter, the external anal sphincter is a cylindrical muscular structure; it is partially interwoven with the levator ani muscle and extends all the way to the anal opening. The external anal sphincter is composed of striated muscle under voluntary control.

Pelvic Nerves and Vasculature

The perineum is perfused by the internal pudendal artery and branches of the pudendal artery, including the inferior rectal artery. These vessels are branches of the anterior division of the internal iliac (hypogastric) artery. Perineal innervation occurs via the pudendal nerve and its branches, which originate from S2, S3, and S4. The pudendal nerve also innervates the levator ani, rectal sphincter, skin of the vulva and lower portion of the birth canal, and muscles of the urogenital diaphragm.

The Obstetric Pelvis

The pelvis is divided into the “false pelvis” and the “true pelvis” by the linea terminalis, an invisible line that runs along the pelvic brim from the superior part of the symphysis pubis around to the sacral promontory (**Figure 10-11**). The false pelvis, which includes the iliac fossa and iliac crest, is deemed “false” because it has little obstetric significance yet defines the lower border of the abdominal cavity. The true pelvis constitutes the bony passageway through which the fetus must maneuver to be born vaginally. This bony passage is actually a curved canal with a shallow anterior wall that is approximately 5 centimeters long, and a deep concave posterior wall that is approximately 10 centimeters long.

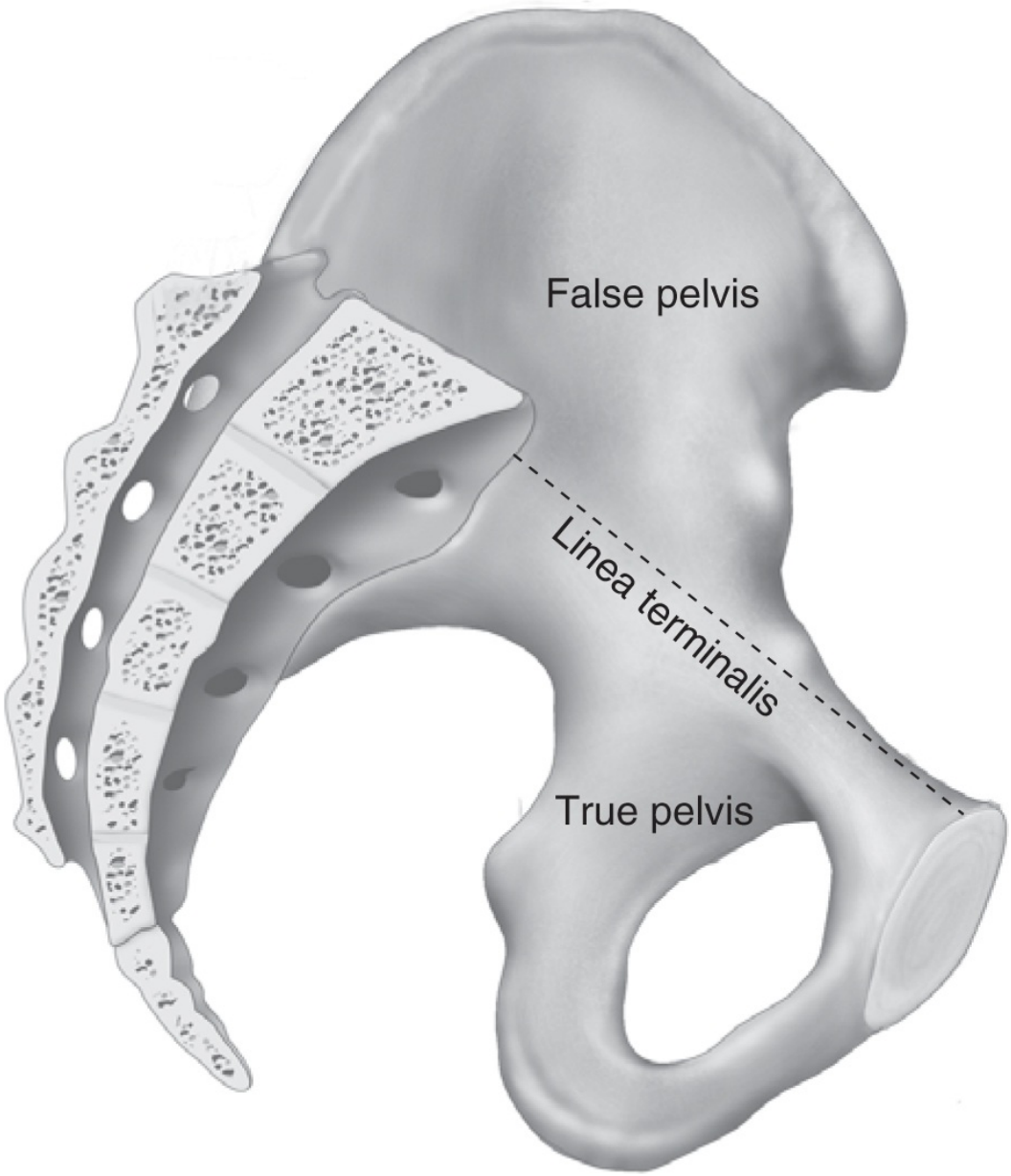


Figure 10-11 The linea terminalis is a plane that separates the true and false pelvis. The linea terminalis is made of the superior posterior border of the symphysis pubis, iliopectineal (acruate) line, and sacral promontory.

The true pelvis has five boundaries:

1. *Superiorly:* the sacral promontory, linea terminalis, and upper margins of the pubic bones
2. *Inferiorly:* the inferior margins of the ischial tuberosities and the tip of the coccyx
3. *Posteriorly:* the anterior surfaces of the sacrum and the coccyx
4. *Laterally:* the sacroiliac notches and ligaments and the inner surfaces of the ischial bones
5. *Anteriorly:* the obturator foramina and the posterior surfaces of the symphysis pubis, pubic bones, and ascending rami of the ischial bones

Clinically Important Aspects of the True Pelvis

The birth canal shape is determined by the architecture of the sacrum, sacrosciatic notch, sidewalls, ischial spines, and subpubic arch. Therefore, the shape of the true pelvis has several clinically relevant aspects.

The true pelvis has three planes of obstetric significance: the inlet, the midplane, and the outlet (**Table 10-2**). Each of these planes has anteroposterior and transverse diameters of import that are critical for evaluation of pelvic adequacy for vaginal birth.

Table 10-2 **Pelvic Planes and Obstetric Diameters**

Plane	Boundaries of Plane	Significant Obstetric Diameters of Plane
Pelvic inlet: (superior strait): upper entry into the true pelvis	**Posterior:** sacral promontory **Lateral:** iliopectineal (arcuate) line that extends to the horizontal rami superior posterior edge of the pubic bones **Anterior:** superior posterior border of the symphysis of the symphysis pubis	**Anteroposterior diameters:** 1. *Conjugata vera:* true conjugate of the inlet; extends from the middle of the sacral promontory to the middle of the upper posterior margin of the symphysis pubis; this diameter is normally 11 cm or more. 2. *Obstetric conjugate of the inlet:* extends from the middle of the sacral promontory to the middle of the posterior symphysis pubis. The pelvis is considered contracted if the obstetric conjugate measures less than 10 cm. 3. *Diagonal conjugate of the inlet:* extends from the middle of the sacral promontory to the middle of the inferior (lower) margin of the symphysis pubis. This is the only diameter of the pelvic inlet that can be measured clinically. A normal clinical measurement is considered to be 11.5 cm or more. **Transverse diameter:** greatest distance between the linea terminalis on either side of the pelvis; this distance is approximately 13.5 cm. **Oblique diameters:** distance between the sacroiliac synchondrosis on one side of the pelvis and the iliopectineal eminence on the opposite side of the pelvis. The oblique diameters average slightly less than 13 cm (12.75 cm) each.
Pelvic midplane: plane of least dimensions	**Posterior:** sacrum at the junction of the fourth and fifth sacral vertebrae **Lateral:** ischial	**Transverse diameter (interspinous diameter):** distance between the ischial spines. Normally measures approximately 10 cm. Smallest diameter of the pelvis that the fetal presenting part must accommodate during the process of labor and birth. **Anteroposterior diameter:** extends from the middle of the inferior

	spines **Anterior:** inferior border of pubic symphysis	margin of the symphysis pubis through the middle of the transverse diameter and to the sacrum. This diameter is normally 11.5 cm or more.
Pelvic outlet: can be thought of as composed of two triangles, with the transverse diameter of the outlet serving as the common base of these two triangles	**Posterior:** sacrococcygeal joint **Lateral:** inner surface of ischial tuberosities **Anterior:** lower border of pubic symphysis	**Anteroposterior diameter:** extends from the middle of the inferior margin of the symphysis pubis to the sacrococcygeal joint. This measurement is normally 11.5 cm or more. **Transverse diameter (intertuberous, or biischial, diameter):** distance between the inner aspect of the lowermost part of ischial tuberosities. This diameter has a measurement of approximately 10 cm.

The descending rami of the pubic bones and the inferior margin of the symphysis pubis form what is known as the pubic arch. The angle of this arch should be at least 90°, as determined just below the symphysis pubis. An arch that is 90° a few centimeters below the symphysis pubis but narrow above that (just below the symphysis pubis) decreases the available space in the anteroposterior diameter of the pelvic outlet.

The inner aspect of the forepelvis (the anterior portion of the pelvis) should be rounded. A forepelvis that is not rounded but instead angles sharply toward the lateral portion of the pelvis decreases the oblique diameters of the inlet.

The pelvic sidewalls extend from the upper anterior angle of the sacrosciatic notch at the point of the widest transverse diameter of the pelvic inlet in a downward and forward line to the ischial tuberosities at the point of the widest transverse diameter of the pelvic outlet. They are normally slightly convergent in that, if the lines of their angles were extended beyond the pelvis, the two lines would meet at about the level of the knees; however, when palpated, they feel generally straight. The obstetric importance of the pelvic sidewalls hinges on the angle of the sidewalls. Divergence or convergence is based on whether the point of origin of the sidewall at the inlet and the ending point of the sidewall at the ischial tuberosities are essentially equidistant from the anteroposterior diameter of the pelvis. Convergent sidewalls usually decrease the angle of the pubic arch and may be accompanied by more prominent ischial spines. Divergent sidewalls always indicate a very wide angle of the pubic arch.

The shape and width of the sacrosciatic notch are important because they affect the posterior sagittal diameter of the inlet. This factor combines with the shape and rotation of the sacrum to determine the amount of room in the posterior portion of the pelvis for passage of the fetus.

Pelvic Types

Each plane of the pelvis has a shape, defined by the anterior–posterior and transverse diameters. These shapes were classified into four basic pelvic types by Caldwell and Moloy in X-ray studies done in the 1920s:[22] (1) gynecoid, (2) android, (3) anthropoid, and (4) platypelloid (**Table 10-3**). The pelvic types are determined by the shape of the pelvic inlet and the posterior characteristics of the pelvis, including the posterior sagittal diameter, which is the distance from the midpoint of the transverse diameter to the sacral promontory. Anatomic portions of the pelvis used in evaluation of pelvic types are the inlet, sacrum, sacrosciatic

notch, sidewalls, ischial spines, and pubic arch. Characteristics of these structures influence the obstetric capacity of the pelvis.

Table 10-3	Pelvic Types and Their Identifying Characteristics	
Pelvic Type and Description	**Identifying Characteristics**	**Image**
Gynecoid		
Commonly known as the “female pelvis” because it is the type that occurs most frequently in women; 41–42% of women’s pelves are gynecoid. This shape is optimal for childbearing.	*Inlet:* rounded. Transverse diameter approximately greater than or equal to the anteroposterior diameter. Posterior sagittal diameter only a little shorter than the anterior sagittal diameter. *Sacrum:* parallel with the symphysis pubis. *Sacrosciatic notch:* rounded with an approximate distance of 2½ to 3 fingerbreadths along the sacrospinous ligament. *Sidewalls:* straight pelvic sidewalls. *Ischial spines:* blunt and neither prominent nor encroaching. *Pubic arch:* a wide arch (≥ 90 degrees).	
Android		
Commonly known as the “male pelvis” because it occurs more frequently in men. Occurs in 32.5% of white women and in 15.7% of nonwhite women. The android pelvis poses difficulty for vaginal birth and increases the incidence of posterior position and forceps-assisted delivery. The midplane and outlet contracture of the android pelvis increases the incidence of fetopelvic disproportion and cesarean section.	*Inlet:* heart shaped. Posterior segment is wedge shaped; anterior segment (forepelvis) is narrow and triangular. The posterior sagittal diameter is short in comparison to the anterior sagittal diameter. Limited space in the posterior portion of the pelvis for accommodating the fetal head. *Sacrum:* anteriorly inclined and flat. *Sacrosciatic notch:* highly arched and narrow, with an approximate distance of 1½ to 2 fingerbreadths along the sacrospinous ligament. *Sidewalls:* pelvic sidewalls usually convergent. *Ischial spines:* prominent and frequently encroaching, thereby decreasing the transverse (interspinous) diameter of the midplane. *Pubic arch:* narrow, with an acute angle of much less than 90 degrees.	
Anthropoid		
The anthropoid pelvis is most common among women who are nonwhite, occurring in 40.5% of nonwhite women as compared to	*Inlet:* oval. The anteroposterior diameter is much larger than the	

23.5% of white women. The anthropoid pelvis had the longest sacrum of the four types of pelves and, therefore, is the pelvis with the most generous posterior portion. The potential problem of outlet contracture because of a narrow pubic arch is counterbalanced by the lengthy anteroposterior diameter, thus providing room in the posterior portion of the pelvis for the fetus. The shape of the anthropoid pelvis favors a posterior position of the fetus. It is adequate for vaginal birth if the dimensions overall are relatively large.	transverse diameter. The anterior segment of the pelvis (forepelvis) is pointed and narrower than the posterior segment. *Sacrum:* posteriorly inclined, so the posterior sagittal diameters are long throughout the pelvis. This allows more space in the posterior portion of the pelvis for accommodating the fetal head. *Sacrosciatic notch:* of average height but quite wide; has an approximate distance of 4 fingerbreadths along the sacrospinous ligament between the ischial spine and the sacrum. *Sidewalls:* somewhat convergent. *Ischial spines:* usually prominent but not encroaching. Transverse (interspinous) diameter of the midplane is generally less than that of the gynecoid pelvis but not as contracted as the android pelvis. *Pubic arch:* somewhat narrow.	
Platypelloid		
Platypelloid pelvis is rare and not particularly conducive to vaginal birth. It is the widest of all pelvic types and also shallow. These characteristics make rotation and descent of the fetal head difficult. This shape occurs in less than 3% of women in both white and nonwhite races.	*Inlet:* flat. Short anteroposterior diameter and a wide transverse diameter. The anterior segment of the pelvis (forepelvis) is quite wide. *Sacrum:* inclined posteriorly and quite hollow. *Sacrosciatic notch:* wide and flat, with an acute angle between the ischial spines and the sacrum. *Sidewalls:* slightly convergent. *Ischial spines:* somewhat prominent but, because of wide transverse diameters throughout the pelvis, this prominence has no effect. *Pubic arch:* quite wide. This pelvis is the widest of all the pelvic types.	

Caldwell and Moloy went on to characterize the labor outcomes associated with each type of pelvis. They generated essential knowledge of the mechanisms of labor as well as information about how the position and diameter of the fetal head affects the progress of labor.[22] This information is a critical component of the assessment of fetal descent during labor.

The pelves of most women are not pure types, but rather a mixture of types. By convention, the pelvis is named for the posterior characteristics with a *tendency* for the anterior characteristics. For example, a pelvis with a curved sacrum and wide sacrosciatic notch may have slightly convergent sidewalls that narrow the anterior segment of the midpelvis and

outlet. Such a pelvis would be described as a gynecoid pelvis with android tendencies.

The Menstrual Cycle: An Introduction

"Menstruation" is a term derived from the Latin word *mensis*, meaning "month." In several cultures, if not most, this single biological event engenders negative connotations. According to the *British Medical Journal* in 1878, "it is undoubtedly the fact, that meat will be tainted if cured by women at the catamenial [*sic* menstrual] period."[23] In today's English language, euphemisms for the event abound, including "the curse" and "being sick this month." In the Polynesian language, the word "taboo" is synonymous with "menstruation."[24] Even today, national advertisements for sanitary products continue to avoid using the term "menstruation."

Much still remains unknown about the menstrual cycle, including the basic question of the role of menstruation for a healthy woman. Although human females usually menstruate monthly, some other primates do not; many other mammals have an estrus cycle that includes ovulation and reabsorption instead of menstruation. Some scientists, such as the evolutionary biologist Profit, have postulated that menstruation is useful to a woman because it expels bacteria from the body.[25] Others question whether menstruation has any benefits and instead should be considered obsolete. These scientists are among those who advocate that the average woman who wants contraception should use regular hormonal suppression.[26]

The physiology of the menstrual cycle is a sophisticated process. Over the last few decades, scientific discoveries have revealed more information about the interplay between functions of the female organs and the hormonal milieu. As additional discoveries have been made, understanding the process has become more complicated.

As part of understanding the menstrual cycle, a few assumptions are made. Predominant among them is that every woman has a 28-day cycle and is ovulatory. Neither of these "facts" is necessarily true. The normal menstrual cycle can be 24 to 35 days in duration and can be more variable in the first 5 to 7 years after menarche and in the last 10 years before menopause.

The menstrual cycle has two phases: The follicular phase starts with the onset of menses and ends on the day before the luteinizing hormone surge occurs. The luteal phase begins on the day of the luteinizing hormone surge and ends at the onset of menses. For the purpose of explanation, the menstrual cycle events are initially separated and presented in a set time sequence. Nevertheless, it must be recognized this type of presentation is an artificial convention. The discussion of the menstrual cycle presented in this chapter divides the physiology into several sections, beginning with a discussion of the steroidogenesis and hypothalamic–pituitary–ovarian axis. Next the anatomy of the ovarian cycle with associated physiology and anatomic developments is reviewed, and finally the endometrial cycle is presented.

Steroidogenesis

A number of hormones exist in the human body, which generally are one of three different types: amines, prostaglandins, or steroid hormones. Sex hormones, also termed gonadocorticoids, are steroid hormones. All sex steroid hormones are synthesized from cholesterol through a series of chemical reactions known as steroidogenesis. Sex steroids are commonly identified as estrogen, progesterone, and androgen. The major site of biosynthesis for all sex steroid hormones in healthy ovulating women is the ovary. Other peripheral sites, including adiposity and skeletal muscle, produce small amounts of these hormones.

All of the sex hormones are derived from the 27-carbon-chain cholesterol. From the 27 carbons, a mitochondrial enzyme called P450 scc ("scc" signifies cholesterol side-chain cleavage) catalyzes conversion of cholesterol to pregnenolone. Pregnenolone is the precursor from which 21-carbon-chain progestogens and corticoids emerge. Further chemical reactions reduce the carbons from hydroxypregnenolone to 19-carbon-chain androstenedione, the precursor from which androgens, including testosterone, are derived. Aromatization further reduces the 19 carbons to the 18-carbon-chain estranes, which include all of the estrogens[27,28] (**Figure 10-12**). In summary, steroidogenesis is an exquisitely efficient method by which all sex hormones are derived in a few chemical steps by starting with cholesterol and reducing the carbons in cholesterol from 27 to 18 in number.

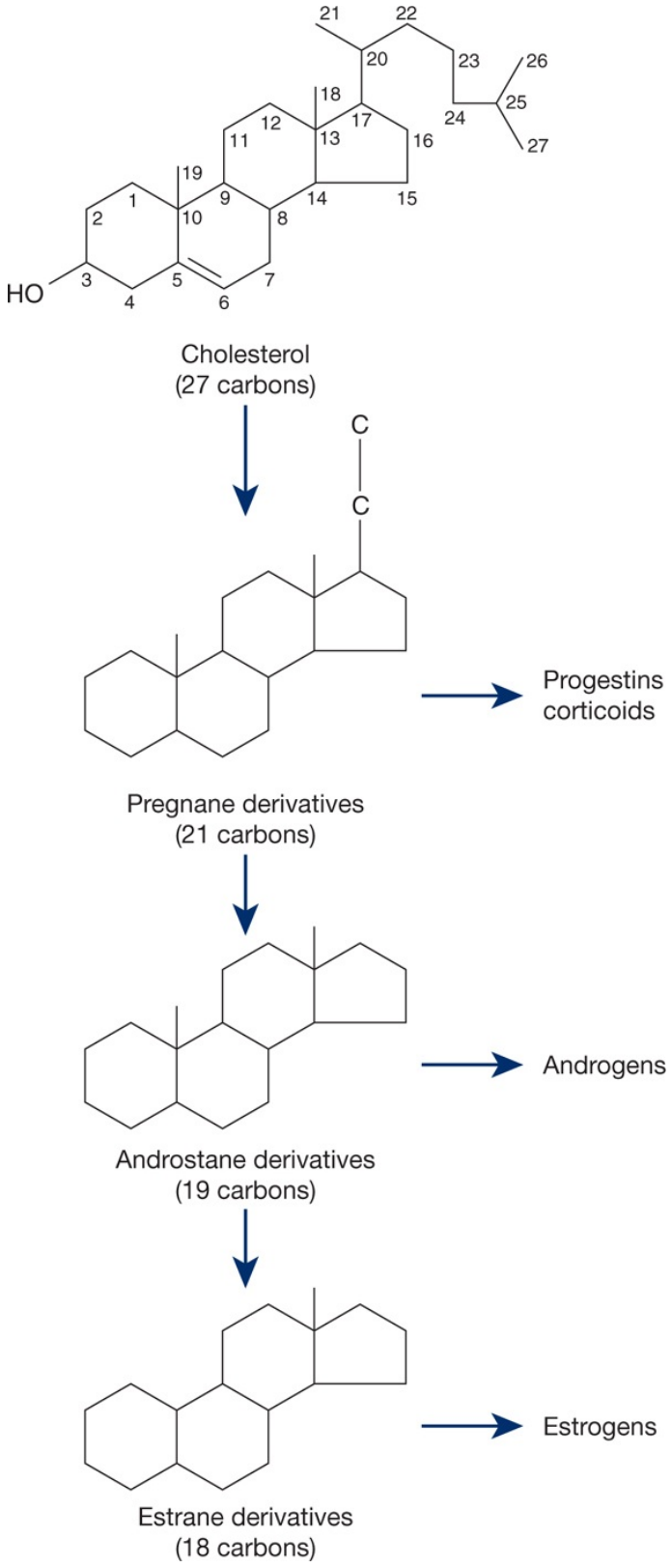


Figure 10-12 Biosynthesis of sex steroids.

Reproduced with permission from Brucker MC, Likis FE. Steroid hormones. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:355-376.[27]

Steroidogenesis does not occur without interaction with the gonadotropins, follicle-

stimulating hormone (FSH), luteinizing hormone (LH), and normal reproductive organs. Production of FSH and LH is part of the hypothalamic–pituitary–ovarian axis, as illustrated in **Figure 10-13**.

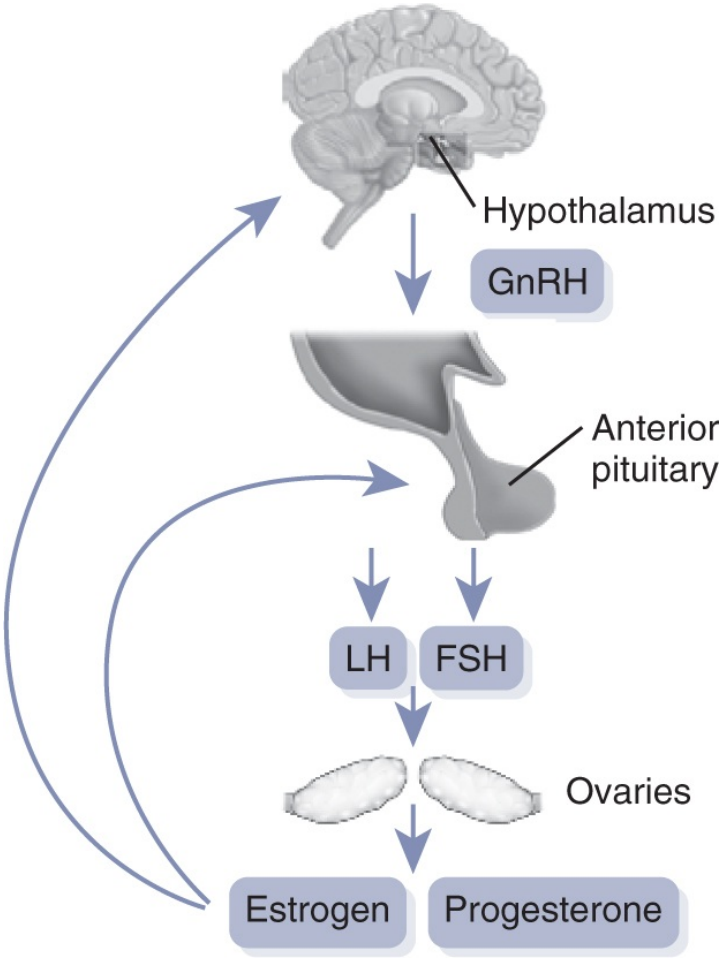


Figure 10-13 The hypothalamic–pituitary–ovarian axis.

Abbreviations: FSH, follicle-stimulating hormone; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone.

The Hypothalamic–Pituitary–Ovarian Axis

"Hypothalamic–pituitary–ovarian axis" is the term used when referring to the effects created by these individual endocrine organs when they work in concert as if they were a single endocrine organ. As will be described, the interplay between the hypothalamus, pituitary, and ovary is controlled through feedback loops.

The hypothalamus controls the reproductive system by secreting, in a pulsatile fashion, either releasing or inhibitory factors into the hypothalamic–pituitary portal system.[29] These factors then act on the anterior pituitary gland. The hypothalamus releasing factors that control reproduction include follicle-stimulating releasing factor (FRF), luteinizing hormone releasing factor (LRF), and prolactin-inhibiting factor (PIF). The releasing factors FRF and LRF also are collectively referred to as gonadotropin-releasing factors.

The pituitary gland is composed of two main lobes: the adenohypophysis and the neurohypophysis. When stimulated by hypothalamic releasing hormones, the cells of the adenohypophysis secrete the gonadotropins FSH and LH as well as prolactin. After their release, LH and FSH travel via the circulation to the ovary. A woman's ovaries are the site of production of estrogen and progesterone from either the ovarian theca or granulosa cells.

Control of the Hypothalamic–Pituitary–Ovarian Axis Through Feedback Loops

A feedback system is a method in which factors either inhibit or stimulate production of a specific agent. Positive feedback indicates stimulation, whereas negative feedback is inhibitory. It was not long ago that the feedback system for the hypothalamic–pituitary–ovarian axis was depicted as a simple graph, which was then used as the universal prototype to illustrate negative feedback in basic science classes. However, this simple diagram failed to account for several factors found in a woman's body. For example, gonadotropin-releasing hormone (GnRH) is not static but rather pulsatile, with its release amplitude changing as the result of feedback modulation of steroids and peptides that originate in the dominant follicle and act directly on the hypothalamus and anterior pituitary.

Also, although the hypothalamic–pituitary–ovarian axis sometimes does function as a negative feedback system, it does not exclusively fill this function, nor are estrogen and progesterone the only agents of note that are involved in the hypothalamic–pituitary–ovarian axis. Some agents stimulate the hypothalamus *and* the pituitary, others stimulate neither, and still others stimulate one but not the other. For example, *low* levels of estrogen *inhibit* LH, whereas *high* levels of the same hormone *stimulate* LH.

Among the factors that influence feedback are three specific peptides—inhibin, activin, and follistatin—that are synthesized by ovarian granulosa cells in response to FSH stimulation and secreted into the follicular fluid. Inhibin has two different subtypes: Inhibin A and Inhibin B. As the name implies, both inhibit FSH secretion. This peptide also augments LH stimulation of theca cells to accelerate production of androgens, and it diminishes actions of GnRH. In contrast, activin simulates FSH and diminishes progesterone production from the granulosa cells. It is hypothesized that follistatin, the third major peptide, may inactivate activin by binding it, thereby decreasing FSH activity.

Other factors are also known to have important actions on ovarian function. For example, the ovary is a site of production for insulin-like growth factor 1 (IGF-1), which acts independently of most other factors. This growth factor also amplifies androgen production in ovarian theca cells and FSH action in granulosa cells. In general, FSH has multiple activities in the granulosa cells, with all being influenced by growth factors—of which IGF-II is theorized to be the most important.[30]

In summary, no simple feedback loop exists that describes the entire menstrual cycle. The menstrual cycle is known to have a potent negative feedback system. Importantly, at different times of the cycle, a positive feedback process may be seen as well. In general, as more factors influencing the hypothalamic–pituitary–ovarian axis feedback loop are discovered, there is greater acknowledgment that much remains unknown.

The Ovarian Cycle

Some midwives like to remind a woman that if she is pregnant with a daughter, she also is carrying the eggs for her grandchildren. The intrauterine daughter, like all women, has the largest number of primary oocytes when she is a fetus of approximately 20 to 28 weeks' gestational age. The number of primary oocytes decreases from millions to thousands by the time of puberty via atresia. The mechanism of atresia itself is not well understood.

After establishment of menses, meiosis occurs monthly as part of ovum maturation. During this process, the primary oocyte, which originally had 46 chromosomes, splits into a secondary oocyte consisting of 23 chromosomes and a polar body. The latter usually disappears and is reabsorbed. Granulosa cells surround each secondary oocyte, and the surrounded oocyte is called a primordial follicle (**Figure 10-14**). Follicles are scattered throughout the cortical zone of the ovary.

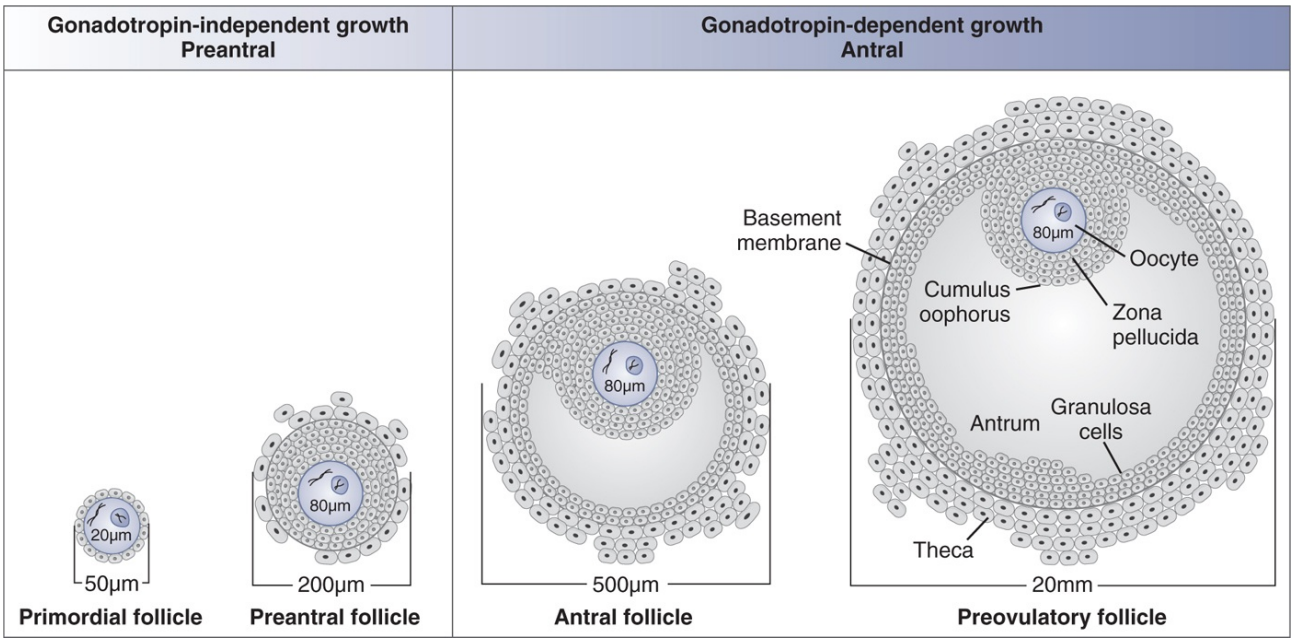


Figure 10-14 Follicular development.

During her reproductive life, a woman's ovaries tend to be in a constant state of change. These changes are characterized by the ovarian cycle within the menstrual cycle. The ovarian cycle is subdivided into two major phases—follicular and luteal phases—with events such as ovulation and menstruation identified separately. Because hormones are receptor specific, the cells within the ovary contain receptor sites that are essential to normal ovarian function and a normal menstrual cycle. As the various ovarian changes occur, receptors on ovarian cells appear and interface with the feedback system.

Follicular Phase of Ovarian Cycle

The follicular phase varies in length but averages 10 to 14 days. This phase of the ovarian cycle produces an ovum for fertilization.

Follicles can be found in an ovary at different stages of maturation. Groups of immature

ovarian follicles develop over time. Taking more than a year on average, these primordial follicles grow until one major follicle matures and the others regress. As early as menstrual cycle day 5, one follicle out of a group begins to emerge as dominant. It remains unknown why one follicle is chosen and others regress to stromal tissue, but local growth factors are theorized to be involved. During the dominant follicle's development, it has an increased number of FSH receptors compared to other follicles, enabling production of its own estrogenic environment. As estrogen levels in the circulation increase secondary to follicular production, a concomitant decrease in FSH secretion occurs—an action that may seal the fate of those follicles unable to produce an estrogenic milieu. Simultaneously, theca cells grow rapidly in the ovary. Perhaps in response to the high estrogen level, the granulosa cells of the dominant follicles begin to develop LH receptors.

Preantral Follicle

As a primordial follicle grows, a layer of theca cells forms around the granulosa cells, with a basement membrane separating the two groups. This follicle then is known as a preantral follicle. Under the influence of FSH, the granulosa cells surrounding the maturing follicle proliferate and begin to produce androgens and estrogens, especially the latter. FSH receptors appear on the preantral granulosa cells. Using energy from cyclic adenosine monophosphate (cAMP), the cell is able to convert, though aromatization, androgens into estrogens as described in the discussion of steroidogenesis. Simultaneously, the theca cells, which have LH receptors, use cAMP to produce the androgens. In the preantral follicle, FSH and estrogen promote the development of more FSH receptors.

Although the follicular cycle usually progresses normally, in some women large amounts of androgens are produced. When this occurs, the granulosa cells cannot compensate for these high levels of androgens and convert the androgen into estrogen. In such a case, eventually the follicle regresses secondarily to atresia. This phenomenon is noted to occur among women with polycystic ovary syndrome, a topic discussed in more detail in the *Gynecologic Disorders* chapter.

Antral Follicle

Eventually a cavity, called the antrum, forms in the intercellular spaces of the granulosa cells. The appearance of the antrum is pathognomonic (i.e., clearly indicative) of the antral follicle. The antrum is filled with fluid that is heavily estrogenic in nature. Like the preantral follicle, the antral follicle continues to have LH receptors exclusively on theca cells and FSH receptors only on the granulosa cells.

Preovulatory Follicle

The outer layer of the follicle becomes somewhat translucent, at which point it is termed the zona pellucida. The zona pellucida then is surrounded by more granulosa cells, also known as the corona radiate. The zona pellucida is attached to the main body of the follicle by a mass of granulosa cells, called the "egg cloud" or cumulus oophorus. When these changes become

apparent, this follicle is called the preovulatory follicle or the Graafian follicle. By day 12 or 13 of a menstrual cycle, the oocyte is floating in the antrum; by cycle day 14, the mature or Graafian follicle is protruding from the ovary. Even before ovulation occurs, the granulosa cells begin to increase in size and assume a yellow shading of lutein as the LH receptors appear on the cells.

As illustrated in **Figure 10-15**, the hormones estrogen and LH are intimately related to each other. Approximately 24 to 36 hours before ovulation, estrogen levels peak, priming the cycle for the LH surge. Some researchers have found that women athletes are more likely to sustain injuries when estrogen is high during this time and theorize this propensity is related to collagen metabolism, a process influenced by estrogen.[31] At this time, androgen levels also begin to increase, perhaps in an evolutionary-based effort to stimulate the libido.[32]

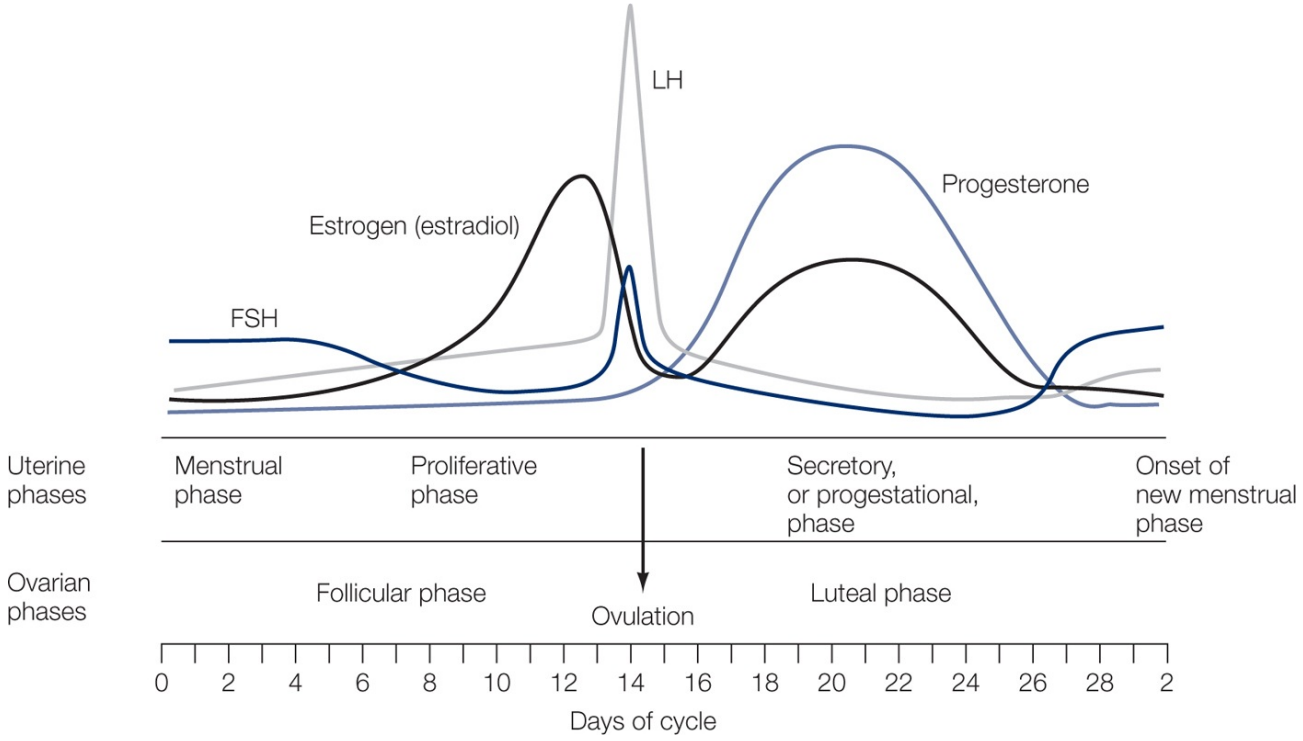


Figure 10-15 Hormonal changes throughout the menstrual cycle.

Abbreviations: FSH, follicle-stimulating hormone; LH, luteinizing hormone.

Ovulation

Ovulation, as defined by release of the ovum, usually occurs 10 to 12 hours after the LH peak and 24 to 36 hours after the estrogen surge. No one single event causes ovulation. The oocyte is released with the cumulus oophorus and antral fluid. The fimbria of the ovary sweeps by and catches the ovum within the fallopian tube. Some women experience discomfort, known as mittelschmerz, at this time. The remaining follicular cells (granulosa and theca cells) in the ovary are transformed into a structure called the corpus luteum (yellow body), which grows for at least a week and forms a mature corpus luteum. If fertilization does not occur, the corpus luteum begins to degenerate into the corpus albicans.

The LH surge is the most reliable single indicator of ovulation.[33] Likely to last for 48 hours or longer, it promotes luteinization of the granulosa cells in the follicle and the synthesis of prostaglandins and other eicosanoids needed for follicle rupture. Progesterone levels continue to rise with the LH surge and may, with the FSH peak that accompanies the peak of LH, be part of the termination of the event. Low levels of progesterone contribute to LH and FSH peaks, whereas high levels can prevent LH surge. Progesterone works with prostaglandins to enhance digestion of the follicle's wall, thereby promoting ovulation. High levels of intrafollicular prostaglandins may cause the oocyte to be retained. These physiologic events form the basis for some theories regarding the decrease in ovarian cancer associated with use of oral contraceptives. Appropriate surges mean nothing if the dominant follicle is not present, and without the mature preovulatory follicle it is likely that the surges may not occur. These kinds of complex interdependencies characterize ovarian cycle processes.

Other physiologic events accompany ovulation. For example, under the influence of estrogen in the mid- to late follicular phase, the cervical mucus becomes clear, thin, and more profuse; the cervix softens; and the external os opens slightly. These changes in cervical mucus facilitate sperm transport. After ovulation, higher progesterone levels lead to the production of thick cervical mucus, which inhibits the entry of sperm into the uterus.

Luteal Phase

A luteinized granulosa cell is able to produce both estradiol and progesterone. An older, and less nuanced, description of the menstrual cycle implied that progesterone was produced only after ovulation. Increases in progesterone levels may be noted as early as day 10 of the cycle, although progesterone production rises sharply after ovulation and peaks at 8 days after the LH surge. Progesterone suppresses new follicular growth.

Whereas the length of the follicular phase of the ovarian cycle may vary, the duration of the luteal phase essentially remains constant at 14 days. This length of time is directly related to corpus luteum longevity, assuming no interference by conception. In the luteal phase, estrogen is still produced and may facilitate destruction of the corpus luteum. Because progesterone is associated with an elevation in basal body temperature (BBT), daily measurement of BBT will demonstrate a rise *after* ovulation has occurred. This makes assessment of thermal activity useful as a post hoc measure and as the basis of some natural family planning methods. A variety of other factors, including oxytocin, are likely to be involved in this period.

Luteal–Follicular Transition Period

During the luteal–follicular transition, the corpus luteum dies and estrogen, progesterone, and inhibin levels decrease such that there is transitional time between one cycle and the next. Because production of inhibin A drops, FSH levels begin to increase. Conversely, because estrogen and progesterone levels are low, GnRH pulses increase, which in turn facilitates production and release of FSH and LH from the hypothalamus and pituitary. FSH helps a group of follicles to be rescued from atresia, and the cycle to begin anew.

The Endometrial Cycle

Many sources subdivide the endometrial cycle into two periods: proliferative and secretory. Other authorities note that ischemic changes occur at the end of the secretory phase, with this ischemic period being designated as a separate phase. Still other sources identify menses as a first phase of the endometrial menstrual cycle (**Figure 10-16**).

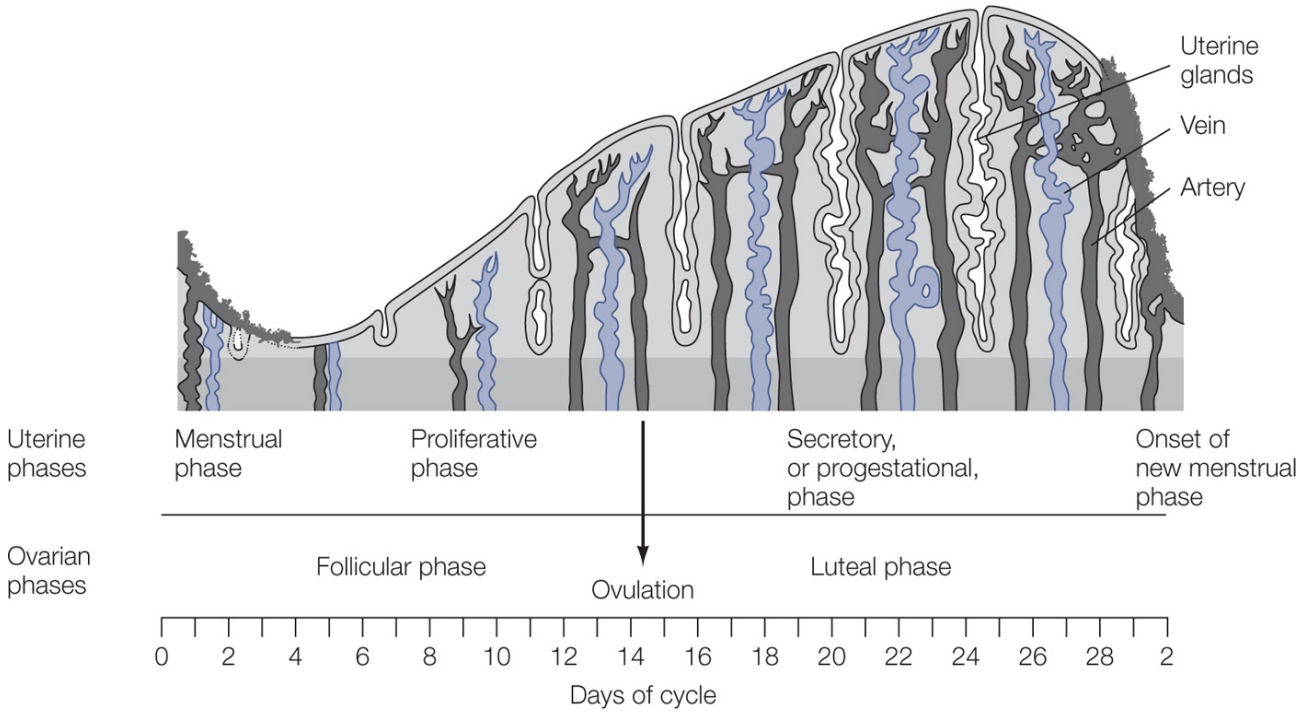


Figure 10-16 The endometrial cycle.

Proliferative Phase

The proliferative phase of the endometrial cycle corresponds to the follicular phase of the ovarian cycle. During this time the endometrial tissue develops under the increasing influence of estrogen. Endometrial glands change from being narrow and tubular to being enlarged to an extent that they link to each other, forming a continuous lining.

In rare situations, such as during unopposed treatment with exogenous estrogen or among women with anovulation, a proliferative endometrium becomes persistent. When this occurs, there is a higher risk that endometrial hyperplasia will ensue; this state is associated with endometrial cancer. These conditions are discussed in more detail in the *Menstrual Cycle Abnormalities* chapter.

Secretory Phase

The rising progesterone levels that occur with ovulation have a major influence on the endometrium, turning a proliferative endometrium into a secretory one. The length of the endometrium does not regress, but the appearance of the tissue does. Notably, the coiling of spiral vessels and tortuosity of the glands increase. The stroma become edematous and at the

time of implantation, approximately days 21–27 of the cycle, three distinct zones have appeared: the stratum spongiosum (the most superficial layer), the stratum compactum (midportion), and the stratum basale (the innermost layer). Once an endometrium becomes secretory, the risks associated with persistent proliferative endometrium are diminished. Since the discovery that exogenous progesterone facilitates development of a secretory endometrium, it has become standard practice for women on perimenopausal hormone therapy to also take progesterone as an adjunctive treatment with the estrogen.

Ischemic Period

By approximately cycle day 25, if implantation does not occur, the decreasing levels of estrogen and progesterone result in disorganization of the endometrium. The tissue shrinks, venous drainage diminishes, and blood flow within the spiral arteries is compromised, resulting in rhythmic constriction and relaxation. The process of apoptosis transpires and the uterus is ready for the next step—menses.

Menstruation

As the lining of the uterus weakens, blood escapes into the endometrial cavity. The appearance of blood for the woman marks the first day of her next menstrual period. The consistency of bleeding after the withdrawal of estrogen and progesterone is indicative of a normal menstrual cycle with functional organs and endocrine system.

Investigation of amenorrhea often involves a challenge test in which women are given progesterone to ascertain whether, after completion of the dose, withdrawal bleeding occurs. If so, the assumption is made that there is no end-organ disorder and there is adequate endogenous estrogen. More information about this testing is found in the *Menstrual Cycle Abnormalities* chapter.

Considering that menstruation is a normal event experienced daily by millions of women around the world, it is somewhat surprising that much remains unknown about the physiology of this event. It is a testament to the remarkable innate capacities of the female body that in spite of the complexity of the menstrual cycle, the vast majority of women's menses occur on a regular basis without major mishap. **Figure 10-17** depicts the full menstrual cycle, including the ovarian, hormonal, and uterine cycles, in one illustration.[34] **Table 10-4** provides an overview of the menstrual cycle, including sequencing of the major events.

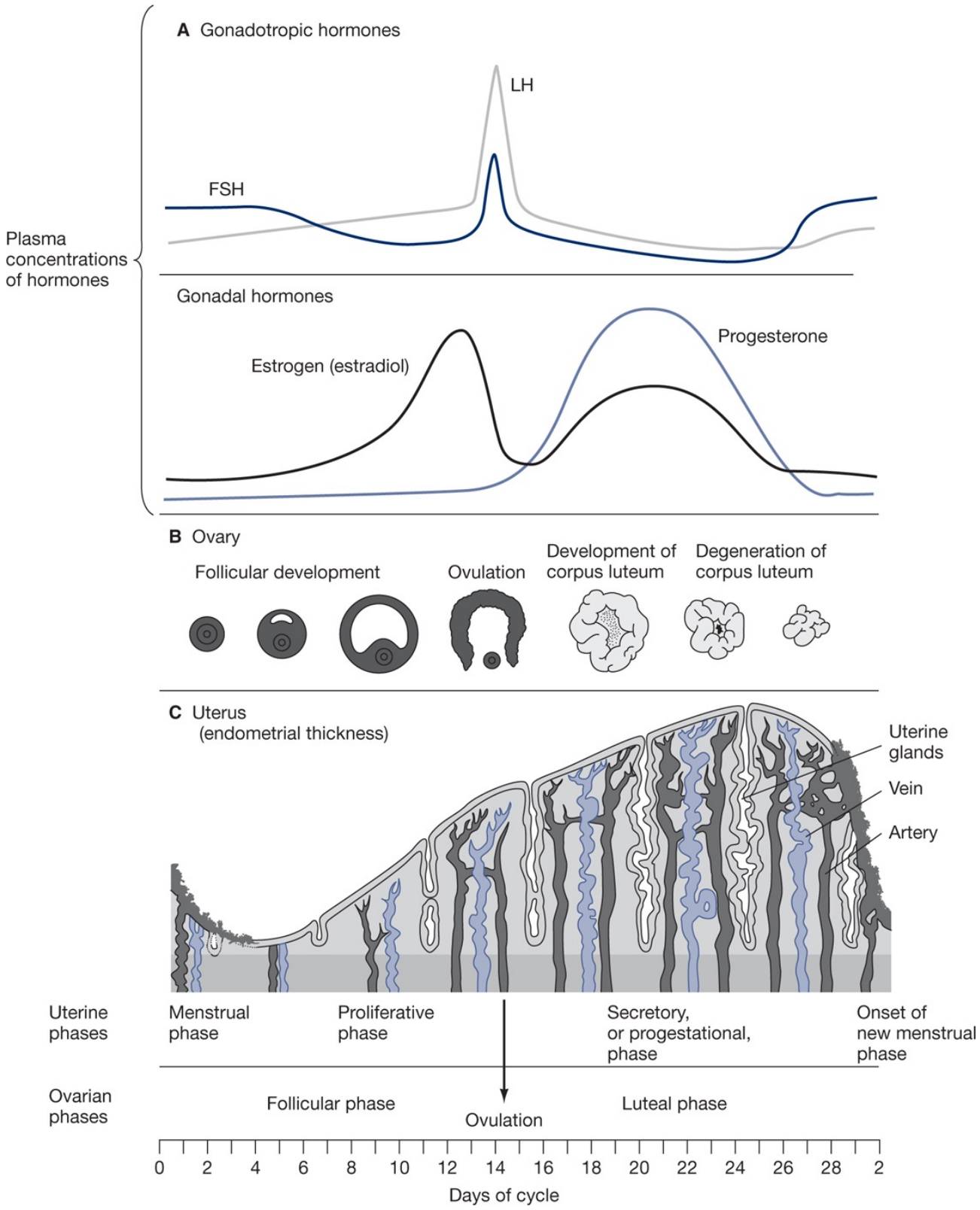


Figure 10-17 The menstrual cycle. **A.** Hormonal cycles. **B.** The ovarian cycle. **C.** The uterine (endometrial) cycle.

Abbreviations: FSH, follicle-stimulating hormone; LH, luteinizing hormone.

Reproduced with permission from Chiras DD. *Human Biology.* 9th ed. Burlington MA: Jones & Bartlett Learning; 2019.[34]

Table 10-4 Overview of the Menstrual Cycle

Days	Ovarian Cycle	Endometrial Cycle	Estrogen and Progesterone	Actions
1–5	Follicular	Menstrual	Low estrogen and progesterone	Inhibin falls. LH and FSH begin to slowly rise, starting on approximately day 27.
6–14	Follicular	Proliferative	Building estrogen (and, to a lesser extent, progesterone)	LH initiates luteinization and progesterone production in the granulosa layer. The rising level of progesterone facilitates the positive feedback action of estrogen and may be necessary for the midcycle FSH peak, although the complete reason for the midcycle FSH peak remains unclear. Estrogen production becomes sufficient to achieve and maintain the threshold concentration of estradiol that is required to induce the LH surge. A midcycle increase in local and peripheral androgens occurs, derived from the thecal tissue of unsuccessful follicles. A few hours after the LH surge, changes in hormone levels release suppression of oocyte maturation.
14	Ovulation	Proliferative	High estrogen and building progesterone	High levels of estrogen induce the LH surge at midcycle and lead to sustained elevated LH secretion. Suppression of inhibition of oocyte maturation allows final maturation of the oocyte. The LH surge occurs and is responsible for luteinization of the granulosa, and synthesis of progesterones and prostaglandins in the follicle. Progesterone and prostaglandins work together to digest, weaken, and eventually rupture the follicular wall. The midcycle FSH peak frees the oocyte from follicular attachments and ensures sufficient LH receptors to allow an adequate normal luteal phase.
15–26	Luteal	Secretory (implantation phase)	Progesterone most dominant, but estrogen present	Progesterone from the corpus luteum acts both centrally and within the ovary to suppress new follicular growth. High levels of progesterone inhibit pituitary secretion of gonadotropins by inhibiting GnRH pulses at the level of the hypothalamus. High levels of progesterone antagonize the pituitary response to GnRH by interfering with estrogen action. Regression of the corpus luteum may involve luteolytic action of its own estrogen production, mediated by an alteration in local prostaglandin concentration. In early pregnancy, hCG maintains luteal function until placental steroidogenesis is established.
27–28	Luteal	Late secretory (ischemic)	Dropping estrogen and progesterone	The degeneration of corpus luteum results in low levels of estrogen, progesterone, and inhibin. The low level of inhibin removes the suppression of FSH on the pituitary. The low level of estrogen and progesterone allows an increase in GnRH pulsatile secretion and the removal of the pituitary from the negative feedback suppression. The removal of inhibin and estradiol and increased GnRH pulses allow an increase in FSH (and to lesser extent LH). The increasing FSH is instrumental in the maturation of a dominant follicle.

Abbreviations: FSH, follicle-stimulating hormone; GnRH, gonadotropin-releasing hormone; hCG, human chorionic gonadotropin; LH, luteinizing hormone.

Reproduced with permission from Brucker MC, Likis FE. Steroid hormones. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:355-376.[27]

Female Sexual Response

The female sexual response is a complex interaction between mental processes and physical stimulation that has an infinite number of individual variations. The term “sexarche” designates the age of initial sexual intercourse although sexual activity can include many different expressions. Although this section describes the physiologic response, it is important to recognize that individual sexuality and individual sexual responses cannot be adequately described by knowing the basic physiology of the female sexual response. The entire areas of human sexuality and behavior have not been the subject of significant scientific research.

Sexual activity may have many components. From a physiological standpoint, the female sexual response has four primary phases, as shown in **Table 10-5**.[35,36] This model was originally developed by Masters and Johnson, who labeled the four phases as excitement, plateau, orgasm, and resolution.[35] A newer model developed by Basson et al., which incorporates emotional, cognitive, and psychological factors, has expanded understanding and provides a more detailed template for elucidating this complex phenomenon (**Figure 10-18**).[37] Notably, female orgasm is considered just one aspect of the female sexual response. The four phases outlined in Table 10-5 may overlap, repeat, or be absent. In addition, women do not always progress through these phases in the same sequence.[38]

Table 10-5 **Model for Female Sexual Response**

Stage	Description
Desire	Desire to have sexual activity; sexual thoughts and images present.
	Vaginal lubrication.
	Inner two-thirds of vagina expands, and vagina becomes lubricated. The cervix and uterus are pulled forward; the labia majora become flatter and, in parous women, increase in size. The labia minora enlarge.
	Blood flow to genitals increases and the erectile tissue of the clitoris expands.
	Nipples become erect and breasts slightly increase in size.
	Some increase in heart rate, respiratory rate, blood pressure, and muscle tension occurs.
	Vasocongestion of skin, called “sex flush,” occurs in 50–75% of women.
Arousal	Sense of sexual pleasure with physiologic changes.
	The inner-two thirds of the vagina expands as the uterus is elevated (called tenting). The outer third of the vagina becomes congested and the opening narrows.
	The labia minora and labia majora become engorged and enlarged.
	The clitoris withdraws into the clitoral hood.
	The breasts enlarge and blood flow to areola increases.
	A blotchy skin pattern may be seen as sex flush.
	Further increases occur in heart rate, respiratory rate, and blood pressure.
Orgasm	Peaking of sexual pleasure.
	Rhythmic muscular contractions of the uterus, outer third of the vagina, anal sphincter, and clitoris occur.
	Heart rate, respiratory rate, and blood pressure are at their highest values.
	Other muscles, such as those in the buttocks or feet, may involuntarily contract.

Resolution	Sense of well-being.
	If continued sexual stimulation and interest, multiorgasmic response is possible; if not, the organs return to their unaroused state.
	Vasocongestion dissipates; the uterus, vagina, and clitoris return to their normal size and position.
	The breasts decrease in size; the areola and nipples flatten.
	Breathing, heart rate, and blood pressure return to normal.

Based on Masters WH, Johnson VE. *Human Sexual Response.* New York, NY: Bantam; 1966[35]; American Psychiatric Association. Sexual dysfunctions. In: *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013:423-450.[36]

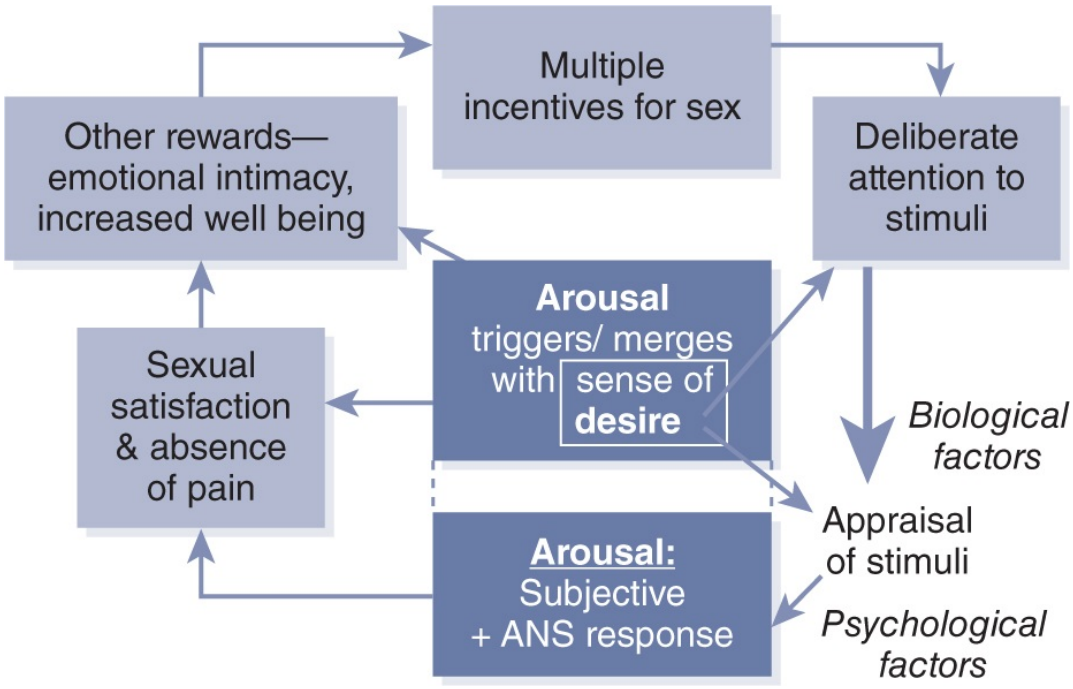


Figure 10-18 Human sexual response is depicted as a motivation/incentive-based cycle of overlapping phases of variable order. A sense of desire may or may not be present initially; it can be triggered alongside the sexual arousal resulting from attending to sexual stimuli. Sexual arousal comprises subjective and physical responses. Psychologic and biologic factors influence the brain's appraisal of the sexual stimuli. The sexual and nonsexual outcomes influence present and future sexual motivation.

Abbreviation: ANS, autonomic nervous system.
Reproduced with permission from Basson R. Human sexual response. *Handbook Clin Neurol.* 2015;130:11-18.[37]

Orgasm may be stimulated by vaginal penetration or clitoral stimulation, although it may also occur without these actions. In general, clitoral stimulation is the most common source of physical stimulation for initiation of orgasm. One interesting controversy about the anatomy of the female sexual response is the "G-spot," which is a presumed anatomic collection of nerves in the anterior vagina that is the source of vaginally stimulated orgasm. Subsequent anatomic and physiologic studies have not confirmed that this spot exists.[38,39]

Orgasm is regulated by both sensory and autonomic nerves. It starts when the pudendal nerve transmits information from the clitoris and external genitalia; this is followed by increased blood flow to the area. A reflex wave of smooth and skeletal muscle contractions then occurs in the vagina, urethra, anus, and uterus.

Estrogen—in particular, estradiol—is the primary hormone that mediates the female sexual response, but the roles of the hormones of import are not fully understood. However, the lower levels of estradiol that occur following menopause are the etiology of vaginal atrophy, as well as difficulty with vasoconstriction and vaginal lubrication. These changes associated with aging can lead to diminished sexual interest, arousal, and orgasm.[40]

Conclusion

This chapter presents a brief overview of the female reproductive anatomy and physiology. Midwives who will care for women with specific disorders such as pelvic floor dysfunction, sexual dysfunction, or menstrual disorders will need additional detailed knowledge about how the anatomy and physiology of these organs affect a woman's health. Some resources for reviewing these topics are included in the Resources section.

Resources

Organization	Description	Webpage
Bartleby.com	*Gray's Anatomy of the Human Body* online; free access to the book and illustrations	http://www.bartleby.com/107/
Association of Reproductive Health Professionals (ARHP)	Female sexual response	http://www.arhp.org/publications-and-resources/clinical-fact-sheets/female-sexual-response

References

1. Clemente CD. *Anatomy: A Regional Atlas of the Human Body*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.
2. Geddes DT. Inside the lactating breast: the latest anatomy research. *J Midwifery Womens Health*. 2007;52:556-563.
3. Ramsay DT, Kent JC, Hartmann RA, Hartman PE. Anatomy of the human breast redefined with ultrasound imaging. *J Anat*. 2005;206:525-534.
4. Love RM, Barsky SH. Anatomy of the nipple and breast ducts revisited. *Cancer*. 2004;101:1947-1957.
5. Howard BA, Gusterson BA. Human breast development. *J Mammary Gland Biol Neoplasia*. 2000;5(2):120-137.
6. Pauls RN. Anatomy of the clitoris and the female sexual response. *Clin Anat*. 2015;28:376-384.
7. Hayes D, Clark NR. Gynecologic anatomy and physiology. In Schuiling KD, Likis FE, eds. *Women's Gynecologic Health*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017:77-93.
8. Moore, KL, Persaud TVN, Torchia MG. *The Developing Human: Clinically Oriented Embryology*. 10th ed. Philadelphia, PA: Elsevier; 2016.
9. Sivridis E, Giatromanolaki A. Proliferative activity in the postmenopausal endometrium: the lurking potential for giving rise to an endometrial carcinoma. *J Clin Pathol*. 2004;57(8):840-844.
10. Deveduex D, Marque C, Masour S, Germain G, Duchêne J. Uterine electromyography: a critical review. *Am J Obstet Gynecol*. 1993;169(6):1636-1653.
11. Vink JY, Qin S, Brock CO, et al. A new paradigm for the role of smooth muscle cells in the human cervix. *Am J Obstet Gynecol*. 2016;215(4):478.e1-478.e11.
12. Nott JP, Bonney EA, Pickering JD, Simpson NAB. The structure and function of the cervix during pregnancy. *Translat Res Anatom*. 2016;2:1-7.
13. Rimon-Dahari N, Yerushalmi-Heinemann L, Alyagor L, Dekel N. Ovarian folliculogenesis. *Results Probl Cell Differ*. 2016;58:167-190.
14. Jørgensen A, Rajpert-De Meyts E. Regulation of meiotic entry and gonadal sex differentiation in the human: normal and disrupted signaling. *Biomol Concepts*. 2014;5(4):331-341.
15. Gilbert SF. The saga of the germ line. In: *Developmental Biology*. 6th ed. Sunderland, MA: Sinaur Associates; 2000. Available at: http://www.ncbi.nlm.nih.gov/books/NBK10008. Accessed October 1, 2012.
16. Huseynov A, Zollikofer CP, Coudyzer W, et al. Developmental evidence for obstetric adaptation of the human female pelvis. *Proc Natl Acad Sci USA*. 2016;113(19):5227-5232.
17. Ritchie JR. Orthopedic considerations during pregnancy. *Clin Obstet Gynecol*. 2003;46(2);456-466.
18. Chermansky CJ, Moalli PA. Role of pelvic floor in lower urinary tract function. *Auton Neurosci*. 2016;200:43-48.
19. Ashton-Miller JA, DeLancey JOL. Functional anatomy of the female pelvic floor. *Ann NY Acad Sci*. 2007;1101:266-296.
20. Stoker J. Anorectal and pelvic floor anatomy. *Best Pract Res Clin Gastroenterol*. 2009;23:463-475.
21. Barleben A, Mills S. Anorectal anatomy and physiology. *Surg Clin North Am*. 2010;90:1-15.
22. Caldwell WE, Moloy HC. Anatomical variations in the female pelvis: their classification and obstetrical significance. *Proc R Soc Med*. 1938;32(1):1-30.
23. Story W. Menstruation and the curing of meat. *Br Med J*. 1878;1(904):633.
24. Mills J. *Womanwords*. New York, NY: Holt; 1989.
25. Howes M. Menstrual function, menstrual suppression, and the immunology of the human female reproductive tract. *Perspect Biol Med*. 2010;53(1):16-30.
26. Hitchcock CL. Elements of the menstrual suppression debate. *Health Care Women Int*. 2008;29(7):702-719.
27. Brucker MC, Likis FE. Steroid hormones. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:355-376.
28. Miller WL, Auchus RJ. The molecular biology, biochemistry, and physiology of human steroidogenesis and its disorders. *Endocr Rev*. 2011;32(1):81-151.
29. Plant TM. The hypothalamo–pituitary–gonadal axis. *J Endocrin*. 2015;226(2):T41-T54.
30. Kranc W, Budna J, Kahan R, et al. Molecular basis of growth, proliferation, and differentiation of mammalian follicular granulosa cells. *J Biol Regul Homeost Agents*. 2017;31(1):1-8.
31. Vescovi JD. The menstrual cycle and anterior cruciate ligament injury risk: implications of menstrual cycle variability. *Sports Med*. 2011;41(2):91-101.
32. Bullivant SB, Sellergren SA, Stern K, et al. Women's sexual experience during the menstrual cycle: identification of the sexual phase by noninvasive measurement of luteinizing hormone. *J Sex Res*. 2004;41(1):82-93.
33. Sherman JJ, LeResche L. Does experimental pain response vary across the menstrual cycle? A methodological review. *Am J Physiol Regul Integr Comp Physiol*. 2006;291(2):R245-R256.
34. Chiras DD. *Human biology*. 9th ed. Burlington MA: Jones & Bartlett Learning; 2019.
35. Masters WH, Johnson VE. *Human Sexual Response*. New York, NY: Bantam; 1966.
36. American Psychiatric Association. Sexual dysfunctions. In: *Diagnostic and Statistical Manual of Mental Disorders*.

5th ed. Arlington, VA: American Psychiatric Association; 2013:423-450.
37. Basson R. Human sexual response. *Handbook Clin Neurol.* 2015;130:11-18.
38. Mazloomdoost D, Pauls RN. A comprehensive review of the clitoris and its role in female sexual function. *Sex Med Rev.* 2015;3(4):245-263.
39. Puppo V, Puppo G. Anatomy of sex: revision of the new anatomical terms used for the clitoris and the female orgasm by sexologists. *Clin Anat.* 2015;28(3):293-304.
40. Clayton AH, Harsh V. Sexual function across aging. *Curr Psychiatry Rep*. 2016;18(3):28. PATRICIA AIKINS MURPHY AND FRANCES E. LIKIS

CHAPTER

11

Menstrual Cycle Abnormalities

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Introduction

Changes in the menstrual cycle can create a range of cultural, emotional, physical, and psychological concerns for women. There are many ways in which an individual views menstrual and reproductive cycles, which are taken into consideration when a midwife provides accurate and relevant information for shared decision making. Acknowledging the normalcy of variation in menstrual cycles provides a framework within which individuals and midwives can identify and address abnormal changes should they occur. This chapter addresses the complex condition of amenorrhea as well as the different bleeding patterns associated with abnormal uterine bleeding (AUB). A discussion of dysmenorrhea, or painful menses, completes the chapter.

The Normal Menstrual Cycle and Variations

A wide variation in menstrual cycles exists, with only 15% of reproductive-age women reporting a cycle of 28 days.[1] The average range is 21 to 34 days and may vary between 2 and 20 days over a 12-month period.[2] The average duration of bleeding is 4.5 to 8 days. The amount of blood loss considered normal during menses has been described as approximately 30 mL based on research conducted in the 1960s, with anything more than 80 mL deemed abnormal.[1] However, it is common to either over- or underestimate the amount of menstrual bleeding that occurs on a monthly basis.[2] Additional information about the menstrual cycle is found in the *Anatomy and Physiology of the Female Reproductive System* chapter.

The onset of menstruation, termed *menarche*, occurs usually between 11 and 13 years (median age is 12.4 years), which is 2 to 3 years after thelarche (breast budding). The initial bleeding is usually anovulatory and unpredictable.[1,2] Cycles become regular and of the normal adult frequency by the third year after menarche.[2] By late adolescence, the typical ovulatory menstrual pattern is predictable in cycle regularity within a few days, and may be accompanied by mild dysmenorrhea. A few individuals also experience mild discomfort or sensation during ovulation, known as *mittelschmerz*.

Changes in menstrual cycle length are primarily due to differences in the follicular phase of the ovarian cycle; by comparison, the luteal phase tends to remain consistent. Women in their late 30s typically experience slightly shorter and less variable menstrual cycles than younger women due to increases in follicle-stimulating hormone (FSH) levels and decreases in inhibin levels, which results in a shorter follicular phase.[1]

Cycles typically lengthen again 2 to 4 years prior to menopause, followed by a complete cessation of menstruation. Menopause occurs at a median age of 51 years, with the range being 45 to 55 years.

Thus, the greatest fluctuation in duration of menstrual cycles occurs at either end of the reproductive spectrum, primarily due to an increase in anovulatory cycles among women younger than 20 years and those older than 40 years.[2] Because there can be a great deal of normal variation in the menstrual cycle in the early and later reproductive years, the initial steps in clinical evaluation of anyone who has menstrual irregularities differs for (1) adolescents, (2) women of reproductive age, and (3) those who are entering the menopausal transition. This distinction by age and proximity to menarche or menopause is always one of the initial considerations that is factored into the evaluation.

Many people describe any bleeding as a “period,” and some automatically interpret any blood found in underwear or on toilet tissue as of vaginal origin. For example, withdrawal bleeding associated with combined oral contraceptives may be mischaracterized by users as a menses. Nevertheless, an important distinction should be made between “menstrual bleeding” that occurs as a component of the menstrual cycle, even if it may vary in timing and amount, and AUB, which occurs independent of the menstrual cycle. **Table 11-1** reviews menstrual cycle parameters, and **Table 11-2** provides a glossary of terms used to describe forms of AUB.[3-6]

Table 11-1 Definitions of Terms to Describe Menstrual Irregularities

Description	Quantification
Frequency of Menses	
Absent	Amenorrhea
Frequent	More often than every 24 days
Normal	Occurs between 24–38 days
Infrequent	Less often than every 38 days
Regularity: Cycle to Cycle	
Regular	Variation of 2–20 days[a]
Irregular	Variation of > 20 days[a]
Duration of Menses	
Prolonged	> 8 days
Normal	4.5–8 days
Shortened	< 4.5 days
Volume per Menses	
Heavy	Volume that interferes with the woman's physical, social, emotional and/or material quality of life Menstrual blood loss of ≥ 80 mL
Normal	5–80 mL
Light	< 5 mL

[a] Normal variation in menstrual cycle regularity depends on age.

Based on Fraser IS, Critcheley HOD, Broder M, Munro MG. The FIGO recommendations on terminologies and definitions for normal and abnormal uterine bleeding. *Sem Reprod Med.* 2011;29(5):383-391[3]; Munro MG. Practical aspects of the two FIGO systems for management of abnormal uterine bleeding in the reproductive years. *Best Pract Res Clin Obstet Gynaecol.* 2017;40:3-22[4]; Munro MG, Critchley HO, Fraser IS. The FIGO systems for nomenclature and classification of causes of abnormal uterine bleeding in the reproductive years: who needs them? *Am J Obstet Gynecol.* 2012;207:259-265[5]; Munro MG, Critchley HO, Broder MS, Fraser IS. The FIGO classification system (PALM-COEIN) for causes of abnormal uterine bleeding in the non-gravid reproductive years including guidelines for clinical investigation. *Int J Gynaecol Obstet.* 2011;113:3-13.[6]

Table 11-2 Glossary of Terms That Define Menstrual Abnormalities and Abnormal Menstrual Bleeding

Term	Description
Abnormal uterine bleeding (AUB)	Any variation from the normal menstrual cycle Changes include those in regularity, duration of flow, amount of flow, and frequency of flow
AUB: Acute	An episode of bleeding in an individual of reproductive age, who is not pregnant, of sufficient quantity to require immediate intervention to prevent further blood loss
AUB: Chronic	Bleeding from the corpus uterus that is abnormal in duration, volume, and/or frequency that has been present for the majority of the last 6 months
AUB: Intermenstrual	Bleeding between clearly defined cyclically predictable menses
Amenorrhea	No menses for ≥ 3 previous consecutive months

	Subclassified into primary (prior to menarche) or secondary (post menarche)
Heavy menstrual bleeding (HMB)	Excessive menstrual blood loss (≥ 80 mL/cycle), which interferes with a person's physical, social, emotional, and/or quality of life
Heavy and prolonged menstrual bleeding (HPMB)	Heavy menstrual bleeding lasting > 8 days
Irregular menstrual bleeding	Range of varying lengths of bleeding-free intervals of > 20 days within one 90-day reference
Infrequent menstrual bleeding	Bleeding at intervals > 38 days apart (1–2 episodes in a 90-day period)
Frequent menstrual bleeding	Bleeding at intervals < 24 days apart (> 4 episodes in a 90-day period)
Prolonged menstrual bleeding	Menstrual blood loss which exceeds 8 days in duration
Shortened menstrual bleeding	Menstrual bleeding < 3 days in duration
Irregular, nonmenstrual bleeding	*Intermenstrual: Irregular episodes of bleeding, often light and short occurring between otherwise normal menstrual cycles*
	Postcoital: Bleeding after intercourse
	Premenstrual and postmenstrual spotting: Bleeding that may occur on a regular basis for ≥ 1 days before or after the recognized menstrual period
Bleeding outside of reproductive age	Postmenopausal bleeding: Bleeding occurring > 1 year after the acknowledged menopause
	Precocious menstruation: Bleeding occurring before the age of 9 years

Based on Fraser IS, Critcheley HOD, Broder M, Munro MG. The FIGO recommendations on terminologies and definitions for normal and abnormal uterine bleeding. *Sem Reprod Med.* 2011;29(5):383-391[3]; Munro MG. Practical aspects of the two FIGO systems for management of abnormal uterine bleeding in the reproductive years. *Best Pract Res Clin Obstet Gynaecol.* 2017;40:3-22.[4]

Amenorrhea

Amenorrhea is the absence of menses or menstrual bleeding.[7,8] Amenorrhea is further categorized as *primary* or *secondary* based on occurrence prior to or after menarche, respectively.[7-9] Although some causes of primary amenorrhea are distinct, most of the causes of secondary amenorrhea can also be the etiology of primary amenorrhea. Furthermore, amenorrhea can be the result of common events such as pregnancy, lactation, menopause, use of hormonal contraception, or the result of a pathologic condition. Both normal and pathologic causes of amenorrhea involve a disruption in the hypothalamic–pituitary–ovarian (HPO) axis or anatomic anomaly within the outflow tract. Thus, it is helpful to consider five possible categories when evaluating causes of amenorrhea: (1) outflow tract abnormality; (2) ovarian disorder; (3) pituitary disorder; (4) hypothalamus disorder; or (5) endocrine disorder that interferes with the HPO axis.

The list of differential diagnoses for amenorrhea is long. However, the majority of women who have amenorrhea that is not related to a normal condition (e.g., pregnancy, menopause, hormonal contraception, or lactation) have one of the following four disorders: (1) polycystic ovary syndrome; (2) hypothalamic amenorrhea (i.e., amenorrhea without an organic cause secondary to an eating disorder, stress, or excessive exercise); (3) hyperprolactinemia; or (4) primary ovarian insufficiency (also referred to as premature ovarian failure, premature menopause, gonadal dysgenesis).[7]

The etiologies unique to primary amenorrhea include anomalies of the outflow tract, genetic disorders that affect gonadal function such as Turner syndrome, and central anomalies of the HPO axis.[7-9] Approximately 30% to 40% of adolescents with primary amenorrhea are found to have Turner syndrome (45,XO karyotype).[9,10] The next most common causes of primary amenorrhea are structural abnormalities of the outflow tract (uterus, vagina, hymen) and central HPO axis anomalies.

Table 11-3 lists differential diagnoses associated with amenorrhea.

Table 11-3 Classification of Causes of Amenorrhea

Category	Examples
Expected physiologic findings	Pregnancy, lactation, hormonal contraceptive use, menopause
Outflow tract defects	Congenital: Müllerian agenesis, vaginal septum, imperforate hymen
	Acquired: Cervical stenosis, Asherman syndrome
Primary hypogonadism: ovarian insufficiency or ovarian failure	Congenital (e.g., Turner syndrome, Müllerian agenesis, gonadal dysgenesis, imperforate hymen, vaginal septum)
	Acquired (e.g., chemotherapy, autoimmune disorder); primary ovarian insufficiency
Hypothalamic disorders	Functional hypothalamic amenorrhea (secondary to weight loss, eating disorder, excessive exercise, stress, rarely pseudocyesis), gonadotropin deficiency, infection, chronic debilitating disease, malabsorption, traumatic brain injury, tumor
Pituitary disorders	Prolactinomas, hormone secreting pituitary tumor, autoimmune disease, Cushing syndrome, medications, Sheehan syndrome

Endocrine disorders	Polycystic ovary syndrome,[a] thyroid dysfunction, adrenal disease (Cushing syndrome), ovarian tumors

[a] Polycystic ovary syndrome is sometimes categorized as a sixth category termed “multifactorial” because several abnormalities of the hypothalamic–pituitary–ovarian axis are involved in this disorder.

The definition of *primary amenorrhea* is not standardized. One definition is the absence of menses by age 15 years in young women with developed secondary sex characteristics, within 5 years after breast development if breast development occurs before age 10 years, or if there is no pubertal development by age 13 years.[7,9] Another definition is a more relative description based on time from appearance of secondary sexual characteristics. Using this definition, primary amenorrhea is lack of menarche within 3 to 5 years of thelarche (Tanner breast stage II development) or by the age of 15 to 16 years regardless of the presence of secondary sex characteristics. Age at menarche of other family members may be helpful in determining whether the initiation of menses is within normal parameters for the individual.

Secondary amenorrhea is defined as absence of menses for 3 months in a previously menstruating woman or for 9 months in a woman who has experienced previous irregular menses with fewer than nine cycles in a year.[7,11] The incidence of secondary amenorrhea unrelated to pregnancy is approximately 5%, but rises to 80% in some populations, including those diagnosed with the female athlete triad (addressed later in this chapter).[7,9,12,13]

Evaluation for a Woman with Amenorrhea

The assessment of someone with amenorrhea starts with a determination of primary versus secondary amenorrhea and secondly consideration of age and proximity to menarche or menopause. These distinctions will direct the focus of the history and physical examination. The list of possible etiologies of amenorrhea is long and multiple different algorithms exist for evaluation this condition. **Figure 11-1** provides a representative example of the common steps involved in the initial evaluation. Follow-up tests to assess for specific disorders are recommended after initial screening to identify the most likely category of etiologies.

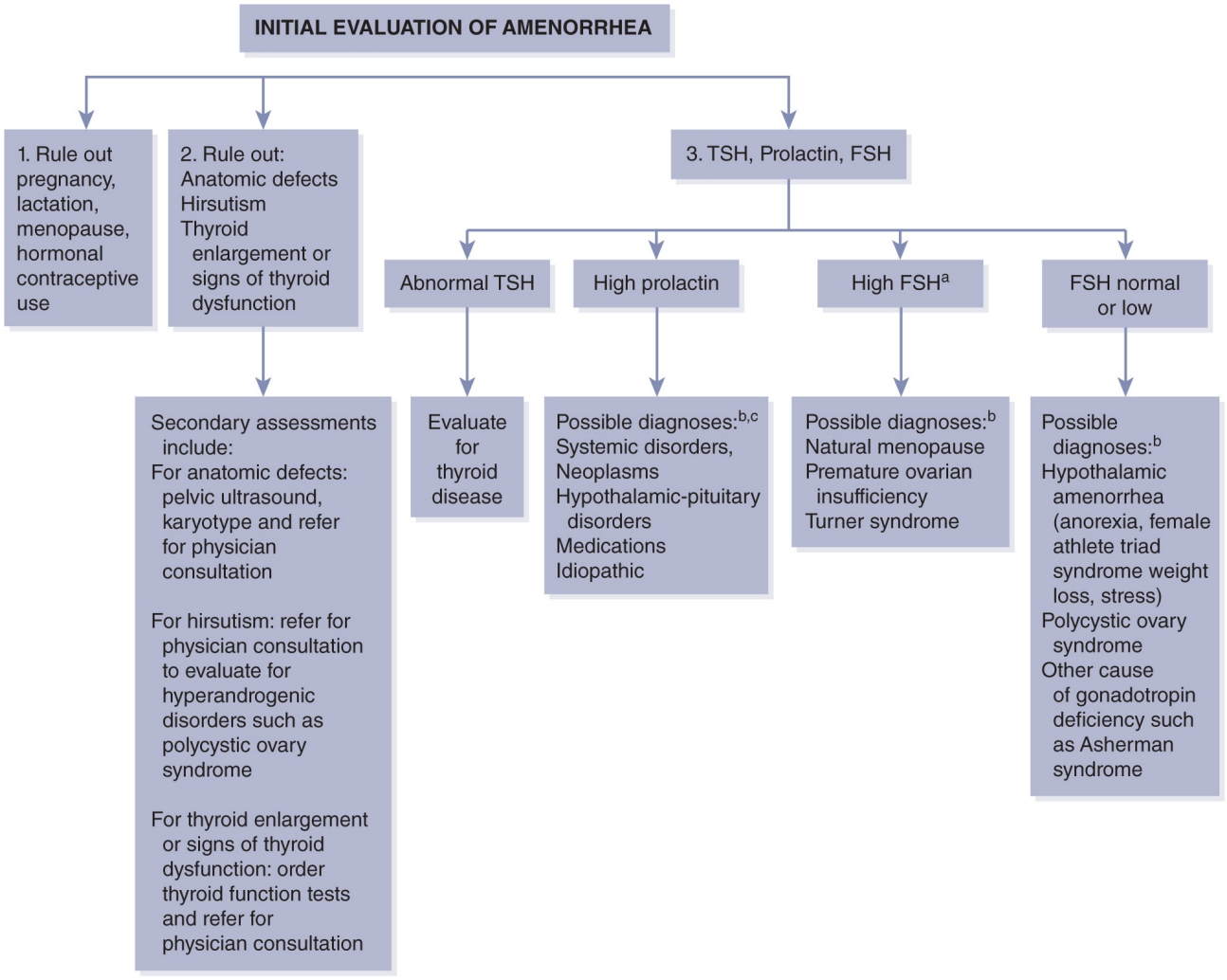


Figure 11-1 Initial evaluation of amenorrhea and common etiologies.

Abbreviations: FSH, follicle-stimulating hormone; MRI, magnetic resonance imaging; TSH, thyroid stimulating hormone.

[a] Some clinicians add a test for estradiol and/or luteinizing hormone to this initial evaluation.

[b] This list of diagnoses reflects common diagnoses and is not a complete list of the diagnoses possible.

[c] Prolactinemia can be secondary to many different disorders, including systemic disease, hypothalamic-pituitary disorders, medication use, or neoplasm. Prolactinemia may also be idiopathic. Approximately 50–60% of women with a high prolactin level have a pituitary tumor. The standard follow-up examination is an MRI.

Based on Practice Committee of the American Society for Reproductive Medicine. Current evaluation of amenorrhea. *Fertil Steril*. 2008;90(suppl 3):S219-S225[7]; Marsh C, Grimstad F. Primary amenorrhea: diagnosis and management. *Obstet Gynecol Surv.* 2014;69:603-612[8]; Klein DA, Poth MA. Amenorrhea: an approach to diagnosis and management. *Am Fam Phys.* 2013;87(11):781-788.[11]

History and Physical Examination

When initiating an evaluation of a woman with amenorrhea, a thorough history and physical examination are required. A detailed menstrual and family history will establish primary versus secondary amenorrhea and can provide insight into any familial patterns of irregular menses or early menopause. The history should also include information about use of hormonal

contraception, any change in overall health status, current and past medications, galactorrhea, presence of premenopausal symptoms, increase in stress levels, eating and exercise patterns, and any physical or emotional trauma.

The physical examination includes an assessment of body mass index (BMI), breast development (an indicator of previous estrogenic influence) and an evaluation of the presence of hirsutism or extensive muscle mass, both of which are suggestive of excessive testosterone levels. Thyroid palpation may detect possible thyroid dysfunction. External and internal genitalia should be thoroughly assessed for structural anomalies such as a blind or absent vagina. The findings of a genital examination will be abnormal in an estimated 15% of women with amenorrhea.[7] For most women with amenorrhea, the findings on physical examination will be normal, and the evaluation continues with an assessment of laboratory data.

Laboratory Studies

If the history and physical examination do not detect signs of congenital abnormalities, the next step for all women (unless the individual is postmenopausal) is to rule out pregnancy because pregnancy remains the most common reason for amenorrhea and results of this test will directly influence the next steps in evaluation. Once pregnancy is ruled-out, initial laboratory testing for FSH level, thyroid-stimulating hormone (TSH), and prolactin level will generate results that guide the rest of the evaluation. Other tests that may be indicated include pelvic ultrasound, especially in the event of nonpalpable ovaries. Such imaging provides information about structural and congenital abnormalities and endometrial thickness. Magnetic resonance imaging (MRI) is indicated if a pituitary tumor is suspected. The complex relationship of the HPO axis to menstruation can be challenging to assess. The midwife may initiate the evaluation to establish the category of possible diagnoses, but after the initial evaluation, consultation with and referral to a reproductive endocrinologist is recommended to ensure appropriate diagnosis and management.

High FSH levels suggest ovarian problems whereas low or normal FSH levels indicate either pituitary or hypothalamus problems. Elevated FSH levels in the absence of secondary sex characteristics indicate a functioning hypothalamus but lack of ovarian function which could be secondary to natural or premature menopause, or a genetic disorder such as Turner syndrome. Normal or low FSH levels in persons with secondary sexual development (with or without signs of hyperandrogenism) may indicate hypothalamic amenorrhea, polycystic ovary syndrome, or other rare causes of gonadotropin deficiency.

Elevated prolactin levels (typically > 100 ng/L) with or without headache or visual changes may indicate a pituitary adenoma and in an indication imaging (MRI) to evaluate the pituitary. Prolactin suppresses release of gonadotropin-releasing hormone (GnRH) and hyperprolactinemia can develop at any point of sexual development. Subclinical or overt hypothyroidism can also affect ovulatory function. Elevated TSH levels suppress GnRH release either directly or by causing increased prolactin release. Prolactin inhibits GnRH as part of a physiologic negative feedback mechanism.

Hormonal Challenge Testing (Progesterone Challenge, Estrogen/Progestin

Challenge)

Historically a progesterone challenge test was included in the evaluation of women with amenorrhea but current bioassays are sensitive tests and withdrawal bleeding correlates poorly with estrogen status and therefore many experts no longer use this test as part of an initial evaluation.[7,14]

Evaluation of a Young Person with Primary Amenorrhea

The most common causes of primary amenorrhea can be identified by performing a physical examination to determine the presence of secondary sex characteristics or structural anomalies, rule out pregnancy, and assess the HPO axis through measurement of FSH, TSH, and prolactin levels.[9-11] The work-up begins with a thorough history, including review of any sexual activity, age at menarche for the adolescent's mother, and any underlying conditions, medications, lifestyle, or nutritional deficits that may affect the menstrual cycle, including recent onset of headaches or visual disturbances.[1,9,15] When assessing adolescent sexual activity, it is advised to engage in a private conversation away from family or anyone who may prevent open communication or pose a threat. Issues of suspected sexual or physical abuse, use of recreational drugs, or other risk-taking behaviors should also be assessed in a private space and addressed as appropriate.

A thorough physical examination assessing sexual development, presence of galactorrhea, external genitalia, presence of pelvic organs, and any evidence of outflow tract anomalies is essential. Sensitivity to the fact that this may be the first such examination an adolescent may have experienced is important in building rapport and being able to conduct a thorough assessment.

A young person with primary amenorrhea requires care from a specialist. If secondary sex characteristics are not present, a bone age may need to be determined. Elevated FSH levels indicate the need for karyotyping to further investigate reasons for ovarian dysfunction. Therefore, the skills of a midwife may best be employed to interact with the adolescent and to refer her to appropriate care for this condition.

Management of women with secondary amenorrhea largely focuses on treatment of the underlying etiology and midwives are often involved in initial assessment and primary care for these women. The conditions seen most frequently in women of reproductive age are polycystic ovary syndrome and functional hypothalamus amenorrhea.

Polycystic Ovary Syndrome

Polycystic ovary syndrome (PCOS)—the most common diagnosis implicated in infertility related to ovarian dysfunction—is also the most common endocrine disorder in women with a prevalence of approximately 6% to 10% of the female population, depending on the population studied.[16,17] The incidence varies according to the criteria used to diagnose the condition. Historically, this condition was termed Stein–Leventhal syndrome, but later was renamed more descriptively as polycystic ovary syndrome.

PCOS is characterized by menstrual dysfunction, hyperandrogenism, and ovulatory

disruption.[16-18] The three classic features are hirsutism, irregular menstrual cycles and obesity. While amenorrhea is common in conjunction with this syndrome, menstrual cycle disruption is just one aspect of PCOS. Women with PCOS are often insulin resistant and are at increased risk for type 2 diabetes. Acanthosis nigricans—a brown velvety appearance to the skin in various folds, such as the back of the neck, in the axillae, under the breasts, or in the groin is a marker of insulin resistance and appears in some individuals with PCOS.[19,20] Polycystic ovaries are common in women with PCOS but can be seen in women without this disorder, so by itself, this finding is somewhat nonspecific. Women with PCOS are also at increased risk for developing endometrial cancer, sleep apnea, steatohepatitis, and mood disorders.

The cause of PCOS remains unknown, but a finer recognition of the possible adverse long-term sequelae is emerging, resulting in greater attention to diagnosis and treatment.[21,22] For example, women with PCOS—even those who are not obese—may have significant insulin resistance and, therefore, are at a higher lifetime risk for the development of both type 2 diabetes and cardiovascular disease.[1,17,22] Chronic anovulation associated with PCOS can result in unopposed estrogen stimulation of the endometrium and hyperplasia.

Diagnosis of Polycystic Ovary Syndrome

Criteria for diagnosis of PCOS continue to be debated by international experts.[17,19,20, 23-25] The Rotterdam PCOS Consensus Group criteria listed in **Table 11-4** are currently recommended by the United States National Institutes of Health.[23,25] There is some suggestion that infrequent menses or episodes of amenorrhea must be present for at least 2 years after menarche or primary amenorrhea at 16 years to make this diagnosis.[17] Additionally, true hyperandrogenism should be documented through measurement of testosterone levels, rather than just signs of androgen excess such as acne.[17,21] The Rotterdam criteria recognize three distinct clinical phenotypes of PCOS:

Table 11-4 Rotterdam PCOS Consensus Group Criteria for Diagnosis of Polycystic Ovary Syndrome

Exclusion of other etiologies and two out of three of the following:
Oligo- and/or amenorrhea
Clinical and/or biochemical signs of hyperandrogenism
Polycystic ovaries and exclusion of other criteria

Abbreviation: PCOS, polycystic ovary syndrome.

Based on National Institutes of Health. Evidence-based methodology workshop on Polycystic Ovary Syndrome. December 3-5, 2012. Executive Summary. Available at: https://prevention.nih.gov/docs/programs/pcos/FinalReport.pdf. Accessed December 9, 2017.[25]

- Frank PCOS: Oligomenorrhea, hyperandrogenism and polycystic ovaries
- Ovulatory PCOS: hyperandrogenism, polycystic ovaries, and regular menstrual cycles.
- Nonpolycystic ovary PCOS: Oligomenorrhea, hyperandrogenism, and normal ovaries

The diagnostic criteria for PCOS are different in adolescents than in adults. Menstrual irregularities, multifollicular ovaries, and signs of androgen excess (e.g., acne) are common in adolescents.[21] Thus, current diagnostic criteria for adolescents focuses on evidence of ovulatory dysfunction and androgen excess.[21] Evidence of androgen excess in adolescents includes one or more of the following: moderate to severe hirsutism, persistent acne vulgaris that is resistant to topical treatments, and elevation of serum total and/or free testosterone level. The diagnosis of PCOS is not established until other causes of hyperandrogenism have been ruled out.

If the diagnosis is confirmed, assessment of a risk of diabetes or cardiovascular disease is warranted, including measurement of glucose, hemoglobin A1c (HbA1c), and lipid levels. Insulin resistance does not always reveal itself as elevated blood glucose levels or elevated HbA1c value, but more typically results in blood sugar spikes and abdominal obesity prior to elevation of glucose levels. On examination, an assessment of the woman's BMI, blood pressure, waist-to-hip circumference, and hirsutism should also be made. A waist-to-hip ratio of 0.85 or more indicates abdominal obesity and an increased risk for metabolic and cardiovascular conditions.[26]

Treatment for Persons with Polycystic Ovary Syndrome

Treatment of women with PCOS varies and often depends on the degree of infertility or desire for conception. In women who are obese the abnormalities associated with PCOS may be treated with weight loss and lifestyle changes alone, with the aim of improving insulin sensitivity. Subsequent significant weight loss has been observed to result in more normal ovulatory and menstrual patterns as well as unassisted conception.[27] The use of the anti-hyperglycemic drug metformin (Glucophage) has increased in popularity in recent years, although the effectiveness of this medication in women with PCOS is not clear.[27] Because there is an association between obesity and depression, a screening for depression may be warranted when caring for any woman who has a high BMI.

If conception is not desired immediately, ovarian function and continued physical enlargement of the ovaries may be suppressed with the use of combination hormonal contraceptives.[28] This temporary ovarian suppression optimally will result in the initiation of normal ovarian function for a short time upon discontinuation of the hormonal contraceptive. Use of a hormonal contraceptive method will also provide protection from the potential risks of unopposed estrogen stimulation of the endometrium, which might otherwise result in endometrial hyperplasia or even endometrial cancer. Additional information, including contraindications for the use of combined oral contraceptives, can be found in the *Hormonal Contraception* chapter.

Various medications, such as gonadotropin-releasing hormone (GnRH) agonists and the diuretic spironolactone (Aldactone), have also been employed as antiandrogens. Induction of ovulation, through administration of clomiphene citrate (Clomid), is a common strategy for the prompt treatment of women with infertility as a result of PCOS.[17,19,25] Women are best referred to a specialist for these treatments. **Table 11-5** provides a sample of the critical elements in a transfer or referral note to the specialist.

Table 11-5	Sample Critical Elements for a Transfer Note for an Individual with Possible Polycystic Ovary Syndrome
Menstrual history, including duration of amenorrhea and irregular menses	
Signs and symptoms suggestive of PCOS (e.g., hirsutism, obesity)	
Laboratory results or if pending, including pregnancy test, TSH, HbA1c, and sonography	
Care provided before transfer: procedures and results	
Assessment/diagnosis of the situation	
Summary of rationale for transfer and request for information about treatment plan for follow-up	

Abbreviations: HbA1c, hemoglobin A1c; PCOS, polycystic ovary syndrome; TSH, thyroid-stimulating hormone.

Early diagnosis and intervention for PCOS, with respect to the health risks of hyperglycemia, abnormal cholesterol and lipid profiles, and reproductive cancer risk, may positively influence overall health. In addition, mental health disruptions that may result from a diminished sense of self-esteem due to physical appearance and infertility require the midwife's attention.[17]

Functional Hypothalamic Amenorrhea

Functional hypothalamic amenorrhea can occur when the HPO axis is disrupted by nutritional deficiency, extreme weight loss, exercise, and stress; it is characterized by a chronic low estrogen state along with low to normal FSH levels in the absence of organic disease.[29,30] Women who experience functional hypothalamic amenorrhea have a functional disruption in the normal pulsatile GnRH secretion. The decreased pulses result in absent midcycle LH surge, anovulation, and amenorrhea. The diagnosis is made after excluding other organic disorders that cause amenorrhea. Low serum FHS and low estradiol levels in combination with the presence of precipitating factors such as excessive exercise, anorexia nervosa, stress, and weight loss are usually present. A progesterone challenge test may be indicated to confirm low estrogen status and lack of withdrawal bleeding.

Limited energy availability is associated with hypothalamic dysfunction, which adversely affects menstruation and bone health.[29,30] In contrast, functional hypothalamic amenorrhea is not necessarily associated with an eating disorder, although anorexia nervosa should be considered in the differential diagnosis. Weight loss and low energy may be present as a result of over-exercise and a lack of awareness that the woman is not taking in sufficient calories to support her energy expenditure. Menstrual function can be restored when the energy imbalance is addressed.

Treatment of women with this complex condition requires an interdisciplinary team approach that includes nutritional support, mental health services, and family support. Most importantly, the focus should center on the woman and her shared decision making about lifestyle choices. Treatment should include close monitoring for osteopenia or osteoporosis, as the hypo-estrogenic state may negatively influence bone density over time.[1,29]

Female Athlete Triad and Eating Disorders

Women who are diagnosed with functional hypothalamic amenorrhea should be assessed for the syndrome called *female athlete triad*, which can occur in women who are not elite athletes, and in persons with eating disorders, especially anorexia nervosa.[29,30] The female athlete triad is the result of energy imbalance affecting hypothalamic function and involves three components: (1) low energy availability with or without an eating disorder, (2) menstrual dysfunction, and (3) low bone density.[29] It is not necessary to have all three components present to recognize the triad. Any one of the three may be present individually or in combination.

Women who have anorexia nervosa may have many of the same symptoms as women with female athlete triad and women who have female athlete triad are at increased risk for eating disorders. The primary distinction between the two conditions is that individuals with anorexia maintain a low body weight, severely restrict nutritional intake, and have a fear of gaining weight. Anorexia is more common in younger adolescents in general. Amenorrhea may be present as well but anorexia is not always associated with excessive exercise.

Anorexia is associated with multiple endocrine abnormalities and more morbidity. The most common long-term risk for women with female athlete triad is osteoporosis. Because there is significant overlap between these two conditions, any woman who has symptoms of either should be referred to a physician and multidisciplinary team for further evaluation, diagnosis, and treatment. Midwives who participate in care of these women as part of an interprofessional team, will have additional training in the healthcare services needed by this population.

Hyperprolactinemia (Pituitary Adenoma)

Elevated prolactin levels can suppress the normal gonadotropin release necessary to stimulate ovulation, resulting in amenorrhea. Prolactin levels can be elevated by a number of conditions, including pituitary adenoma and prolonged hypothyroidism.[11] Other causes of elevated prolactin levels include use of medications such as tricyclic antidepressants and opiates that block dopamine receptors. Dopamine normally inhibits prolactin secretion, so anything that blocks dopamine can result in increased prolactin levels.[10] High prolactin levels much higher than the maximum of the normal range, which is 20 ng/mL, that may or may not be accompanied by visual changes or headaches may indicate the presence of a pituitary adenoma and require evaluation by MRI and an endocrinologist.[8],[10] Care of persons with hyperprolactinemia is best provided by specialists.

Rare Conditions Associated with Secondary Amenorrhea

Amenorrhea is also associated with premature ovarian insufficiency, autoimmune disorders, and ovarian tumors.[6,7] Premature ovarian insufficiency is the inability of the ovary to respond to gonadotropin stimulation, and is usually found in a woman 40 years or younger.[15] Premature ovarian insufficiency affects 0.5% to 3.0% of reproductive-age women.[15] This condition can result from natural causes, chemotherapy, radiation, extreme stress, other medications, or autoimmune causes.[1,7,31] Autoimmune disorders that are associated with premature ovarian

insufficiency include adrenal insufficiency (Addison's disease) and autoimmune thyroid disease (Graves' or Hashimoto disease); metabolic conditions (e.g., type 1 diabetes) may also cause secondary amenorrhea. Ovarian tumors can disrupt menstrual function by changing the responsiveness of the ovary to gonadotropins.

Less common conditions associated with amenorrhea include late-onset congenital adrenal hyperplasia, Cushing syndrome, adrenal and androgen-secreting tumors, Asherman syndrome, and Sheehan syndrome. Congenital adrenal hyperplasia may present later in a woman's life as increased androgen production and observable androgenic effects affect the normal menstrual cycle. Other hyperandrogenic states resulting from conditions such as Cushing syndrome and adrenal tumors affect the menstrual cycle by suppressing normal ovarian function through overproduction of androgens. Ovulation and menstruation often return after correction of the hyperandrogenic state.

Asherman syndrome is the development of intrauterine adhesions and/or fibrosis of the endometrium as a result of trauma, excessive instrument manipulation, or evacuation of the uterus.[14] The endometrium is not functional and does not respond to hormonal stimulation. The entire endometrium may not be affected, resulting in lighter than normal cyclical endometrial shedding versus complete amenorrhea.

Sheehan syndrome is ischemic necrosis of the pituitary gland, which usually occurs as a result of a profound postpartum hemorrhage.[14] Destruction of the pituitary gland results in amenorrhea, hypothyroidism, and impaired initiation of lactation.[14]

Complementary/Alternative Therapies for the Treatment of Women with Amenorrhea

Herbalists may recommend blessed thistle or blue cohosh for the relief of amenorrhea, while acupuncture and homeopathic remedies have been suggested as other treatment modalities.[32] To date, no published, evidence-based investigations have addressed the safety of using these remedies and effectiveness has not been conclusively demonstrated.

Abnormal Uterine Bleeding

AUB is an overarching term that refers to uterine bleeding that is abnormal in frequency, duration, regularity, and/or volume.[5,6,33] AUB affects as many as 30% of women at some point between menarche and menopause.[34-36] AUB can be acute or chronic. This condition is often subcategorized as anovulatory or ovulatory patterns. Anovulatory AUB is associated with prolonged unopposed estrogen stimulation of the endometrium and subsequent irregular bleeding. PCOS, thyroid dysfunction, hyperprolactinemia, uncontrolled diabetes, and use of some medications are typical causes of anovulatory AUB. Anovulatory AUB is associated with an increased risk for endometrial cancer. Ovulatory AUB is associated with heavy menstrual bleeding (menorrhagia) and can be caused by thyroid dysfunction, coagulation defects, endometrial polyps or uterine fibroids (submucosal).

In practice, women with AUB experience patterns of bleeding that encompass more than one of these categories. For example, a woman may experience heavy menstrual bleeding that is longer in duration and higher in volume than normal bleeding. Intermenstrual bleeding may be frequent and irregular. AUB also refers to any bleeding that occurs in a postmenopausal woman, as discussed in the *Midlife, Menopause, and Beyond* chapter.

Similar to the causes associated with amenorrhea, the most common etiologies of AUB differ by age. In adolescents younger than 18 years, AUB is often the result of anovulation that is secondary to dysregulation of the HPO axis and can be a normal physiologic finding. The second most common cause of AUB in this age group is an inherited coagulopathy.[33,34] In women between the ages of 19 and 39 years, pregnancy, leiomyomas, polyps, PCOS, and use of hormonal contraception are common causes of AUB.[34] In women 40 years and older, AUB may again be a symptom of the normal physiology of menopause but can also be a symptom of endometrial hyperplasia, leiomyomas, or endometrial carcinoma.[34]

A Newer Lexicon for Abnormal Uterine Bleeding

Many terms have been used to define menstrual irregularities. The lack of a common nomenclature has hindered research and development of evidence-based treatments. In 2011, the International Federation of Gynecology and Obstetrics (FIGO) proposed a system, PALM-COEIN,[3-6,35-38] that recommended replacing the term “dysfunctional uterine bleeding” (DUB) with “abnormal uterine bleeding” (AUB), and then adding a suffix to identify the proposed etiology. The PALM-COEIN nomenclature uses a classification system of structural and nonstructural causes followed by specific etiologies.[3-6] This terminology standardized the nomenclature and parameters for both normal uterine bleeding and AUB that occurs in reproductive age women, based on the 5th–95th percentiles identified in large epidemiologic studies.[4,6] The PALM-COEIN nomenclature has been adopted as the standard and outdated terms such as menorrhagia are no longer recommended to be used.[39,40] The widespread use of more precise language ultimately may facilitate both diagnostic efforts and health education/communication with women. **Table 11-6** summarizes the PALM-COEIN system.

Abnormal Menstrual Bleeding Assessment Framework: PALM-COEIN

Table 11-6

Terminology	Abbreviation	Description
PALM: Discrete Structural Entities Measurable Using Imaging Techniques and/or with Histopathology		
Polyp	AUB-P	Endometrial or endocervical polyps Categorized as present or absent, with no distinction regarding size or number
Adenomyosis	AUB-A	Disorder in which the ectopic endometrial tissue (endometrial glands and stroma) are present in the uterine myometrium. This causes uterine myometrial hypertrophy and hyperplasia. Clinical presentation is heavy menstrual bleeding and dysmenorrhea Adenomyosis is diagnosed via ultrasound or MRI. Subcategories based on diffuse vs focal or multifocal disease and volume have been proposed
Leiomyoma	AUB-L	Leiomyomatas (uterine fibroids) are fibromuscular tumors of the myometrium. First subclassification: Present or absent Second subclassication: Submucosal (SM) or other (O). Submucosal are further categorized as 0 (pedunculated intracavity) or 1/2 based on if less than 50% intramural or more than 50%. SM fibroids contribute to AUB. Other types are 3 (contacts endometrium, 100% intramural), 4 (intramural), 5 (subserosal ≥ 50% intramural), 6 (subserosal < 50% intramural), 7 (subserosal pedunculated), and 8 (includes cervical or parasitic and other lesions not related to the myometrium). Third subclassification: Used for hybrid lesions that describes the endometrial relationship first and serosal relationship second, separated by a hyphen
Malignancy and hyperplasia	AUB-M	Endometrial intraepithelial neoplasia (atypical hyperplasia) and/or malignancy. Subclassified by separate WHO and FIGO systems that stage malignancy based on histopathology and extent of the tumor.
COEI: Entities Not Defined by Imaging or Histopathology		
Coagulopathy	AUB-C	AUB secondary to a coagulopathy or systemic disorder that causes a coagulopathy. Approximately 13% of women with heavy menstrual bleeding have von Willebrand disease.
Ovulatory dysfunction	AUB-O	Disorders of ovulation can cause a spectrum of menstrual abnormalities that range from amenorrhea to heavy menstrual bleeding. Examples include PCOS, hypothyroidism, hyperprolactinemia, anorexia, extreme stress, gonadal steroids, and tricyclic antidepressants. Otherwise unexplained ovulatory dysfunction can occur during adolescence and the menopause transition.
Endometrial	AUB-E	AUB occuring within predictable and cyclic menstrual cycle, such as prolonged bleeding. This indicates ovulation is occuring. If no other etiology is identified, the primary disorder is likely to be in the endometrium.
COEI: Entities Not Defined by Imaging or Histopathology		
		The etiology may be infection, or a combination of mechanisms that interfere with local production of vasoconstrictors (e.g., prostaglandin F), increased production of hormones that break down endometrial clot (e.g., plasminogen activator) and/or increased production of substances that promote vasodilation (e.g. prostacyclin).
Iatrogenic	AUB-I	Many medical devices and medications can cause or contribute to AUB. Examples include intrauterine devices and warfarin (Coumadin). The AUB associated with estrogen and progestogen formulations administered continuously to induce amenorrhea is termed “break through bleeding.”
Not Otherwise Classified		

Not otherwise classified AUB-N	Causes of AUB that are rare or their role in causing AUB is not well defined. Examples include arteriovenous malformations and cesarean scar defects.

Abbreviations: AUB, abnormal uterine bleeding; FIGO, International Organization of Gynecology and Obstetrics; MRI, magnetic resonance imaging; PCOS, polycystic ovary syndrome; WHO, World Health Association.

Based on Munro MG, Critchley HO, Broder MS, Fraser IS. The FIGO classification system (PALM-COEIN) for causes of abnormal uterine bleeding in the non-gravid reproductive years including guidelines for clinical investigation. *Int J Gynaecol Obstet.* 2011;113:3-13.[6]

Evaluation of Abnormal Uterine Bleeding

The initial steps in evaluating a woman of reproductive age who has AUB are listed in **Table 11-7**. Medications associated with AUB are listed in **Table 11-8**. Because the list of differential diagnoses is extensive, the initial steps in the evaluation to assess for common etiologies of AUB first takes into consideration the following three primary factors: (1) the woman's age (i.e., proximity to menarche or menopause), (2) whether the AUB pattern is indicative of ovulatory or anovulatory status, and (3) the woman's a priori risk for endometrial cancer.

Table 11-7	Initial Evaluation of a Reproductive-Age Woman with Abnormal Uterine Bleeding

A. Initial History and Assessment

1. Determine if the bleeding is acute or chronic. Individuals who have acute vaginal bleeding or are hemodynamically unstable should be immediately referred for emergent care.
2. If chronic, establish the clinical impact that AUB has on activities of life.

   Assess for iron deficiency and symptoms of anemia
3. Assess for pregnancy: Pregnancy-related bleeding may present as any degree of bleeding, from light spotting to menstrual-like flow. The latter underscores the importance of considering pregnancy for all reproductive-age women who present with AUB.
4. Review health history using the PALM-COEIN system to focus on possible etiologies.

   Menstrual history:

   Age of menarche and menopause

   Detailed history of frequency, duration, predictability, amount of bleeding, interval, relation of AUB to normal menstrual cycle, associated symptoms.

   Sexual and reproductive history: Assess use of contraception, STIs, infertility, possible pregnancy.

   Symptoms associated with a systemic cause of AUB such as obesity, PCOS, hypothyroidism, hyperprolactinemia, or adrenal disorder

   Chronic medical illness that might cause a coagulopathy such as liver disease, renal disease, inherited bleeding disorders, systemic lupus erythematosus

   Medication history: Medications associated with AUB are listed in **Table 11-8**.

   Family history of bleeding disorders or women with "heavy periods," or thromboembolic disorders, or hormone sensitive cancers.

   Surgical history: Especially obstetric or gynecologic surgery

B. Physical Examination

A complete physical examination with attention focused on the following components:

Vital signs, BMI, blood pressure

Skin: The woman's skin should be assessed for signs of:

Coagulation disorder: pallor, bruising, bleeding gums, petechiae, swollen joints

Hyperandrogenism: hirsutism, acne, male-pattern baldness,

Insulin resistance: acanthosis nigricans on neck

Thyroid examination

Abdominal assessment for tenderness, distension, and palpable masses.

Gynecologic examination:

1. Origin of the bleeding: Bleeding that originates from the cervix, vagina, external genitalia, urinary tract, or rectum may appear different from uterine bleeding in volume, color, quality, and timing.
2. External genitalia should be examined for signs of trauma, lesions, hemorrhoids, or infection.
3. Speculum examination to identify sources of bleeding outside of the uterus such as cervical polyp, cervicitis, or vaginal laceration.
4. A bimanual pelvic examination to assess the uterus and adnexa for tenderness, size, and/or palpable masses.

C. Laboratory Tests

Initial laboratory testing for all types of AUB:

CBC with platelets

Ferritin or total iron and iron saturation

Pregnancy test

TSH

Transvaginal ultrasound to assess pelvic structures and endometrial thickness is usually included in a standard evaluation of reproductive-age women and postmenopausal women, but may not be necessary for adolescents near menarche.

Additional laboratory testing as indicated:

Endocrine disorders: FSH, LH, estradiol, progesterone level (obtained 22–24 days in menstrual cycle), free testosterone level, prolactin level,

Coagulation abnormalities: Coagulation profile, or von Willebrand diagnostic panel

STI: Chlamydia, wet mount of vaginal discharge, vaginal culture.

Cervical cancer screening

Additional tests as indicated:

Endometrial biopsy to evaluate the endometrium

Hysteroscopy

MRI

Abbreviations: AUB, abnormal uterine bleeding; BMI, body mass index; CBC, complete blood count; FSH, follicle-stimulating hormone; LH, luteinizing hormone; MRI, magnetic resonance imaging; PCOS, polycystic ovary syndrome; STI, sexually transmitted infection; TSH, thyroid-stimulating hormone.

Based on Munro MG, Critchley HO, Broder MS, Fraser IS. The FIGO classification system (PALM-COEIN) for causes of abnormal uterine bleeding in the non-gravid reproductive years including guidelines for clinical investigation. *Int J Gynaecol Obstet.* 2011;113:3-13[6]; American College of Obstetricians and Gynecologists. Committee Opinion No. 128: diagnosis of abnormal uterine bleeding in reproductive age women. Obstet Gynecol. 2012;120:197-206[34]; American College of Nurse-Midwives. Clinical Bulletin No. 15: abnormal uterine bleeding. *J Midwifery Womens Health*. 2016;61:522-527.[40]

Table 11-8 **Selected Drugs That Can Cause Abnormal Vaginal Bleeding**

Drug Category	Drugs: Generic (Brand)
Analgesics	Aspirin NSAIDs
Anticoagulants	Warfarin (Coumadin) Heparin
Anticonvulsant	Valproic acid (Depakene)
Antibiotics	Rifampin (Rifadin) Griseofulvin
Antidepressants	SSRIs[a]
Antiemetics	Metoclopramide (Reglan)[a] Prochlorperazine (Compazine)[a]
Antipsychotics	Phenothiazines (e.g., thioridazine [Mellaril], chlorpromazine [Thorazine], risperidone [Risperdal])[a] Tricyclics (e.g., amitriptyline [Elavil])[a]
Corticosteroids	Prednisone (Deltasone) Dexamethasone (Decadron)
Herbal supplements	Ginseng, ginkgo, motherwort
Hormonal contraceptives	Combined oral contraceptives: transdermal, vaginal, injectable Progestin-only pills Levonorgestrel-releasing intrauterine device
Menopausal hormone therapy	Estrogen, progesterone, androgens and combination formulations
Opioids	Methadone[a] Morphine[a]
Selective estrogen receptor modulators	Tamoxifen (Nolvadex)

Abbreviations: NSAIDs, nonsteroidal anti-inflammatory drugs; SSRIs, selective serotonin reuptake inhibitors.

[a] Prolactin secretion from the pituitary is normally suppressed by dopamine. Any drug that blocks dopamine receptors can cause hyperprolactinemia which can then cause ovulatory dysfunction and AUB.

Based on Fritz MA, Speroff L. *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011[1]; Munro MG, Critchley HO, Broder MS, Fraser IS. The FIGO classification system (PALM-COEIN) for causes of abnormal uterine bleeding in the non-gravid reproductive years including guidelines for clinical investigation. *Int J Gynaecol Obstet*. 2011;113:3-13.[6]

For example, the physical examination for an adolescent will include assessment of sexual maturity and if the bleeding is heavy, signs of coagulopathy, but may not require a pelvic examination if she is not sexually active or just recently past menarche. The risk of endometrial cancer is quite low in adolescence, whereas etiologies discernable via laboratory evaluation are more likely. In contrast, a pelvic ultrasound and possibly endometrial biopsy is always considered for postmenopausal women in order to rule out malignancy, which is much more likely as women age.

The choice of imaging studies or endometrial biopsy in the evaluation of AUB is also an example of how the evaluation of AUB differs on the basis of age, type of bleeding, and risk

for endometrial cancer. Approximately 70% to 90% of women who develop endometrial cancer will have AUB. Risk factors for endometrial cancer that indicate a need for evaluation of the endometrium are listed in **Table 11-9**.[35] Evidence of an endometrial stripe larger than 4.0 mm on ultrasound suggests the possibility of endometrial hyperplasia or carcinoma, and is an indication for further investigation such as endometrial biopsy and possible surgical treatment such as endometrial curettage.[34,35,37] Endometrial biopsy is an office procedure that can be performed by midwives who have received training in the technique. The procedure is reviewed in **Appendix 11A**.

Table 11-9 **Risk Factors for Endometrial Cancer That Require Evaluation of the Endometrium**[a]

Age	Indication for Evaluation of the Endometrium
Postmenopausal	Any bleeding, staining, or spotting
45 years to menopause	Any AUB including intermenstrual bleeding, heavy menstrual bleeding, prolonged bleeding
19–45 years	Prolonged period of amenorrhea
	Any AUB in women with conditions associated with unopposed estrogen exposure such as obesity (BMI ≥ 30 kg/m^2)
	AUB that is persistent in women who are not obese but have one of the following:
	Chronic anovulation
	Failed medical management of bleeding
	Lynch syndrome

Abbreviations: AUB, abnormal uterine bleeding; BMI, body mass index.

[a] Additional risk factors not related to AUB include abnormal cervical cytology results, monitoring of women with endocervical hyperplasia.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 136: management of abnormal uterine bleeding associated with ovulatory dysfunction. *Obstet Gynecol*. 2013;122:176-185. [Reaffirmed 2016].[35]

Trauma and subsequent bleeding from genital lacerations is one of the differential diagnoses for women who present with AUB. Intimate-partner violence or sexual assault requires a more extensive assessment, and should involve consultation or referral to a community center or a professional experienced in working with individuals who have experienced assault or abuse (e.g., a rape crisis center or a sexual assault nurse examiner [SANE]).[41] Depending on the local laws that govern such cases, the midwife may also be legally required to report suspicion, or confirmation, of abuse. Most importantly, the midwife should initiate safety and support mechanisms. Referral to a surgeon may be necessary if anatomic damage is identified that appears to require surgery or extensive treatment.

Management of Women with Abnormal Uterine Bleeding

After normal menstrual changes, pregnancy, infection, trauma, and malignancy have been ruled out, interventions for AUB will vary based on the following initial considerations:

- Specific etiology
- Bleeding severity
- Associated symptoms such as pain or infertility
- Reproductive planning: desire for contraception and plans for pregnancy
- Medical comorbidities
- The woman's preferences with consideration of side effects of possible treatments

Furthermore, effective treatments can be either surgical or medical. For example, infection can be treated with antibiotics and leiomyomata can be treated with hormonal contraceptives or surgical resection. Therefore, when offering treatment for AUB, the woman's age, desire for pregnancy, and degree of bleeding factor into the management plan. Women with heavy menstrual bleeding secondary to leiomyomata who want to become pregnant may choose surgical therapy to preserve fertility whereas those who do not want to become pregnant can treat this condition with contraceptives that decrease menstrual bleeding.

In general, indications for treatment of AUB include heavy menstrual bleeding and AUB with ovulatory dysfunction (AUB-O). Women who are anemic should start iron supplementation regardless of the type of bleeding.

Heavy Menstrual Bleeding

Heavy menstrual bleeding (HMB) is defined as excessive bleeding, either in amount or in duration, at the regular interval of normal menstruation and by definition, presumes the woman is ovulatory. HMB should be treated if it is interfering with the woman's quality of life or when it causes anemia. The most common etiologies of HMB in reproductive age women are leiomyomatas and adenomyosis. The goal of treatment is to reduce the volume of menstrual flow, correct anemia, and prevent cancer.

The first line therapies for women with heavy menstrual bleeding are monophasic low-dose combined oral contraceptives or levonorgestrel-releasing intrauterine device (LNG-IUD).[33,37,40,42] Combined oral contraceptives have been shown to reduce menstrual blood loss by 35% to 69%.[43]

The choice between combined oral contraceptives and the LNG-IUD is based on the woman's desire for contraception, the presence, or absence of contraindications to estrogens, and personal preference. The combined oral contraceptives will induce a regular withdrawal bleed while the LNG-IUD is associated with irregular bleeding in the first months and then progress to amenorrhea.

While the typical treatment algorithm calls for combined oral contraceptives to be prescribed in the same manner as for women using the pills for contraception, evidence suggests that extended or continuous usage may be a more effective approach for treating heavy menstrual bleeding. This approach facilitates the stabilization of the endometrium and offers a

brief respite from the previously experienced excessive bleeding.[1] Injection of depot medroxyprogesterone acetate (DMPA) is also effective. The progesterone formulations are associated with irregular bleeding; headaches and weight gain in some women but ultimately these agents will result in amenorrhea. Amenorrhea, or menstrual suppression can be a lifestyle benefit as well as therapeutic value for some individuals, especially transmen. Additional information about menstrual suppression can be found in the *Hormonal Contraception* chapter.

If combined oral contraceptives, the LNG-IUD, or DMPA are not acceptable or are contraindicated, physician consultation is generally recommended unless the midwife is working as part of a physician–midwife team wherein guidelines for use of other agents are available. Other effective treatments for heavy menstrual bleeding include progesterone, GnRH agonists, nonsteroidal anti-inflammatory drugs (NSAIDs), danazol (Danocrine), a synthetic steroid that suppresses HPO axis activity and tranexamic acid (Lysteda). Progestin-only contraceptives and hormonal contraceptive inserts are not effective for treating heavy menstrual bleeding. However, orally administered progesterone in higher doses can be effective if used continuously for 21 days.

GnRH agonists (e.g., leuprolide [Lupron]) affect the HPO axis, hindering the ability of the ovaries to release the hormones required for normal function of the menstrual cycle. Interfering with cyclic menstruation causes the woman to enter a physiologic state resembling menopause. However, because of the bone mass loss associated with the decrease in estrogen levels and vasomotor menopausal symptoms, use of these medications tends to be limited to persons for whom other therapies have been unsuccessful, and may be best managed by a practitioner specializing in gynecologic disorders.[1] Prolonged treatment using GnRH agonists requires bone density monitoring.

Reduction in heavy menstrual bleeding occurs in women who are taking NSAIDs, due to the ability of these medications to block the synthesis of prostaglandins necessary for cyclic endometrial sloughing.[1] However, NSAID therapy is not always effective and can cause gastrointestinal bleeding with long-term use and for others, NSAID use can result in increased vaginal bleeding due to inhibition of platelet aggregation and prolongation of bleeding time. When using combined oral contraceptives, a woman also may take NSAIDs at the time of menstruation, and benefit from the combination of these medications.

Danazol (Danocrine), a synthetic steroid, has been successful in controlling heavy menstrual bleeding in some women, although it requires daily dosage for 3 to 6 months, given as 100 mg orally twice a day, during which time a state of amenorrhea is likely to occur. Significant androgenic side effects, including weight gain, acne, and seborrhea, are common with this medication, and the availability of other treatments has resulted in danazol no longer being the first choice of treatment for HMB.

Complementary/Alternative Treatments

While some observational studies have findings that support the treatment of women with HMB with traditional Chinese medicines and acupuncture, as well as with the use of herbal, homeopathic, and aromatherapy remedies, there is insufficient evidence to support use of

nonpharmacologic methods at this time.

Treatment for Abnormal Uterine Bleeding-Ovulatory Dysfunction

If AUB is not heavy menstrual bleeding but is suspected to be ovulatory dysfunction (AUB-O), the woman may present with irregular erratic bleeding that can vary from light to heavy. The underlying etiology is that lack of ovulation results in failure to produce a corpus luteum and the ovaries do not secrete progesterone. The lack of progesterone affects the uterine endometrium so that it continues to proliferate without the progesterone withdrawal induced shedding. Thus, the bleeding is irregular in timing and amount. After malignancy has been ruled out, the choice of treatment with progestin, estrogen, or a combination of the two has historically depended on the type of bleeding with which the woman presents. In general, infrequent menses has been treated first with progestin, while irregular bleeding has been treated initially with estrogen.[1] However, combination hormonal contraceptives are effective in controlling a number of menstrual disorders. Thus, many clinicians use combined oral contraceptives or another combined hormonal method as the first line of treatment, especially if the woman is interested in, or has no objection to the use of, contraception. With each monthly withdrawal cycle while on the combined hormonal methods, bleeding and cramping should decrease.

Dysmenorrhea

Painful menstruation, particularly in the lower abdomen and back and usually of a cramping nature, is known as *dysmenorrhea*. Dysmenorrhea usually is associated with regular, predictable menses. In the United States, this condition is experienced by 60% to 91% of menstruating adolescents and women.[1] Dysmenorrhea that occurs in the absence of other disease is termed *primary dysmenorrhea*. Dysmenorrhea that is caused by a disorder such as endometriosis or leiomyomata is termed *secondary dysmenorrhe*a.

In distinguishing dysmenorrhea from other causes of pelvic pain, it is important to establish this pain as cyclic in nature, coinciding with the onset of menses and resolving after cessation of the menstrual flow. Primary dysmenorrhea is typically recurrent, crampy, and may radiate to the back or thighs. It can be accompanied by nausea, fatigue, and general malaise. Primary dysmenorrhea generally starts just before the onset of menses and lasts 2 or 3 days.

The principal cause of primary dysmenorrhea is the presence of prostaglandins that are released from the endometrium at the beginning of menses. These prostaglandins induce uterine contractions which result in uterine ischemia and accumulation of metabolites that stimulate pain nerves (**Figure 11-2**).[44,45] Primary dysmenorrhea usually begins in adolescence after ovulatory cycles are established. Pain can be mild to severe and generally improves as the woman ages.

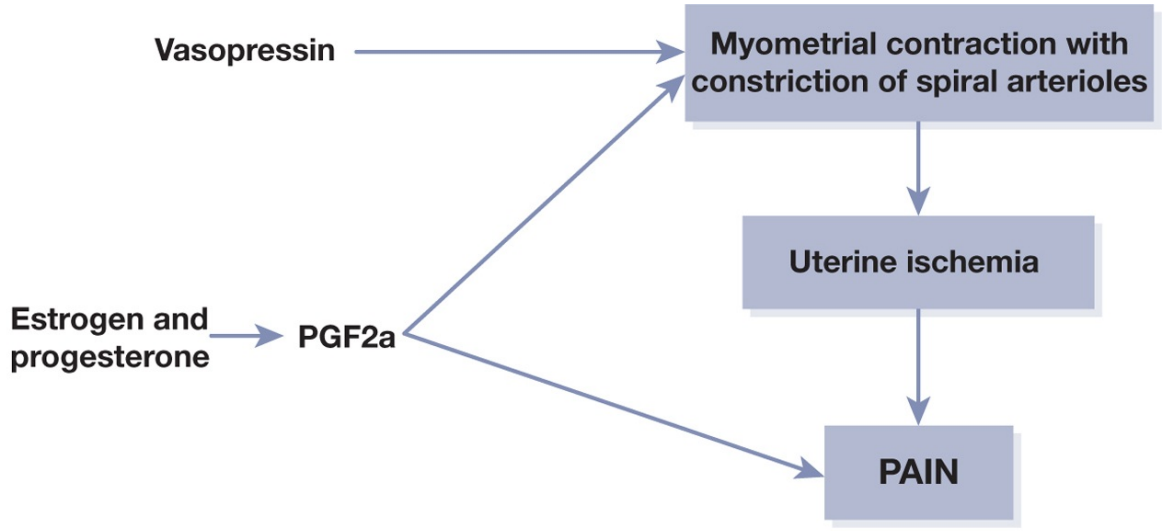


Figure 11-2 Pathophysiology of primary dysmenorrhea.

Reproduced with permission from Durain D, McCool WF. Pelvic and menstrual disorders. In: Brucker MC, King TL. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:901-928.[44]

Evaluation of an Individual with Dysmenorrhea

The evaluation of women with dysmenorrhea focuses on excluding the presence of disorders

that can cause cramping uterine pain.[46] Although endometriosis is the most common cause of secondary dysmenorrhea, other etiologies include pregnancy, an IUD in place, pelvic inflammatory disease, adenomyosis, ovarian cysts, pelvic adhesions, or cervical stenosis. Nongynecologic causes include inflammatory bowel disease and irritable bowel syndrome.

Treatment of an Individual with Primary Dysmenorrhea

The goal of treatment for women with primary dysmenorrhea is to relieve pain. Application of local heat to the abdomen has demonstrated effectiveness that in some women is equal to the relief achieved after taking ibuprofen (Advil).[47] The first line pharmacologic agents recommended are NSAIDs because these drugs inhibit prostaglandin synthesis.[46] There is no one NSAID that is more effective than others so the choice of agent depends on individual choice and tolerability as these drugs are associated with some adverse effects. The medications should be taken for 2 to 3 days beginning on the first day of symptoms on a fixed schedule to maximize effectiveness.

The primary adverse effect of NSAIDs in healthy women is gastrointestinal distress, but these agents are also (albeit less frequently) associated with peptic ulcer, renal injury, hepatotoxicity, and bronchospasm in persons with asthma. Furthermore, these induce several drug–drug interactions. Therefore, before prescribing an NSAID that will be taken on a regular basis, a review of the woman's health and current medications is needed. Although acetylsalicylic acid (aspirin), another NSAID, has also been found to offer some relief from dysmenorrhea, there is no evidence that the non-NSAID analgesic acetaminophen (Tylenol) is beneficial in eliminating menstrual pain.[47]

If dysmenorrhea persists, consideration of alternative therapies may become necessary. Because combined hormonal contraceptives decrease prostaglandin synthesis and menstrual flow, these drugs are frequently used on an off-label basis for the treatment of women with dysmenorrhea.[48] In addition, the various progestin-only methods of contraception (injectable DMPA, progestin-only oral contraceptives, levonorgestrel-containing IUDs, and etonogestrel-containing implants) often result in temporary states of amenorrhea due to decreased endometrial stimulation. In turn, these methods can result in less dysmenorrhea.[49]

Several nonpharmaceutical approaches to the treatment of women with dysmenorrhea have been widely advocated. Among them are the application of local heat in the form of small heat packs continuously applied to the abdomen, homeopathy (e.g., belladonna and chamomilla), acupuncture, biofeedback, dietary supplements, relaxation techniques, massage, exercise, aromatherapy (e.g., rose oil), and the use of certain herbs (e.g., black cohosh, raspberry leaf, shakuyaku-kanzo-to, semen coicis, and chaste berry).[50-56] With the exception of the use of local heat application, there is limited scientific evidence of effectiveness for the various nonpharmaceutical modalities.

Conclusion

Menstrual cycle abnormalities are a common reason for seeking healthcare services and assessment requires a sensitive and thorough approach. Exploring the individual's sense of reproductive or hormonal well-being or normalcy can often be achieved by taking a comprehensive history of the menstrual pattern and examining attitudes and beliefs about the significance of, and even need for, cyclic bleeding. Treatment of menstrual abnormalities can positively influence a person's long-term health well beyond the specific change appreciated by the person at the outset of care. This aspect of health care calls upon midwives' expertise as primary and gynecologic care clinicians who are concerned with the entirety of women's health.

Resources

Organization	Description	Webpage
Point-of-Care Apps		
Agency for Healthcare Research and Quality (AHRQ).	Diagnosis of AUB in reproductive-aged women	https://www.guideline.gov/summaries/summary/38623
Clue[a]	One of the many consumer-oriented vaginal bleeding/menstrual trackers. This well-reviewed app is available for both the iOS and Android platforms. Among the team involved in “Clue” are transgender men so language tends to be inclusive. The app includes features that allow women to track a variety of symptoms as well as bleeding that may be of benefit when presenting for care.	https://www.helloclue.com

[a] Multiple apps for menstrual cycle tracking are available. Many apps are supported by ads from companies that produce menstrual supplies.

References

1. Fritz MA, Speroff L. *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.
2. American College of Obstetricians and Gynecologists. Committee Opinion No. 651: menstruation in girls and adolescents: using the menstrual cycle as a vital sign. *Obstet Gynecol*. 2015;126:e143-e146. [Reaffirmed 2017].
3. Fraser IS, Critchley HOD, Broder M, Munro MG. The FIGO recommendations on terminologies and definitions for normal and abnormal uterine bleeding. *Sem Reprod Med*. 2011;29(5):383-391.
4. Munro MG. Practical aspects of the two FIGO systems for management of abnormal uterine bleeding in the reproductive years. *Best Pract Res Clin Obstet Gynaecol*. 2017;40:3-22.
5. Munro MG, Critchley HO, Fraser IS. The FIGO systems for nomenclature and classification of causes of abnormal uterine bleeding in the reproductive years: who needs them? *Am J Obstet Gynecol*. 2012;207:259-265.
6. Munro MG, Critchley HO, Broder MS, Fraser IS. The FIGO classification system (PALM-COEIN) for causes of abnormal uterine bleeding in the non-gravid reproductive years including guidelines for clinical investigation. *Int J Gynaecol Obstet*. 2011;113:3-13.
7. Practice Committee of the American Society for Reproductive Medicine. Current evaluation of amenorrhea. *Fertil Steril*. 2008;90(suppl 3):S219-S225.
8. Marsh C, Grimstad F. Primary amenorrhea: diagnosis and management. *Obstet Gynecol Surv*. 2014;69:603-612.
9. American College of Obstetricians and Gynecologists. Committee Opinion 605: primary ovarian insufficiency in adolescents and young women. *Obstet Gynecol*. 2014;123:193-197. [Reaffirmed 2016].
10. Deligeoroglou E, Athanasopoulos N, Tsimaris P, Dimopoulos D, Vrachnis N, Creatsas G. Evaluation and management of adolescent amenorrhea. *Womens Health Dis*. 2010;1205:23-32.
11. Klein DA, Poth MA. Amenorrhea: an approach to diagnosis and management. *Am Fam Phys*. 2013;87(11):781-788.
12. Thein-Nussenbaum J, Hammer E. Treatment strategies for the female athlete triad in the adolescent athlete: current perspectives. *Open Access J Sports Med*. 2017;8:85-95.
13. Javed A, Tebben PJ, Fischer PR, Lteif AN. Female athlete triad and its components: toward improved screening and management. *Mayo Clin Proc*. 2013;88(9):996-1009.
14. Nelson LM. Primary ovarian insufficiency. *N Engl J Med*. 2009;360(6):606-614.
15. Moltich M. Diagnosis and treatment of pituitary adenomas. *JAMA*. 2017;317(5):516-524.
16. Shannon M, Wang Y. Polycystic ovary syndrome: a common but often unrecognized condition. *J Midwifery Womens Health*. 2012;57:221-230.
17. Fauser B, Talatzis B, Rebar R, et al. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): Amsterdam ESHRE/ASRM-sponsored 3rd PCOS Consensus Workshop Group. *Fertil Steril*. 2012;97:28-38.
18. Ferreira SR, Motta AB. Uterine function; from normal to polycystic ovary syndrome alterations. *Curr Med Chem*. 2017. [Epub ahead of print]. doi:10.2174/0929867325666171205144119.
19. Legro RS, Arslanian SA, Ehrmann DA, et al. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2013;98(12):4565-4592.
20. Dumesic DA, Oberfield SE, Stener-Victorin E, Marshall JC, Laven JS, Legro RS. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. *Endocr Rev*. 2015;36(5):487-525.
21. Rosenfield RL. The diagnosis of polycystic ovary syndrome in adolescents. *Pediatrics*. 2015; 136:1154-1158.
22. Wang ET, Calderon-Margalit R, Cedars MI, et al. Polycystic ovary syndrome and risk for long-term diabetes and dyslipidemia. *Obstet Gynecol*. 2011;117(1):6.
23. Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod*. 2004; 19:41.
24. Azziz R, Carmina E, Dewailly D, et al. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. *Fertil Steril*. 2009; 91:456.
25. National Institutes of Health. Evidence-based methodology workshop on Polycystic Ovary Syndrome. December 3-5, 2012. Executive Summary. Available at: https://prevention.nih.gov/docs/programs/pcos/FinalReport.pdf. Accessed December 9, 2017.
26. Klein S, Allison DB, Heymsfield SB, et al. Waist circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, the Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Obesity*. 2007;15(5):1061-1067.
27. Tang T, Lord JM, Norman RJ, Yasmin E, Balen AH. Insulin sensitizing drugs. *Cochrane Database Syst Rev*. 2012;5:CD003053. doi:10.1002/14651858.CD003053.pub5.
28. Dokras A. Noncontraceptive use of oral combined hormonal contraceptives in polycystic ovary syndrome: risks versus benefits. *Fertil Steril*. 2016;106(7):1572-1579.
29. American College of Obstetricians and Gynecologists. Committee Opinion 702: female athlete triad. *Obstet Gynecol*.

2017;129:e160-e170.
30. Gordon CM, Ackerman KE, Berga SL, et al. Functional hypothalamic amenorrhea: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab* 2017;102:1413.
31. Komorowska B. Autoimmune premature ovarian failure. *Menopause Rev*. 2016;15(4):210-214.
32. Lim C, Ng RWC, Xu K, et al. Acupuncture for polycystic ovarian syndrome. *Cochrane Database Syst Rev*. 2016;5:CD007689. doi:10.1002/14651858.CD007689.pub3.
33. Davidson B, DiPiero C, Govoni K, Littleton S, Neal J. Abnormal uterine bleeding in the reproductive years. *J Midwifery Womens Health*. 2012;57:248-254.
34. American College of Obstetricians and Gynecologists. Committee Opinion No. 128: diagnosis of abnormal uterine bleeding in reproductive age women. *Obstet Gynecol*. 2012;120:197-206.
35. American College of Obstetricians and Gynecologists. Practice Bulletin No. 136: management of abnormal uterine bleeding associated with ovulatory dysfunction. *Obstet Gynecol*. 2013;122:176-185. [Reaffirmed 2016].
36. Munro M, Critchley H, Fraser I. Research and clinical management for women with abnormal uterine bleeding in the reproductive years: more than PALM-COEIN. *BJOG*. 2017;124(2):185-189.
37. Bradley L, Gueye NA. The medical management of abnormal uterine bleeding in reproductive age women. *Am J Obstet Gynecol*. 2016;214:31-44.
38. Bacon JL. Abnormal uterine bleeding: current classification and clinical management. *Obstet Gynecol Clin North Am*. 2017;44(2):179-193.
39. Deneris A. PALM-COEIN nomenclature for abnormal uterine bleeding. *J Midwifery Womens Health*. 2016;61(3):376-379.
40. American College of Nurse-Midwives. Clinical Bulletin No. 15: abnormal uterine bleeding. *J Midwifery Womens Health*. 2016;61:522-527.
41. Adams P, Hulton L. The sexual assault nurse examiner's interactions within the sexual assault response team: a systematic review. *Adv Emerg Nurs J*. 2016;38(3):213-227.
42. Heikinheimo O, Fraser I. The current status of hormonal therapies for heavy menstrual bleeding. *Best Pract Res Clin Obstet Gynaecol*. 2017;40:111e-120e.
43. Matteson KA, Rahn DD, Wheeler TL, et al. Non-surgical management of heavy menstrual bleeding: a systematic review and practice guidelines. *Obstet Gynecol*. 2013;121(3):632-643.
44. Durain D, McCool WF. Pelvic and menstrual disorders. In: Brucker MC, King TL. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:901-928.
45. Dawood MY. Primary dysmenorrhea: advances in pathogenesis and management. *Obstet Gynecol*. 2006; 108:428-441.
46. Osayande AS, Mehulic S. Diagnosis and initial management of dysmenorrhea. *Am Fam Phys*. 2014;89(5):341-346.
47. Akin M, Price W, Rodriguez G Jr, et al. Continuous, low-level, topical heat wrap therapy as compared to acetaminophen for primary dysmenorrhea. *J Reprod Med*. 2004;49:739-745.
48. Marjoribanks J, Ayeleke RO, Farquhar C, Proctor M. Nonsteroidal anti-inflammatory drugs for dysmenorrhea. *Cochrane Database Syst Rev*. 2015;7:CD001751. doi:10.1002/14651858.CD001751.pub3.
49. American College of Obstetricians and Gynecologists. Practice Bulletin No. 110: noncontraceptive uses of hormonal contraceptives. *Obstet Gynecol*. 2010;115:206-218.
50. Pattanittum P, Kunyanone N, Brown J, et al. Dietary supplements for dysmenorrhea. *Cochrane Database Syst Rev*. 2016;3:CD002124. doi:10.1002/14651858.CD 002124.pub2.
51. Uysal M, Doru HY, Sapmaz E, et al. Investigating the effect of rose essential oil in patients with primary dysmenorrhea. *Complement Ther Clin Pract*. 2016;24:45-49.
52. Hong GY, Shin BC, Park SN, et al. Randomized controlled trial of the efficacy and safety of self-adhesive low-level light therapy in women with primary dysmenorrhea. *Int J Gynecol Obstet*. 2016;133(1):37-42.
53. Lee HW, Jun JH, Kil K-J, Ko B-S, Lee CH, Lee MS. Herbal medicine (Danggui Shaoyao San) for treating primary dysmenorrhea: a systematic review and meta-analysis of randomized controlled trials. *Maturitas*. 2016;85:19-26.
54. Zhu X, Proctor M, Bensoussan A, Wu E, Smith CA. Chinese herbal medicine for primary dysmenorrhoea. *Cochrane Database Syst Rev*. 2008;2:CD005288. doi:10.1002/14651858.CD005288.pub3.
55. Sut N, Kahyaoglu-Sut H. Effect of aromatherapy massage on pain in primary dysmenorrhea: a meta-analysis. *Comp Therap Clin Pract*. 2017;27:5-10.
56. Smith CA, Armour M, Zhu X, Li X, Lu ZY, Song J. Acupuncture for dysmenorrhea. *Cochrane Database Syst Rev*. 2016;4:CD007854. doi:10.1002/14651858.CD007854.pub3.

APPENDIX

11A

Endometrial Biopsy

WENDY GRUBE AND WILLIAM F. McCOOL

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Endometrial biopsy (EMB) using a flexible plastic device is a cost-effective, safe, and simple method of collecting a histologic sample of the uterine endometrium. A number of clinical circumstances can require investigation of the endometrium, especially among women who experience abnormal uterine bleeding (AUB) that might be associated with premalignant hyperplasia or carcinoma. The risk of uterine perforation associated with EMB is less than the risk of uterine perforation associated with dilatation and curettage (D & C) (0.1–0.2%, versus 0.3–2.6%, respectively.)[1] The diagnostic D & C is no longer considered the "gold standard" for this purpose due to surgical risks, need for anesthesia, and cost.[1,2] The evolution of sampling devices such as the Pipelle has allowed EMB to be easily accomplished in an office setting by providers.

The sensitivity and specificity of EMB have been noted to be 100% for detection of endometrial carcinoma, hyperplasia, proliferative and secretory endometrium.[2] Sensitivity for detection of endometritis has been estimated at 88.9%, and sensitivity for detection of endometrial polyps has been estimated at 60%.[2] This limitation is primarily due to the limited surface area of the uterine lining that is capable of being sampled via EMB devices. Combining transvaginal ultrasonography and sonohysterography with EMB can assist with the identification of such structural abnormalities as well as location of focal pathology.

Indications

1. Postmenopausal women with any uterine bleeding
2. AUB in women older than 45 years, or women at any age if AUB has not been controlled with appropriate medications
3. Examination of thickened endometrium (> 4 mm) noted on transvaginal ultrasound of the uterus in the sagittal position to rule out endometrial cancer or precursors
4. Evaluation of atypical glandular cells (endometrial or other) found in cervical cytology

sampling
5. Women at high risk for endometrial cancer, such as those on tamoxifen therapy, a history of Lynch syndrome therapy, a history of Lynch syndrome, and women who have undergone treatment for endometrial cancer with uterus-sparing therapy
6. Assessment of adverse pregnancy outcomes that may be secondary to chronic subclinical endometritis

Contraindications

1. Viable and desired pregnancy
2. Known or suspected cervical cancer
3. Clotting disorder
4. Inability to visualize cervical os
5. Obstructing cervical lesion

Relative Contraindications Requiring Consultation

1. Infection of the vagina, cervix, or uterus requires evaluation and treatment prior to EMB procedure
2. Use of medications that may alter clotting requires consultation with a provider who is knowledgeable in hematologic disorders
3. History of prosthetic heart valve requires cardiologist consultation, and antibiotic prophylaxis
4. Fever (temperature > 38°C [100.4°F]) at the time of the procedure
5. Severe cervical stenosis or atypical uterine anatomy requires consultation with or referral to a gynecologist

Potential Side Effects/Complications and Preventive Measures

1. Cramping, uterine spasm, and vasovagal response are the most common side effects associated with this procedure. Prophylactic analgesia with 600–800 mg ibuprofen (Advil) administered 60 minutes prior to the procedure is recommended. In addition, women should be encouraged to eat prior to the procedure to avoid symptoms of hypoglycemia.
2. Uterine perforation. The following decrease the risk of uterine perforation:
   a. Make certain that the woman is not pregnant or has a well-involuted postpartum uterus
   b. Perform a thorough pelvic examination prior to the procedure to note the uterine and

cervical size, position, and angulation, as well as any structural abnormalities

c. Use a tenaculum to straighten the utero-cervical angle if the uterus is not close to axial in position.

3. Uterine infection, pelvic infection, and bacteremia.
4. Excessive uterine bleeding is rare but may occur if the woman has an undiagnosed coagulopathy.

Procedure

1. If the purpose of the sample is confirmation of ovulation/diagnosis of luteal-phase defects, schedule the procedure for day 22 or 23 of the menstrual cycle. Timing is not important if the sampling is being performed to detect cancer or its precursors.
2. Gather equipment needed for the procedure:[1-3]
   a. Informed consent form
   b. Nonsterile and sterile gloves
   c. Vaginal speculum of appropriate size
   d. Ring forceps and cotton balls or large cotton swabs
   e. Antiseptic solution (such as Betadine or Hibiclens)
   f. Topical anesthetic such as Benzocaine gel 20% (Hurricaine) or 2% lidocaine
   g. Scissors
   h. Labeled specimen containers with 10% formalin
   i. Endometrial sampling devices (make certain there are at least two available)
   j. Tenaculum

A sample tray for endometrial biopsy is shown in **Figure 11A-1**.



Figure 11A-1 Sample tray for endometrial biopsy.

3. Thoroughly review the woman's health history, including the date of last menses, contraception and possibility of pregnancy, risk of sexually transmitted infection, known bleeding disorder, medication or supplement use, and allergies. Based on this information, contraindications may emerge that preclude performance of the procedure, or additional testing prior to the EMB may be necessary such as a pregnancy test, STI diagnostic testing, saline or potassium hydroxide (KOH) slide tests, or complete blood count.
4. Provide education and instruction regarding the procedure, side effects, and possible adverse effects of EMB. Informed consent must be obtained before proceeding.
5. Offer an NSAID oral agent to decrease cramping and uterine spasm associated with the procedure.
6. Assist the woman to assume a lithotomy position. Using nonsterile gloves, perform a bimanual and rectovaginal examination (if needed) to verify uterine and cervical position, angle, and structure to ensure that the curette can be placed in the appropriate direction and to minimize the risk of uterine perforation.
7. Insert the appropriately sized speculum.
8. Apply antiseptic solution to the cervix with a large cotton swab or cotton ball.
9. If desired, apply topical anesthetic (e.g., Benzo-caine gel 20%) to the anterior lip of the cervix and into the os with a small cotton swab to lessen the pain associated with the curette or tenaculum. If additional pain reduction is indicated, 5 mL of 2% lidocaine can be instilled into the endometrial cavity through a small catheter.[1]

10. If a tenaculum is needed, place the tenaculum on the anterior lip of the cervix, and gently pull the device to straighten the utero-cervical angle. An assistant can be asked to hold the tenaculum and thereby maintain this angle.
11. Change to sterile gloves, remove the curette (the outer sheath and inner piston) from the sterile package as instructed on the package insert.
12. With the piston fully inserted into the sheath, gently introduce the curette through the cervical os and into the uterine cavity until resistance is felt (**Figure 11A-2).** If strong resistance is encountered prior to reaching the fundus, stop the procedure.
13. If there is difficulty advancing the device through the inner os, the introduction of a small cervical dilator or a uterine sound by using steady, moderate pressure may be helpful. Another option is to discontinue the procedure and insert a 3-mm osmotic laminaria, which can be placed in the cervix on the morning of the day of the EMB and removed that afternoon prior to conducting the actual procedure.
14. Once resistance is felt, note the distance that the curette has entered the uterus, using the markings located on the sheath. On average, the length of the cervix from external to internal os is 2.5 cm, and the total distance from the external os to the wall of the fundus is approximately 6 to 9 cm. This information should be noted and recorded in the woman's health record.
15. With the tip of the device at the fundus, hold the curette securely, and slowly but consistently partially withdraw the inner piston. Withdrawal of the piston creates the suction, or negative pressure, at the tip needed to collect the tissue sample. It is not necessary to withdraw and replace the piston repeatedly; indeed, doing so is counterproductive. The piston should *not* be totally withdrawn or suction will be lost.
16. Move the sheath of the device back and forth, with the tip moving from fundus to internal os, while simultaneously rolling it between the thumb and fingers to allow collection of cells from different levels of the endometrium as well as different locations. Avoid allowing the tip of the device to slip back out of the internal os into the cervical canal, as this will result in the loss of suction.
17. Complete the simultaneous moving and rolling of the sheath maneuver until the sheath is filled. Both tissue and some blood should be visible.
18. Remove the device. If only blood can be visibly identified within the sheath, after removing the entire curette, place the contents in formalin, and use another curette to attempt the procedure once or twice more depending on the woman's consent and how well the procedure is tolerated. The same curette can be reused if it has maintained sterility, including not touching the formalin.
19. Once the tissue has been adequately collected, cut the distal tip from the device and place the tip in the labeled formalin container. Cutting the tip from the device allows the sample to be removed intact, without causing the cell breakup that can occur when tissue is forced through the tip. Placing the tip of the catheter in the specimen container ensures that any tissue collected in this portion of the device will be analyzed with the remainder of the specimen.
20. Gently press the piston back into the sheath to expel the remaining specimen into the same labeled container.

21. Remove the tenaculum and speculum, but encourage the woman to remain supine for a few minutes prior to getting up and dressed to reduce the risk of a vasovagal response. It is common for a woman to experience some cramping either during the procedure or immediately afterward; anticipatory guidance is indicated on this possibility.
22. Instruct the woman to expect some light spotting during the next few days, but to contact the office if bright red blood or excessive bleeding or clotting, fever, vaginal discharge with a foul odor, pelvic cramping, or pain occurs. Counseling includes advising no vaginal penetration sexual intercourse for 2 to 3 days after the procedure.
23. Document the procedure, including any analgesic or anesthetic given, any abnormal findings during examination or procedure, the sampling device used, the depth of insertion of the curette, adequacy of the specimen obtained, and the woman's toleration of the procedure.

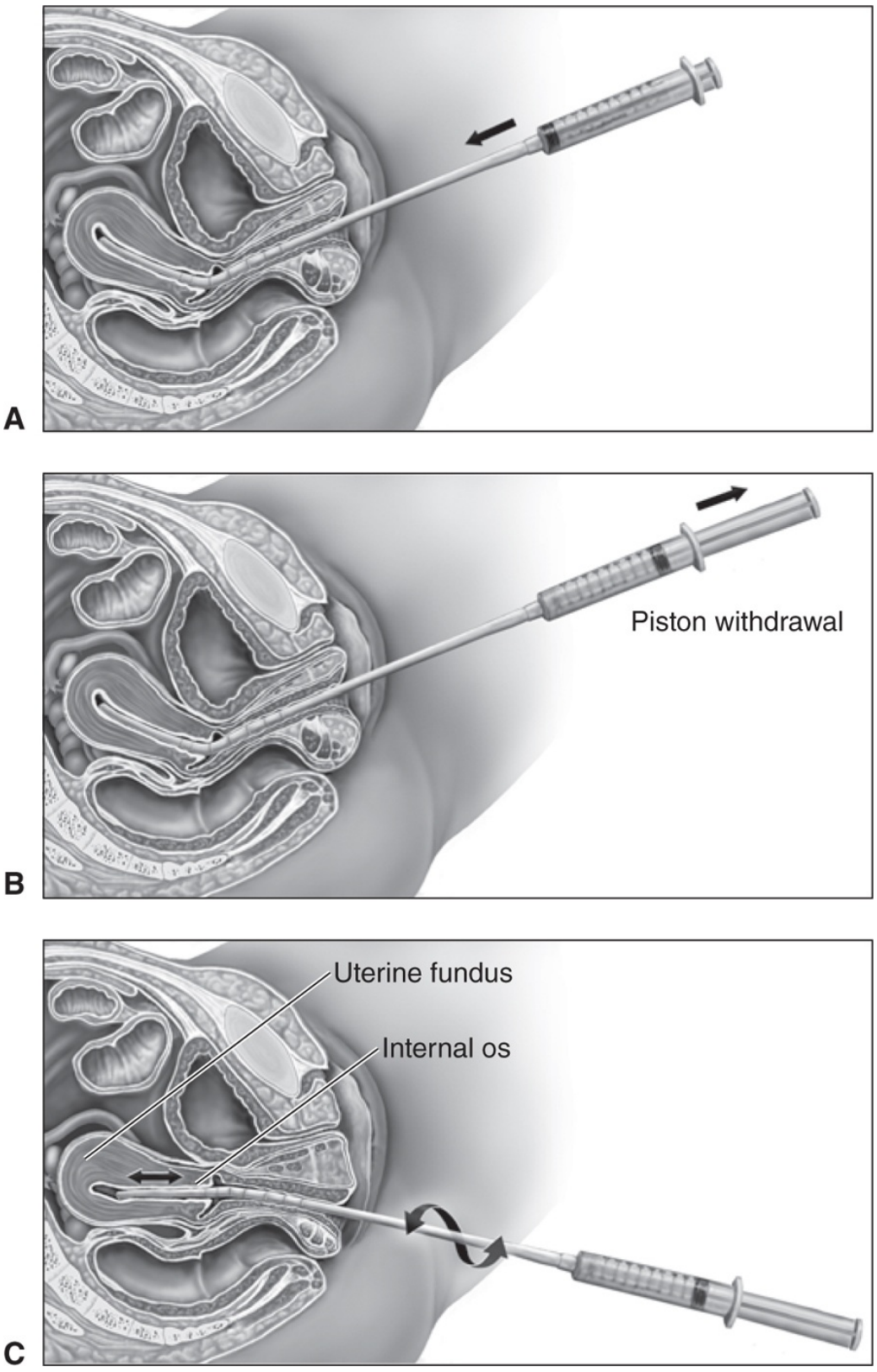


Figure 11A-2 Endometrial biopsy technique. **A.** Insert the device until resistance is felt or the uterine fundus is reached (Step 12). **B.** Withdraw the piston from the fully inserted device (Step 15). **C.** Rotate the device through 360 degrees while moving it back and forth between the fundus and the internal cervical os.

Results and Management

Laboratory findings can vary in language and presentation depending on the laboratory reporting system. In general, the provider will receive a histology report, with information that can contribute to making a diagnosis regarding the status of the endometrium. In communicating the findings to the woman, the provider should ensure that the woman understands the results and the possible courses of action to follow. The most common histologic reports received, with suggestions for follow-up, are listed in **Table 11A-1**.

Table 11A-1 Common Histologic Findings from Endometrial Biopsy Samples

Histology	Probable Diagnosis	Suggested Management	Need for Referral
Insufficient cells or unable to assess	Unable to assess	Dependent on clinical indication for EMB; may repeat EMB or potential referral for dilatation and curettage	Dependent upon specific clinical indication
Inactive endometrium	Hypoestrogenic state	Dependent on clinical presentation and indication for EMB	Dependent upon specific clinical indication
Proliferative (estrogen-influenced preovulation), disorganized proliferative endometrium or secretory (progesterone-influenced postovulation)	AUB	TVUS to rule out structural pathology Hormonal therapy such as combined oral contraceptives, or cycling with progestins or a LNG-IUS if the woman is of reproductive age	Women with an abnormal TVUS, or those who do not respond to hormonal therapies as expected
Simple or complex nonatypical hyperplasia	Premenopausal or perimenopausal woman	Combined oral contraceptives, cycling with progestins, or insertion of a LNG-IUS Repeat EMB in 6 months	No response to hormonal therapy
	Postmenopausal woman	No therapy, or hormonal therapy depending on amount of bleeding, age, and time lapsed from menopause (discussed in more detail in the *Midlife, Menopause, and Beyond* chapter)	Continued irregular bleeding, or no response to hormonal therapy
Atypical hyperplasia (simple or complex)—suspicion of cancer	Rule out endometrial carcinoma	Refer to a specialist (e.g., collaborating physician or gynecologic oncologist) for probable surgery	Continued irregular bleeding, or no response to hormonal therapy

Abbreviations: AUB, abnormal uterine bleeding; EMB, endometrial biopsy, LNG-IUS, levonorgestrel intrauterine system; TVUS, transvaginal ultrasound.

References

1. American College of Nurse-Midwives. Clinical Bulletin No. 17: endometrial biopsy. *J Midwifery Womens Health*. 2017;62;502-506.
2. Zuber TJ. Endometrial biopsy. *Am Fam Phys*. 2001;63:1131-1135.

3. Gordon P. Endometrial biopsy. *N Engl J Med*. 2009;361:e61.

CHAPTER

12

Gynecologic Disorders

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Introduction

Often members of the public envision midwives exclusively as providers of maternity care. That perception is false—and has been for many years.[1] Even in past millennia, midwives were known as healers of their communities and cared for women, and sometimes men, with a variety of ailments. Maternity care and gynecologic care frequently overlap, as may occur during the care of a woman who has an abnormal Pap test during pregnancy. Surveys of certified nurse-midwives (CNMs) and certified midwives (CMs) have found that almost 70% of those in clinical practice offer healthcare services to nonpregnant women.[2]

The word *gynecology* originates from the Greek language and means "the study of women." Although it usually is defined as health care focusing on diseases of women, gynecology today connotes care of nonpregnant women and centers on the maintenance of healthy reproductive organs and the treatment of women with various conditions that may negatively affect the reproductive system. This does not imply that care focuses only on issues of childbearing functions of the reproductive system, as not all women plan to become pregnant.

The organs most closely associated with gynecologic health traditionally include the breast, cervix, endometrium/uterus, ovaries, vulva, and vagina. This chapter provides an overview of common gynecologic conditions that biological females experience throughout their lifetime, and the information needed to screen for gynecologic cancers. Because cervical cancer screening is one of the most frequent services performed by midwives, it is discussed first and in detail. Gynecologic conditions such as pelvic pain, pelvic masses, and other disorders of reproductive organs are reviewed as well. Lastly, multiple-organ conditions such as infertility and sexual disorders are addressed. Although breast conditions are usually considered part of gynecology, they are discussed in detail in the *Breast Conditions* chapter.

Among the most important skills of any midwife is the ability to individualize care, particularly based on a person's culture, gender identity, age, physical/mental characteristics, and risk factors for disease. Recognition of personal beliefs and having the ability to overcome implicit biases are necessary to provide person-centered care.[3,4]

Historically, the annual gynecologic examination has often been for the purpose of obtaining an annual Pap test. However, professional organizations such as the U.S. Preventive Services Task Force have found there is inadequate evidence supporting regular pelvic examinations for asymptomatic women.[5] Today, the conversation has shifted away from the "routine annual Pap test" and toward individualized care and improvement of outcomes based on the individual's health needs. Furthermore, advancements in Pap test technology and greater acceptance of long-acting contraceptives mean that women of reproductive age do not always need an annual visit, although periodic visits for health screening remain cornerstones of preventive care.

Screening for Malignancies

Breast, lung, and colon cancer are the most common causes of cancer among women living in the United States, with lung cancer surpassing breast cancer as the leading cause of cancer death in women.[6] Among the malignancies commonly termed “gynecologic cancers,” only cervical cancer can be detected through regular screening of low-risk and at-risk women. Pap testing is one of the most common screening tests performed by midwives. However, screening for cervical cancer has become more complex over the last few years with regard to time of initiation, years of duration for routine screening, and interpretation of results.

Cancer of the Uterine Cervix

Since the 1980s, it has been well recognized that virtually all cases of cervical cancer are caused by infection with a high-risk type of human papillomavirus (HPV).[6] HPV is a small deoxyribonucleic acid (DNA) virus that is easily transmitted from one person to another via close skin-to-skin physical contact or unprotected penetrative intercourse. HPV enters the body through microscopic breaks in the skin and can be transmitted via any type of sexual stimulation.[7,8] While more than 150 genotypes of HPV have been identified, approximately 40 are known to infect the anogenital area. To date, 12 to 14 major HPV types have been linked to the development of cervical cancer, with types 16 and 18 responsible for approximately 70% of all cervical cancers.[9]

The ability to test for high-risk HPV types when using liquid-based Pap tests has changed cervical cancer screening practices. Cervical cancer can take years to develop, thus, population-based screening can be effective for identification of this cancer or precursor lesions. Testing for high-risk HPV types can be performed singly, as a co-test with a Pap test, or as "reflex" testing if the Pap test identifies abnormal cells.

Cervical Cancer Screening Tests

Since the introduction of liquid-based cytology and the advent of testing for high-risk HPV DNA, liquid-based testing has replaced glass-slide techniques in most developed nations. Today, more than 90% of laboratories use liquid-based Pap testing because this technology assures improved quality of the sample, improved reproducibility, fewer false negatives, and the ability to test for HPV.[10]

Pap Test

As described in the *Anatomy and Physiology of the Female Reproductive System* chapter, the external cervix (ectocervix) is covered with smooth, pink, squamous epithelial cells. The inner section of the cervical os is lined with columnar epithelial cells. The squamocolumnar junction is the site of active metaplasia, or conversion of one cell type (columnar epithelium) to another (squamous epithelium), although these changes are not grossly visible. The squamocolumnar junction is clinically important because the cells around it, called the *transformation zone*, tend to be the site of most abnormalities. For this reason, healthcare providers attempt to obtain cells from this area for a Pap test. When abnormalities occur in the cells within the *transformation zone*, they often are characterized by the term *dysplasia*—an umbrella term referring to abnormal cells that may signify a stage preceding the development of cancer.

In women who are pregnant or using hormonal contraception, the presence of pronounced eversion of the columnar epithelium, also known as *ectopy*, often enhances the possibility of obtaining satisfactory cells for a Pap test. Conversely, during late childhood and early adolescence, the squamocolumnar junction is deep inside the endocervix, and the cervix tends to appear only to have squamous cells. Similarly, women who are in the postmenopausal period may lack any visual appearance of the endocervix. Pap testing may be more challenging

for women in these subpopulations. The procedure for performing a Pap test is described in the *Collecting Urinary, Vaginal, Cervical, and Rectal Specimens for Testing and Interpretation of Saline and KOH Slides* appendix in the *Introduction to the Care of Women* chapter.

Human Papillomavirus DNA Test

In 2014, the U.S. Food and Drug Administration (FDA) approved the use of a specific brand of HPV DNA test (cobas) as a stand-alone screen for cervical cancer in women age 25 years and older. A large trial involving more than 42,000 women, conducted in the United States, demonstrated the effectiveness and sensitivity of HPV infection as a first-line screening test for cervical cancer, finding that it yielded fewer false-negative results when compared with Pap testing.[11] In 2015, the American Society for Colposcopy and Cervical Pathology endorsed use of HPV DNA testing for primary screening; an algorithm that provides guidance for clinicians using HPV-only testing for primary screening is also available.[12-14] When choosing HPV DNA testing for primary cervical cancer screening, it is essential that the referral laboratory use only the FDA-approved HPV test (cobas).

Cervical Cancer Screening Recommendations

Current age-based cervical cancer screening guidelines for all women, including those who have been either fully or partially vaccinated against HPV, are recommended as summarized in **Table 12-1**.[14-15] HPV vaccination does not negate the need for continued cervical cancer screening.

Table 12-1 Cervical Cancer Screening Guidelines for Women at Low Risk for Cervical Cancer[a]

Age and Characteristics	ACS, ASCCP, ASCP	USPSTF	ACOG
< 21 years	No screening	No screening	No screening
21–29 years[b]	Every 3 years: conventional cytology without HPV co-test	Every 3 years: conventional cytology without HPV co-test	Every 3 years: conventional cytology without HPV co-test
≥ 30–64 years	Every 3 years: conventional cytology *or* Every 5 years: with HPV co-test	Every 5 years: with HPV co-testing preferred *or* Every 3 years: conventional cytology	Every 5 years: conventional cytology with HPV co-testing preferred *or* Every 3 years: conventional cytology
≥ 65 years	No further screening if prior screening was negative and no other high-risk factor for cervical cancer exists	No further screening if prior screening was negative and no other high-risk factor for cervical cancer exists	No further screening if no abnormal tests in past 10 years
≥ 65 years with history of CIN II, CIN3, or AIS	Continue screening for 20 years after regression or management		Continue screening for 20 years after regression or management
After hysterectomy with removal of cervix	No screening unless positive history of high-grade precancerous lesion or cervical cancer	No screening unless positive history of high-grade precancerous lesion or cervical cancer	No screening unless positive history of high-grade precancerous lesion or cervical cancer
HPV-vaccinated	Regular age-specific screening	Regular age-specific screening	Regular age-specific screening

Abbreviations: ACOG, American College of Obstetricians and Gynecologists; ACS, American Cancer Society; AIS, adenocarcinoma in situ; ASCCP, American Society for Colposcopy and Cervical Pathology; ASCP, American Society for Clinical Pathology; CIN, cervical intraepithelial neoplasia; DES, diethylstilbestrol; HIV, human immunodeficiency virus; HPV, human papillomavirus; USPSTF, U.S. Preventive Services Task Force.

[a] These guidelines do not apply to women exposed in utero to DES, or those who have had a diagnosis of a high-grade precancerous cervical lesion, have a diagnosis of cervical cancer, are HIV-positive, or are immunosuppressed.

[b] Biological females 21 years or older who have never had sex are very low risk for developing cervical cancer, and screening in this population is dependent on individual clinician judgment.

Based on Saslow, D, Solomon D, Lawson, H, et al. American Cancer Society, American Society for Colposcopy and Cervical Pathology, and American Society for Clinical Pathology screening guidelines for the prevention and early detection of cervical cancer. *J Low Genit Tract Dis.* 2012;16:175-204[13]; American College of Obstetricians and Gynecologists. Practice Bulletin No. 168: cervical cancer screening and prevention. *Obstet Gynecol.* 2016;128:e111-e130[14]; Moyer V. Screening for cervical cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med.* 2012;156:880-891.[15]

Professional organizations in the United States recommend against performing a Pap test for women younger than 21 years, unless the woman has a history of an immunosuppressed condition (e.g., human immunodeficiency virus [HIV] infection).[13-15] Most HPV types, including 16 and 18, resolve spontaneously in younger women and do not increase the risk of cervical cancer later in life unless HPV infection remains persistent over time. If testing occurs inadvertently for a woman younger than 21 years, and the result was either *atypical squamous cells of undetermined significance* (ASC-US) or low-grade squamous intraepithelial lesion (LSIL), a repeat cytology can be performed in 1 year. Alternatively, if the result of the original Pap test is high-grade squamous intraepithelial lesion (HSIL), then a colposcopy is warranted.[15-17]

For women between ages 21 and 29 years, Pap test alone is recommended every 3 years. HPV DNA testing is recommended only for women who have Pap test results showing ASC-US. For women 30 years or older, the recommendation is to perform either co-testing with cytology and HPV DNA testing every 5 years, or Pap test alone every 3 years.[13,14]

Cervical cancer screening is discontinued in women age 65 years or older unless they meet the criteria noted in **Table 12-1**. Women in this age group who have undergone a hysterectomy that included removal of the cervix and have no history of a past high-grade cervical lesion (cervical intraepithelial neoplasia [CIN] 2, 3, or greater) no longer need a Pap test.[13,14]

Interpretation of Cervical Cancer Screening Results

Interpretation of cytology findings, as reported by a properly accredited laboratory, follows the internationally accepted Bethesda Classification System of cervical cytological reporting, which was last updated in 2014 (**Table 12-2**).[18] This classification is based on *cytology*, rather than *histology*. Cytology is the study of function and structure of individual cells, such as cells found on a Pap test; by comparison, histology is the study of the microscopic structure of tissue specimens, belonging to a specific organ, usually obtained via a biopsy. The diagnosis and staging of cervical cancer findings are performed histologically, and necessitate testing beyond that involved in a Pap test—most often a biopsy obtained during a follow-up colposcopic examination or other operative procedure. For women with abnormal Pap test results, the information should be shared with sensitivity because, although cytology results are often revealed ultimately to be normal when a histologic examination is performed, such a result can be a source of anxiety for women.

Table 12-2	**The 2014 Bethesda System**
Specimen Type	
Indicate conventional smear (Pap smear) versus liquid-based preparation versus other	
Specimen Adequacy	
Satisfactory for evaluation (*describe presence or absence of endocervical/transformation zone component and any other quality indicators [e.g., partially obscuring blood, inflammation]*)	
Unsatisfactory for evaluation (*specify reason*)	
Specimen rejected/not processed (*specify reason*)	
Specimen processed and examined, but unsatisfactory for evaluation of epithelial abnormality because of (*specify reason*)	
General Categorization (*Optional*)	
Negative for intraepithelial lesion or malignancy	
Other: See Interpretation/Result (*e.g., endometrial cells in a woman ≥ 45 years*)	
Epithelial cell abnormality: See Interpretation/Result (*specify* “squamous” *or* “glandular” *as appropriate*)	
Interpretation/Result	
Negative for Intraepithelial Lesion or Malignancy (*When there is no cellular evidence of neoplasia, state this in the General Categorization above and/or in the Interpretation/Result section of the report—whether or not there are organisms or other non-neoplastic findings*)	

Non-Neoplastic Findings (Optional to Report)

Non-neoplastic cellular variations

- Squamous metaplasia
- Keratotic changes
- Tubal metaplasia
- Atrophy
- Pregnancy-associated changes

Reactive cellular changes associated with:

- Inflammation (includes typical repair)
- Lymphocytic (follicular) cervicitis
- Radiation
- Intrauterine contraceptive device (IUD)

Glandular cells status post hysterectomy

Interpretation/Result

Organisms

Trichomonas vaginalis

Fungal organisms morphologically consistent with *Candida* spp.

Shift in flora suggestive of bacterial vaginosis

Bacteria morphologically consistent with *Actinomyces* spp.

Cellular changes consistent with herpes simplex virus

Cellular changes consistent with cytomegalovirus

Other

Endometrial cells (*in a woman age ≥ 45 years*) (*Specify if "negative for squamous intraepithelial lesion"*)

Epithelial Cell Abnormalities

Squamous cell

Atypical squamous cells

- Of undetermined significance (ASC-US)
- Cannot exclude HSIL (ASC-H)

Low-grade squamous intraepithelial lesion (LSIL) (*encompassing: HPV/mild dysplasia/CIN 1*)

High-grade squamous intraepithelial lesion (HSIL) (*encompassing: moderate and severe dysplasia, CIS; CIN 2 and CIN 3*)

- With features suspicious for invasion (*if invasion is suspected*)

Squamous cell carcinoma

Glandular cell

- Atypical
- Endocervical cells (not otherwise specified [NOS] *or specify in comments*)
- Endometrial cells (NOS *or specify in comments*)
- Glandular cells (NOS *or specify in comments*)

Atypical

- Endocervical cells, favor neoplastic
- Glandular cells, favor neoplastic

Endocervical adenocarcinoma in situ
Adenocarcinoma
Endocervical
Endometrial
Extrauterine
Not otherwise specified (NOS)
Other malignant neoplasms (specify)
Adjunctive Testing
Provide a brief description of the test method(s) and report the result so that it is easily understood by the clinician.
Computer-Assisted Interpretation of Cervical Cytology
If the specimen is examined by an automated device, specify the device and result.
Educational Notes and Comments Appended to Cytology Reports (*Optional*)
Suggestions should be concise and consistent with clinical follow-up guidelines published by professional organizations (references to relevant publications may be included).

Abbreviations: ASC-US, atypical squamous cells of undetermined significance; ASC-H, atypical squamous cells, cannot exclude a high-grade squamous intraepithelial lesion; CIN, cervical intraepithelial neoplasia; CIS, carcinoma in situ; HPV, human papillomavirus; spp, species.

Reproduced with permission from Nayar R, Wilbur DC. The Pap test and Bethesda 2014. *Cancer Cytopathol.* 2015; 123:271-281.[18]

Miscellaneous Organisms Identified on Pap Test

Pap testing is a screening test designed to detect cervical cancer. However, when reviewing specimens, cytologists and pathologists may note the presence of incidental organisms unrelated to cervical cancer, such as *Trichomonas*, *Candida*, coccobacilli, or the presence of endometrial cells. When *Trichomonas* is identified on a Pap result, this finding is not considered diagnostic for infection that requires treatment because false negatives and false positives are known to occur. In such a case, it is recommended that the midwife recall the woman for confirmatory testing by office-based examination of vaginal secretions via saline prep wet mount, or preferably via nucleic acid amplification testing (NAAT).[19]

Candida found on a Pap test does not necessarily require treatment either. *Candida* forms colonies intravaginally as a component of normal flora, and the presence of this organism on a Pap test report does not correlate well with the woman's clinical presentation. If she reports symptoms of vaginal candidiasis, signs of vulvovaginitis are present, fungal elements are confirmed on microscopic examination, and potassium hydroxide (KOH) testing is positive, then treatment is indicated.

Similarly, Pap tests may report cellular changes associated with the presence of herpes simplex virus (HSV). However, a Pap test is neither a screening nor a diagnostic test for herpes.[19] Obtaining an HSV culture of a visible lesion and testing by polymerase chain reaction (PCR) remain the most accurate methods of identifying herpes. In the asymptomatic woman, type-specific serology will determine the presence of antibodies (evidence of past exposure) to herpes but a positive serology result does not provide information about a current

outbreak or when the woman may have been exposed to herpes. The Centers for Disease Control and Prevention (CDC) recommends against routine serologic screening for herpes in the general population. If a Pap test result indicates the presence of HSV, the woman should be notified, and a discussion regarding further diagnostic testing should occur.

Many laboratories report a predominance of coccobacilli consistent with a shift in vaginal flora. Some clinicians consider this finding suggestive of bacterial vaginosis. Bacterial vaginosis is multibacterial in origin and cannot be diagnosed via cytology. When coccobacilli are identified on a Pap report, the midwife can inquire whether the woman is symptomatic, and offer an examination to assess for bacterial vaginosis. Current research findings are inconsistent concerning the value of screening asymptomatic women for bacterial vaginosis.

A finding of *Actinomyces* on a Pap test result for a woman who is using intrauterine contraception should be further evaluated. *Actinomyces* is a strain of bacteria found in the normal genital tract. In rare cases, its presence has been associated with the development of pelvic inflammatory disease and pelvic abscess in women using intrauterine contraception. If infection is likely or the woman is symptomatic, the intrauterine device (IUD) should be removed and antibiotics initiated. If the woman is asymptomatic, the device can be left in place, and antibiotic treatment is not indicated.[20,21]

To summarize, the purpose of the Pap test is to detect cervical cancer and categorize epithelial cell abnormalities. The Pap test is a *screening*, rather than a diagnostic test. If the clinician visualizes an unusual lesion or mass on the cervix, a Pap test can be performed as an initial step in evaluation. While a variety of incidental findings may be noted on a Pap report, these findings are not diagnostic. Additional follow-up may be needed depending upon the report, the woman's symptoms, and/or abnormal findings during the examination.

Alternative Methods of Cervical Cancer Screening in Low-Resource Areas

In many countries without Pap test screening systems, cervical cancer is a significant cause of death among women. An alternative method used for screening is visual inspection with either a 5% solution of acetic acid or Lugol's iodine solution. This technique involves the application of the agent during a speculum examination and examination of the cervix with the naked eye to identify lesions that appear acetowhite when exposed to acetic acid, or bright yellow when exposed to Lugol's solution. This test is usually conducted by trained lay health workers, making the technique cost-effective and accessible to large populations of women who might not otherwise be screened during their lifetime. Referral is indicated after discovery of abnormal lesions.[22,23]

Another method to screen for cervical cancer sometimes used in low-resource countries is cervicography. This method uses low-magnification photographs of the cervix, now available in digital format, which are interpreted by a cytopathologist, frequently at a different site and later in time. Women with abnormal cervigrams are referred for further evaluation and treatment. This method is not as sensitive or specific as colposcopy, but evidence shows that this technique has a high negative predictive value and serves an important role in cervical cancer screening in locations where cytology screening is unavailable.[24]

Diagnosis and Treatment for Women with Abnormal Pap Test Results

The 2012 guidelines recommended by the American Society for Colposcopy and Cervical Pathology (ASCCP), Society of Gynecologic Oncology (SGO), and American College of Obstetricians and Gynecologists (ACOG) form the basis for management of Pap test findings, as well as the management proposed by ACOG for abnormal cervical cancer screening test results.[13-15] The midwife's role in managing a woman with Pap test results can range from immediate referral to performance of follow-up tests, including colposcopy, depending on the midwife's formal preparation, experience, institutional guidelines, and skill level. If the results of a Pap test include findings such as carcinoma in situ, squamous cell carcinoma, or adenocarcinoma, immediate referral of the woman to a gynecologic oncology specialist is indicated.

Factors such as age, HPV status, pregnancy, previous Pap tests, past treatments, and histologic results may all affect Pap test management, making it complex. For example, the ASCCP guidelines include more than a dozen discrete algorithms based on the Pap test results, describing how to manage women with all of the following: unsatisfactory cytology, absent endocervical cells, ASC-US, LSIL, HSIL, atypical glandular cells, cervical intraepithelial lesions, and adenocarcinoma in situ. More-frequent screening may be indicated for women with HIV and other immunosuppressed conditions such as organ transplant, women exposed to diethylstilbestrol (DES) in utero, and those with a history of prior treatment for CIN 2, CIN 3, or cervical cancer. The ASCCP guidelines are available free of charge and are updated on a regular basis; a link to these guidelines and information about accessing them are provided in the Resources section at the end of this chapter.[13]

Colposcopy

The primary diagnostic method for determining the presence of cervical cancer or precancerous cells is colposcopic examination. Although a detailed description of the procedure is beyond the scope of this chapter, an increasing number of midwives are receiving formal education in performing colposcopy and have formally incorporated the procedure into their clinical practices as congruent with Standard VII in the *ACNM Standards for the Practice of Midwifery*.[25] Principles and standards of colposcopy education and practice for all clinicians are now established and endorsed by several women's health professional organizations.[26] Common indications for colposcopy are listed in **Table 12-3**.[14,17,27]

Table 12-3	Common Indications for Colposcopic Examination of the Cervix
Grossly visible or palpable abnormality of the cervix	
Abnormal cervical cytology	
Positive screening test for cervical neoplasia (e.g., high-risk HPV DNA strain)	
Persistent unsatisfactory cervical cytology	
History of in utero DES exposure	
Unexplained cervicovaginal discharge	
Unexplained abnormal lower genital tract bleeding	

Surveillance for lower genital tract neoplasia (cervical, vaginal, vulvar)
Post–lower genital tract cancer treatment surveillance

Abbreviations: DES, diethylstilbestrol; DNA, deoxyribonucleic acid; HPV, human papillomavirus.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 168: cervical cancer screening and prevention: interim update. *Obstet Gynecol.* 2016;128:e111-e130[14]; Massad L, Einstein M, Huh K, et al., for 2012 ASCCP Consensus Guidelines Conference. 2012 updated consensus guidelines for the management of abnormal cervical cancer screening tests and cancer. *J Low Genit Tract Dis.* 2013;17:S1-S27[17]; Ocque R, Austin M. Follow-up of women with negative Pap test results and abnormal clinical signs or symptoms. *Am J Clin Pathol.* 2016;145:560-567.[27]

When lesions of the vulva, vagina, or ectocervix are identified during colposcopy, a biopsy is performed at the same time. Endocervical curettage is recommended in certain situations (e.g., no lesion is found during a satisfactory colposcopy examination) to evaluate tissue not easily visible on the ectocervix. The purpose of a tissue biopsy or endocervical curettage is to rule out cancer or precancerous lesions, and subsequently guide treatment.

The choice of treatment depends on the size, location, and significance of the abnormality (CIN 1, 2, 3, or cancer), the woman's age, and her desire for future childbearing. As with any gynecologic procedure, it is important to engage in shared decision making and obtain signed informed consent.

Gynecologic Evaluation and Pelvic Pain

Reports of abdominal and/or pelvic pain are common motivations for seeking gynecologic care and account for a significant number of visits to healthcare providers. This section introduces the initial gynecologic evaluation and common differential diagnoses in women of reproductive age.

Acute Pain

Acute abdominal or pelvic pain is a medical emergency. The ability to provide safe care for a woman experiencing acute pain requires that the midwife have relationships with surgical and nongynecologic providers and a system for referral. Careful screening and triage must be done to determine the optimal setting for evaluation of acute pain to ensure appropriate management; this setting may be an emergency department or a targeted visit in an office setting, depending on how stable the woman is and which other resources are available or needed for a thorough evaluation. Immediate consultation/referral is indicated for a woman with acute pain, especially if her pain is accompanied by fever, tachycardia, significant changes in blood pressure, signs of shock, vomiting, unstable vital signs, or evidence of significant blood loss.

Chronic Pain

Chronic pelvic pain may arise secondary to one or several underlying disorders, and it can be difficult to evaluate and treat.[28-30] The woman with chronic pelvic pain may have a history of multiple encounters with healthcare providers. It is crucial to know if a woman has been evaluated for chronic pain previously and to clarify her expectations for midwifery involvement in care. Because chronic pelvic pain is associated with multiple underlying etiologies, women with chronic pain are often best cared for by gynecologists who specialize in this condition. A general understanding of chronic pelvic pain can help a midwife participate in evaluation and team-based care for these women.

Sexual Abuse and Assault

A person who has experienced sexual abuse or trauma may have unique gynecologic needs. When caring for individuals who have been sexually abused or assaulted, it is essential for the midwife to provide care that addresses the person’s needs in a manner that does not add to physical discomfort, emotional distress, or trigger a recurrent sense of trauma.[31] An objective and straightforward explanation of the examination should occur before the woman takes off her clothes. Patience and use of skills in facilitating relaxation are indispensable. If abuse is known or revealed during the visit, the midwife should offer to ensure the presence of another staff member or a support person for the examination, in an effort to give the woman a feeling of some control over the procedure.

If it is difficult for the woman to proceed with a physical examination, it may be conducted in smaller steps, over more frequent encounters when possible, unless there is concern about an acute or emergent problem. If the problem appears to be acute, the midwife should assess

which type of examination other providers may need to do and limit the current examination to only those elements that are absolutely necessary for consultation or referral. For example, if a woman has been recently assaulted, it may be necessary to ask a Sexual Assault Nurse Examiner (SANE) to conduct the examination. Some midwives are SANE certified and may perform a complete forensic examination, including collection of evidence.

Regardless of the reason for the visit, the midwife should clearly acknowledge the person's experience and offer the office as a safe haven for exploration and healing.[32] Suspicion or evidence of physical or sexual abuse or assault requires the midwife to follow institutional guidelines and laws of the jurisdiction where the midwife is practicing, including those that dictate the legal authority to which the midwife must report any suspicion or evidence of abuse or assault.

Differential Diagnoses Associated with Pelvic Pain

Multiple disorders are associated with abdominal pain, pelvic pain, or both. The list of candidates varies depending on a person's age (e.g., premenopausal or postmenopausal) and other demographic characteristics. Common differential diagnoses to be considered for individuals of reproductive age who present with acute or chronic abdominal or pelvic pain are listed in **Table 12-4**.[33] This list can be used to suggest components of the physical examination. Because the list of differential diagnoses is large, three initial assessments will help focus the direction of the evaluation:

Table 12-4	Selected Differential Diagnoses of Acute or Chronic Abdominal and/or Pelvic Pain in Women[a]
System	**Differential Diagnoses**
Gastrointestinal	Appendicitis Bowel obstruction Crohn's disease Constipation Diverticulitis Gallbladder obstruction Inflammatory bowel disease or irritable bowel disease Pancreatitis
Musculoskeletal	Fibromyalgia Herniated disc Hernia Pelvic pain syndrome Sprain or strain
Gynecologic, acute pain in nonpregnant woman, not at risk for pregnancy	Endometriosis (rupture of endometrioma) Dysmenorrhea Intrauterine device imperforation Mittelschmerz Ovarian cyst Pelvic inflammatory disease

	Torsion or degeneration of uterine fibroids
Gynecologic, chronic pain in nonpregnant woman, not at risk for pregnancy	Adenomyosis Adhesions Endometriosis Malignancy Prior pelvic inflammatory disease Ovarian cancer Uterine fibroid
Gynecologic, fertility treatment	Ectopic pregnancy Ovarian hyperstimulation syndrome Ovarian cyst Ovarian torsion
Pregnancy-related	Corpus luteal cyst Ectopic pregnancy Normal pregnancy nausea and vomiting Ovarian torsion Miscarriage Preterm labor or placental abruption
Postmenopausal	Malignancy
Urologic	Interstitial cystitis Pyelonephritis Urinary retention/obstruction Urinary tract infection

[a] This list is not comprehensive. These disorders are included here because they are either commonly encountered or not common but associated with severe morbidity and, therefore, essential considerations during an evaluation.

1. Is the woman premenopausal or postmenopausal?
2. Could she be pregnant or attempting pregnancy?
3. What are the characteristics of the pain and associated symptoms?[34]

Evaluation of a Woman with Abdominal/Pelvic Pain

In all settings, the role of midwifery care when evaluating a person with abdominal or pelvic pain includes a thorough history, physical examination, initial laboratory evaluation, and referral for specialist care as needed.

A helpful tool in the evaluation of any symptom, including pain, is the OLD CARTS system (**Table 12-5**).[34] Use of common validated questionnaires is also recommended when assessing a woman with chronic pain.[35] Some women will answer questionnaires in detail and provide a wealth of information; however, others who have not considered their pain in a comprehensive manner may need to return for a future visit after completing a pain diary or history.

Table 12-5 OLD CARTS Mnemonic for Obtaining a History of Reported Pain

Desired	Questions to Be Asked

Information	
Onset	When did the pain begin? Can the woman identify a particular event or point in time related to the initial recognition of the pain?
Location/radiation	Specifically identify the location of the pain. Does the pain move or radiate or have a different quality in varying locations?
Duration	Is the pain cyclic or noncyclic? Is it related to other health factors (e.g., bowel function, physical activity, sexual activity, menstrual cycle)? Is the onset of the pain related to diet or diet changes? Is it related to work activities? Is it related to the woman's emotional state of being or to the onset of emotional stress?
Character	Ask the woman to describe the pain. Offer a wide variety of descriptors used for pain, such as *sharp, dull*, *gnawing*, *burning*, or *achy*.
Aggravating factors	What makes the pain worse (e.g., exercise, diet, constipation, diarrhea, urination, physical activity, sexual activity, menses)?
Relieving factors	What relieves the pain (e.g., rest, diet change, bowel movement), and to what degree? Which treatments have been used to help alleviate the pain? Were they recommended by other providers or suggested by a family member, friend, book, or website? Were medications used (over-the-counter and/or prescription)? Were any alternative treatment modalities used? What was the success or failure of any treatments that were used?
Timing factors	How often is the pain occurring? Was there a single incident, or is the pain intermittent or chronic in nature? Have the symptoms improved or worsened over time? Is there a specific time of day when the pain is felt more often?
Severity	How severe is the pain on a scale of 0 to 10, with 10 being the worse pain the woman can imagine? Has the pain had an effect on the woman's lifestyle? On her ability to work?

Based on Ball J, Dains J, Flynn J, Solomon BS, Stewart RW. *Seidel's Guide to Physical Examination.* 8th ed. St. Louis, MO: Mosby Elsevier; 2015.[34]

Many causes of acute pain can become causes of chronic pain. For example, 30% of women with pelvic inflammatory disease develop chronic pelvic pain.[36] Finally, it is always important to ask two initial question: What does the individual think is causing the pain? And, how is the pain affecting their quality of life? If the initial history results in an inability to diagnose the cause of pain and the woman wishes to continue investigation with the midwife, a more detailed history of the pain can be suggested. Assessment modalities may include anatomic drawings used to locate the pain; the use of a pain diary with a pain severity scale; or the use of a journal to additionally chart the pain in relation to daily activities, menstrual cycle, bowel habits, sexual activity, and diet.

Physical Examination

Examination of a woman presenting with pelvic pain may require a complete physical examination. Many gynecologic disorders will manifest in different ways, including pain in the abdomen, in the pelvis, or both. If a more narrowly focused examination is indicated, vital signs and both abdominal and pelvic examinations should be performed, and the results documented in the woman's health record. In the absence of an acute presentation, a complete examination of systems can be performed. The woman's demeanor and presentation are included in this evaluation. Evaluation of the back and lower extremities is included to rule out a musculoskeletal injury, such as a muscle strain or stress fracture, or an anatomic variation,

such as scoliosis.

Abdominal Examination

The goal in palpation of the abdomen is to identify organ enlargement, displacement, masses, or enlarged lymph nodes as well as to ascertain if and where the pain can be elicited. The midwife can ask the woman if she can elicit the pain herself. If not, it is advisable to focus on the unaffected side or area first. Communicating findings and letting the person know which part of the examination is next can help maintain an atmosphere of trust and caring. Special attention should be paid to any area of tension or guarding, and knowledge of patterns of referred pain points is useful as well.

Pelvic Examination

At the time of visual inspection of the vulva and vagina, signs of swelling, lesions, trauma, or other skin changes can be assessed. The woman's ability to tolerate a speculum insertion with or without pain is an important observation, especially if she reports pain with sexual activity. After speculum insertion, a sample of vaginal discharge is collected for a saline or KOH examination or cultures, and subsequent testing for sexually transmitted infections (STIs) if indicated. Visual inspection of the cervix may reveal a partially expelled IUD, signs of infection, color change indicative of early pregnancy, laceration, a polyp, or other abnormality.

After the speculum examination is complete, a gentle bimanual examination enables the midwife to observe whether the woman exhibits discomfort at the introitus versus deep in the vagina or pelvis. Any cervical, adnexal, or uterine motion tenderness elicited provides further information in addition to signs of organ displacement.

Palpation for any masses, uterine fibroids (leiomyomata), or uterine or adnexal enlargement indicative of pregnancy is the next step. Assessment of pelvic muscle tone is performed, and the presence of a cystocele, a rectocele, or uterine prolapse is ascertained. Confirmation of findings can be accomplished with a rectal examination as well as assessment for hemorrhoids, polyps, or masses.

Recorded descriptions of a mass or enlarged organ should include the size, shape, location, consistency, mobility, tenderness, and relationship of the mass to other organs. Additionally, if pain is elicited, its intensity and location should be described. If the woman's pain limits the midwife's ability to perform full palpation and/or pelvic examination, that finding should be documented as well.

Laboratory/Screening/Diagnostic Tests

Only tests that will generate results which could directly influence management should be performed. A pregnancy test is inexpensive and generally indicated for any sexually active reproductive-age woman regardless of contraceptive method used. A complete blood count is often ordered as a standard test; however, in the absence of heavy vaginal bleeding, an infectious process, or concerns about anemia, this test may not be useful. If the cause of the pain is considered urologic in nature, a urinalysis or urine culture may be informative. Tests

for STIs or vaginitis may be indicated by history and examination. A fecal occult blood test may aid in the management of any abdominal mass or gastrointestinal condition. Any fetal occult blood test that is positive should result in referral to a specialist for further evaluation and possible colonoscopy or sigmoidoscopy testing.

A pelvic or abdominal ultrasound is commonly used to aid in the diagnosis of uterine or adnexal abnormalities. If an ultrasound is ordered, a transvaginal ultrasound may be more accurate than a transabdominal approach. For women who have never experienced vaginal penetration, women who have cultural prohibitions with regard to pelvic procedures, or those with a personal history of sexual assault trauma, this part of the test may be distressing. Anticipatory guidance includes an explanation of the reason for using ultrasound and shared decision making. If the woman wishes to have the ultrasound performed, it is important to alert staff who may be working with the woman that this is an initial transvaginal pelvic ultrasound and that additional patience and time may be necessary.

Adnexal Masses in Reproductive-Age Women

Adnexal masses are common and frequently found incidentally during a regular pelvic examination. The differential diagnosis of an adnexal mass includes a physiologic cyst (e.g., corpus luteum), ectopic pregnancy, benign ovarian neoplasms (e.g., dermoid cyst), ovarian cancer, tubo-ovarian abscess, endometriosis, or ruptured cyst.[37] Adnexal masses may be found in both premenopausal and postmenopausal women. Women may be asymptomatic or they may present with symptoms of pelvic pressure, pain, or dyspareunia. Some symptoms associated with ovarian cysts may be vague, such as abdominal bloating, a sensation of fullness, or, possibly, urinary frequency or retention.

Pelvic examinations are often inadequate to determine the size, location, and characteristics of an adnexal mass. Such examinations are especially inadequate for identifying ovarian cysts in women with obesity. Ultrasound and magnetic resonance imaging (MRI) are the most commonly used imaging modalities for assessment of the size, character, and malignant characteristics of adnexal masses.

Corpus Luteum Cysts

Corpus luteum cysts develop following hemorrhage of the corpus luteum, most often during days 20–26 of the menstrual cycle. Ultrasound may confirm the diagnosis. In the absence of bleeding, the cyst will regress spontaneously. In the event of significant bleeding, or with acute pain secondary to the bleeding, a negative pregnancy test may assist in differentiating a ruptured corpus luteum cyst from an ectopic pregnancy. If bleeding continues, surgical intervention may become necessary.

Ectopic Pregnancy

Ectopic pregnancy is any pregnancy implanted outside the endometrial cavity. An ectopic pregnancy is considered a life-threatening emergency, even if the woman appears stable and asymptomatic. Thus, ectopic pregnancy is one of the first differential diagnoses to be considered in any reproductive-age woman. Suspicion for an ectopic pregnancy should be high if a woman reports amenorrhea or intermittent abdominal or pelvic pain of varying duration and intensity. Furthermore, women who present with any of these symptoms in addition to shoulder pain (i.e., referred pain secondary to the presence of blood in the abdominal cavity, fever, tachycardia, or low blood pressure) must be evaluated for shock and emergency referral even in the absence of vaginal bleeding. The woman with an ectopic pregnancy may have not suspected she was pregnant and may be required to integrate a great deal of information at a very stressful moment. More discussion of ectopic pregnancy is found in the *Pregnancy-Related Conditions* chapter. **Table 12-6** summarizes the elements to be included in a transfer note for a woman who may have an ectopic pregnancy.

Table 12-6 Critical Elements for a Transfer Note for a Woman Suspected with an Ectopic Pregnancy

Signs and symptoms suggestive of ectopic pregnancy, including last menstrual period and any risk factors (e.g.,

pregnancy with intrauterine device in situ)

Care provided before transfer: procedures and results; laboratory results or if pending; pregnancy test results and sonography findings

Assessment/diagnosis of the situation

Summary of rationale for transfer and request for information about the treatment plan for follow-up

Ovarian Cysts

The management of women with ovarian cysts is based on the presence of symptoms, results of imaging, the woman's age, medical history, physical examination, and blood work. The cyst is first classified as simple or malignant based on features identified on ultrasound.[37] Cysts that have both benign and malignant features require further evaluation to determine the risk for malignancy. Simple ovarian cysts that are thin walled, unilocular, and small are usually benign, but may best be followed with periodic ultrasound assessment. The Society of Radiologists in Ultrasound has published guidelines for management of asymptomatic ovarian cysts detected on ultrasound; these guidelines are based on the woman's premenopausal or postmenopausal status, size, and characteristics.[38,39]

Dermoid Cysts

Dermoid cysts or cystic teratomas are asymptomatic, unilateral ovarian masses that arise from all three germ cell layers (ectoderm, endoderm, and mesoderm) and, therefore, may contain skin, bone, hair, and teeth. Dermoid cysts are generally asymptomatic and first identified during a pelvic examination or ultrasound. Such cysts do not regress and have a small chance of becoming malignant; therefore, the treatment is surgical removal.

Ovarian Torsion

An uncommon cause of acute pelvic pain is ovarian torsion, also referred to as adnexal torsion. While causes of torsion include previous adnexal surgical manipulation (especially tubal ligation), an adnexal structural anomaly, or pregnancy, the majority of cases are related to ovarian or adnexal masses. The woman may present with specific pain or more generalized unilateral pelvic or flank pain, as well as nausea and vomiting.[40] This condition often can be confused with gastrointestinal disorders.

The treatment of a woman with ovarian torsion is surgery, during which the ovary is fixed in place or removed depending on the etiology, if evident, and the presence of tissue damage or necrosis. False-positive diagnoses of ovarian torsion based on ultrasound are not uncommon. In such a case, the pain may be due to endometriosis, ovarian enlargement without torsion, or unknown causes not readily apparent.

Disorders of Uterine Origin

The most common disorders associated with the uterus include endometriosis and uterine fibroids. A less common disorder is adenomyosis.

Endometriosis

Endometriosis is the presence of endometrial tissue outside of the uterine cavity. Although it has been difficult to gauge the prevalence of this condition, it is estimated to affect as many as 15% of women of childbearing age.[41] The incidence is higher in women who present with infertility and chronic pelvic pain. Associated risk factors include a family history, early age at menarche, frequent menses, nulliparity, a history of other pain syndromes (e.g., interstitial cystitis, bladder pain syndrome), inflammatory bowel disease, and a diagnosis of immunologically impaired conditions (including asthma and allergies).[41]

The American Society for Reproductive Medicine (ASRM) categorizes endometriosis into one of four stages depending on location(s), depth of the endometrial lesions (implants), extent of the implants, presence of adhesions, and presence of ovarian endometriomas (a type of ovarian cyst):[42]

- Stage I: Minimal; characterized by few implants and no adhesions
- Stage II: Mild; characterized by superficial implants, and no significant adhesions
- Stage III: Moderate; characterized by the presence of both superficial and deep implants, as well as notable adhesions
- Stage IV: Severe; characterized by multiple implants, dense adhesions, and an association with infertility

Unfortunately, these stages do not correlate with the severity of symptoms or infertility.[43] Despite the limitations of the ASRM system, it is helpful as a uniform classification of surgical findings.

The cause of endometriosis remains elusive, and multiple etiologic theories have been proposed, as listed in **Table 12-7**.[44-48] Retrograde menstruation followed by extrauterine implantation remains the oldest and most widely supported theory, although findings of endometriosis implants in pre-menarche adolescents suggest more than one etiology. Findings during laparoscopy have revealed that during menses, endometrial tissue migrates into the peritoneal cavity via the fallopian tubes, attaching itself to the pelvic organs. However, this finding does not explain all cases of endometriosis that have been documented. Indeed, retrograde menstruation commonly occurs, and is more prevalent than the presence of endometriosis. Thus, there is support for theories suggesting that additional factors—such as hormonal, inflammatory, or immunologic conditions—contribute to the actual development of endometriosis in a subpopulation of individuals.[48]

Table 12-7	Proposed Etiologies of Endometriosis

Proposed Etiology	Rationale
Retrograde menstruation	During menses, endometrial tissue migrates into the peritoneal cavity, via the fallopian tubes, attaching itself onto pelvic organs.
Deviations in cellular physiology	Peritoneal tissue is spontaneously transformed into endometrial tissue.
Deviations in lymphatic system	Endometrial tissue is transported to other organs via the lymphatic pathways.
Deviations in immune system	Menstrual tissue found outside the uterus is normally cleared by the immune system, which appears unable to do so in individuals found to have endometriosis.
Deviations in hormonal system	Unlike in intrauterine endometrial tissue, the estrogenic effects of extrauterine endometrial tissue are not influenced by the antagonistic action of progesterone.
Genetics	A 3 to 9 times increased incidence is found among first-degree relatives of affected individuals (i.e., mothers or siblings) compared to the incidence found in the general population.
Environmental influences	Dioxin-like compounds discovered in industrial waste by-products can alter gene development or estrogen–progesterone balance in a manner that leads to extrauterine development of endometrial tissue.

Based on Burney RO, Giudice LC. Pathogenesis and pathophysiology of endometriosis. *Fertil Steril.* 2012;98:511[45]; Rahmioglu N, Nyholt DR, Morris AP, Missmer SA, Montgomery GW, Zondervan KT. Genetic variants underlying risk of endometriosis: insights from meta-analysis of eight genome-wide association and replication datasets. *Hum Reprod Update.* 2014;20:702-716[46]; Parazzini F, Esposito G, Tozzi L, Noli S, Bianchi S. Epidemiology of endometriosis and its comorbidities. *Eur J Obstet Gynecol Reprod Biol.* 2016;209:3-7[47]; Rice K, Secrist J, Woodrow E, Hallock L, Neal J. Etiology, diagnosis, and management of uterine leiomyomas. *J Midwifery Womens Health.* 2012;57;241-247.[49]

Endometriosis is associated with a number of symptoms—most commonly pelvic pain (particularly dysmenorrhea), dyspareunia, and infertility. However, because endometrial tissue can be found in or proximate to a dozen extrauterine sites (**Figure 12-1**), symptoms can also include lower back pain, heavy menstrual bleeding (menorrhagia), irregular menses, pain between menstrual cycles, dysuria, constipation or diarrhea, postcoital bleeding, lower abdominal pain associated with ovarian cysts, and chronic fatigue. Surgeons also may discover endometriosis among asymptomatic women incidental to an unrelated pelvic surgery or procedure.

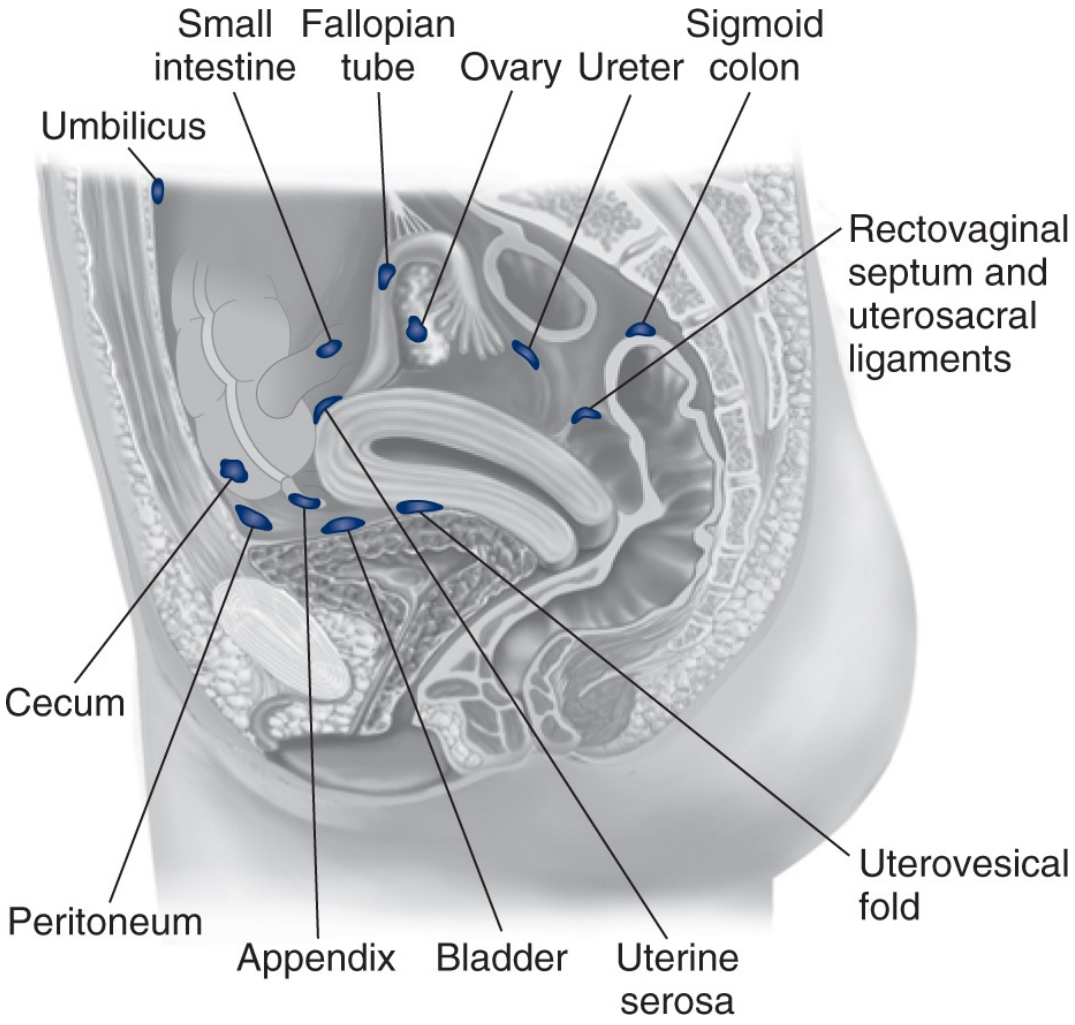


Figure 12-1 Possible locations in which extrauterine endometrial tissue may be found.

Pelvic or lower abdominal pain due to endometriosis may be related to the menstrual cycle, typically appearing at ovulation or intermittently in the time between ovulation and menses. The pain may continue throughout the menses. Pain associated with endometriosis may also occur in patterns unrelated to the menstrual cycle. For example, pain during or after coitus may be so severe as to preclude sexual activities.

Physical examination findings in a woman with endometriosis may be normal, with no significant tenderness associated with organ palpation or movement. In other cases, however, the woman may have cervical motion tenderness, bilateral or unilateral adnexal tenderness, tender nodules on the uterosacral ligaments, ovarian enlargement, or a fixed, retroverted uterus. Definitive diagnosis requires visualization of endometrial implants during surgery. Confirmation of the diagnosis requires biopsy and guides the direction of future treatment. The use of imaging, such as MRI and transvaginal ultrasonography, may provide diagnostic information when the woman has advanced endometriosis; however, these imaging techniques are not useful to identify small or early lesions.

The treatment(s) for endometriosis have mixed success. Providers may select a combination of pharmacologic and surgical therapy, including complete hysterectomy and oophorectomy.[44,48] Pharmacologic treatments include pain relief measures such as use of

nonsteroidal anti-inflammatory drugs (NSAIDs), which suppress hormonal stimulation of the endometrial tissue. Combined or progestin-only hormonal contraceptives and gonadotropin-releasing hormone (GnRH) agonists have the same effects as NSAIDs. Surgical procedures can also be performed to remove lesions. The treatment employed depends on the wishes of the woman including her desire for future childbearing, the degree of pain, the effects of endometriosis on her quality of life, the degree to which her fertility is affected and her age. A multidisciplinary approach is recommended. Small lesions may be removed during the diagnostic laparoscopy followed by hormonal treatment to suppress further growth of the disease, and the use of individually tailored pain management strategies.

Leiomyomas (Uterine Fibroids)

Leiomyomas, also known as uterine fibroids or fibromyomas, are benign smooth muscle tumors that develop from uterine smooth muscle. Uterine fibroids are very common, with a prevalence that ranges from approximately 60% in white women to 80% in black women. As many as 50% of women with fibroids have symptoms, including heavy menstrual bleeding (menorrhagia), pelvic pain, dyspareunia, or urinary frequency if the tumor presses anteriorly on the bladder.[49-51] Thus, half of women with fibroids may never know of the tumors' presence and remain asymptomatic across their lifespan.

Estrogen and progesterone promote fibroid growth and theoretically could facilitate growth in size during pregnancy, although most research has found that fibroids visualized on ultrasound either remain the same size or decrease in size during pregnancy.[51] Generally, fibroids regress after menopause. While fibroids are rarely associated with the development of cancer, any postmenopausal growth of a fibroid or accompanying symptoms, particularly uterine bleeding, warrants evaluation for possible leiomyosarcomas.[51]

Leiomyomas are described on the basis of their anatomic location.

- *Subserosal fibroids* exist just under the uterine serosa and are located outside of the uterus. These growths are attached to the uterus by a large or small base and may be easily palpated on abdominal examination.
- *Intramural* or *myometrial fibroids* are located within the uterine myometrium and may give the uterus an irregular contour.
- *Submucosal fibroids* are located in the uterine endometrium and are usually palpable only as an enlarged uterus.
- *Pedunculated fibroids* present either outside or within the uterine cavity.

When fibroids are present, the uterus will feel firm and irregularly shaped during palpation. If fibroids are suspected during an examination, this finding is shared with the woman and confirmed via an ultrasound examination. The benign nature of fibroids is reviewed, as fears of cancer are possible whenever a woman is told she may have an unexpected mass. **Figure 12-2** depicts fibroids in relation to the uterus as determined by diagnostic imaging or at the time of surgery.

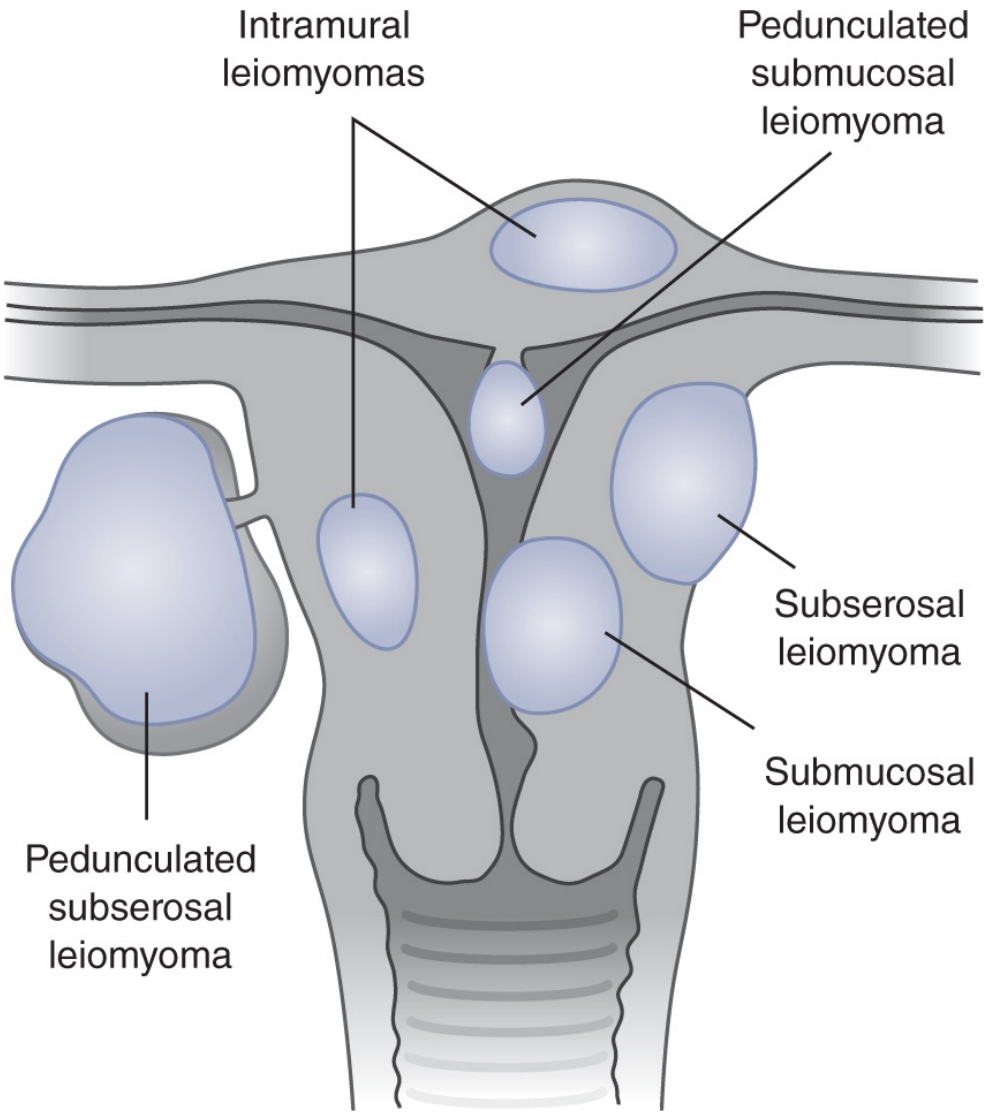


Figure 12-2 Classification of fibroids (leiomyomas) by location in relation to the uterus.

Women with slow-growing fibroids may gradually become accustomed to feelings of pelvic fullness and report few symptoms or little pain, even though fibroids may be large. Other women may present with chronic pelvic pain, lower abdominal pressure, or abnormal uterine bleeding. Fibroids can cause urinary frequency, rectal pressure, interference with sexual activity, and dyspareunia. Depending on their location, these masses can cause infertility, spontaneous abortion, premature labor, malpresentation of a fetus during pregnancy or labor, or an abnormal labor progress.[50,51] Fibroids may also be implicated in abnormal uterine bleeding, especially during the perimenopausal stage of a woman's life. The presence of large fibroids or the degeneration of fibroids may cause pain, including during pregnancy.

Treatment of women with fibroid-related pain depends on the situation and severity of symptoms. Expectant management is recommended for most women, as many fibroids may never be associated with symptoms and most regress after menopause.[50,51] A careful documentation of fibroid size and shape at each annual examination, along with a review of any change in symptoms, is an acceptable management plan.

When intervention is necessary, a variety of nonsurgical interventions, including

pharmacologic agents, can be employed to preserve the uterus.[50,51] Alternative therapies, including the use of acupuncture, bodywork, imagery, and traditional Chinese medicine have been the subject of some research, but results of this body of work are inconclusive.[49,51]

Pharmacologic methods for treating fibroids include the traditional use of GnRH agonists (e.g., leuprolide acetate [Lupron]) as well as the levonorgestrel-releasing IUD. Success rates in diminishing the size or symptoms of fibroids vary considerably with these methods. While the use of GnRH agonists may be most effective, significant side effects preclude their first-line use because they induce menopausal symptoms.[51] However, if surgery is deemed appropriate, a GnRH agonist may be used preoperatively to shrink the fibroid(s). Smaller fibroids are easier to remove, and any reduction in size prior to surgery may give the woman the option of a vaginal versus an abdominal hysterectomy procedure.

Myomectomy is recommended to women with pain or other indications for fibroid removal who also wish to maintain fertility. While a variety of surgical techniques have emerged in recent years, including uterine artery embolization (UAE) and ultrasonography ablation, each method requires individualization depending on the preference of the woman and the characteristics of the fibroid. In the case of large fibroids, persistent heavy vaginal bleeding, or refractory anemia, hysterectomy remains a common treatment option. Midwives should collaborate with specialists in the care of women who are experiencing fibroid-related pain during pregnancy, as a myomectomy during pregnancy may be needed.[49]

Adenomyosis

Unlike fibroids, which are composed of uterine smooth muscle, adenomyosis results from the growth of endometrial tissue in the myometrium layer of the uterus, typically causing uterine enlargement. Because adenomyosis involves growth of endometrial tissue, this condition is considered by some to be a form of endometriosis.[52] Two forms of adenomyosis have been identified: (1) diffuse adenomyosis, in which endometrial tissue is found throughout the myometrium; and (2) focal adenomyosis, in which one or more nodular lesions, known as adenomyomas, are located in specific areas of the uterine myometrium. Diffuse adenomyosis is the more common of the two forms, and is found in approximately two-thirds of diagnosed cases.[53]

Due to difficulty in diagnosing adenomyosis, reports of the incidence of this disorder vary widely—between 20% and 35% of biologic females.[52,53] While many individuals with adenomyosis are asymptomatic, one-third of persons with this diagnosis report menorrhagia, dysmenorrhea, and dyspareunia. In general, most women with adenomyosis have given birth and typically develop the condition in their fourth or fifth decade of life. Transvaginal sonography can be helpful in diagnosing adenomyosis, although the more definitive diagnostic tool is MRI.

Because the symptoms most commonly associated with adenomyosis are heavy menstrual bleeding and dysmenorrhea, traditional pharmacologic treatments include NSAIDs and combined oral contraceptives. However, greater success is reported with use of progestin-only agents, GnRH agonists, tranexamic acid (Lysteda), and levonorgestrel-releasing IUD. Reduction of symptoms with these approaches varies, and the clinician should be familiar with

the risks, benefits, and contraindications of each approach before recommending it. For example, tranexamic acid, a blood clotting agent, is contraindicated for women with a history of venous thromboembolism or women who are using a combined hormonal contraceptive method because tranexamic acid increases the risk of thrombosis. Other approaches include uterine artery embolization and radiofrequency ablation (RFA)—techniques also used to treat fibroids.[54] However, if various therapies have been attempted, with few positive results, the definitive treatment for adenomyosis is hysterectomy.[52]

Uterine Cervical Variations and Disorders

The cervix undergoes cellular maturation and repair processes throughout a woman's lifetime.[55] Inspection of the cervix with the naked eye during the course of a pelvic examination may reveal several normal and abnormal features relative to the woman's age; hormonal status; contraceptive method; stage of pregnancy; and presence or absence of inflammation, infection, or disease. The size and shape of the cervical os is largely determined by parity. **Figure 12-3** and **Figure 12-4** illustrate common variations noted when visualizing the cervix—specifically, nabothian cysts, cervical ectopy, polyps, and cervical lacerations.

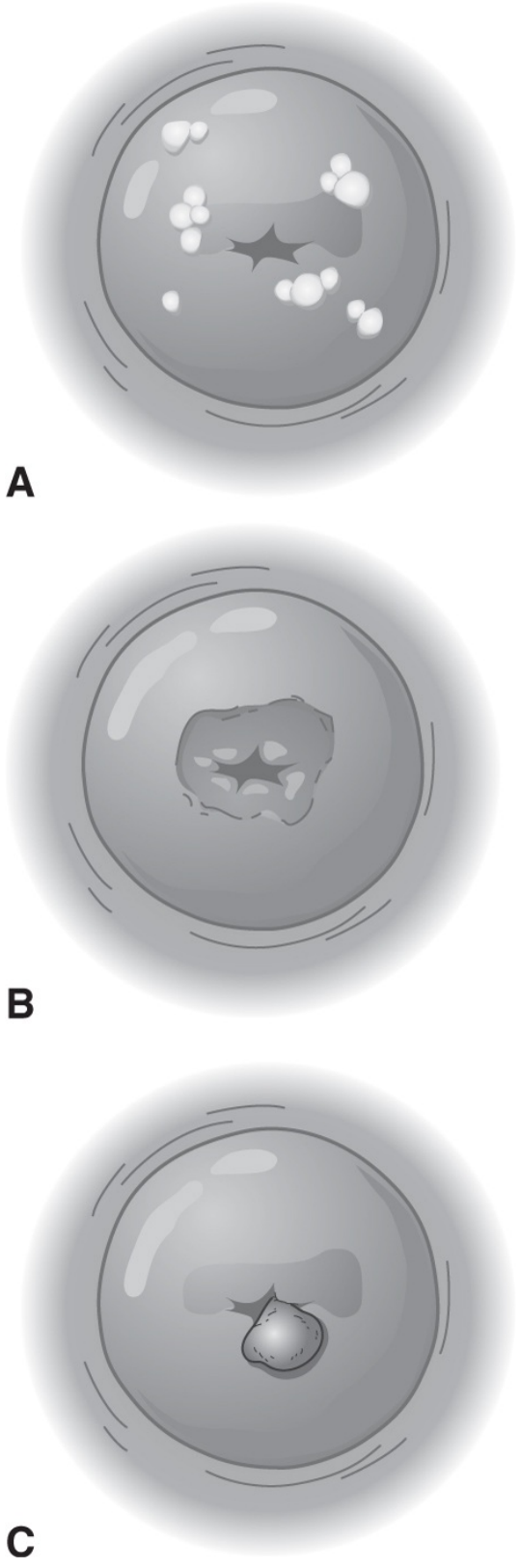


Figure 12-3 **A.** Nabothian cyst. **B.** Cervical ectopy. **C.** Endocervical polyp.

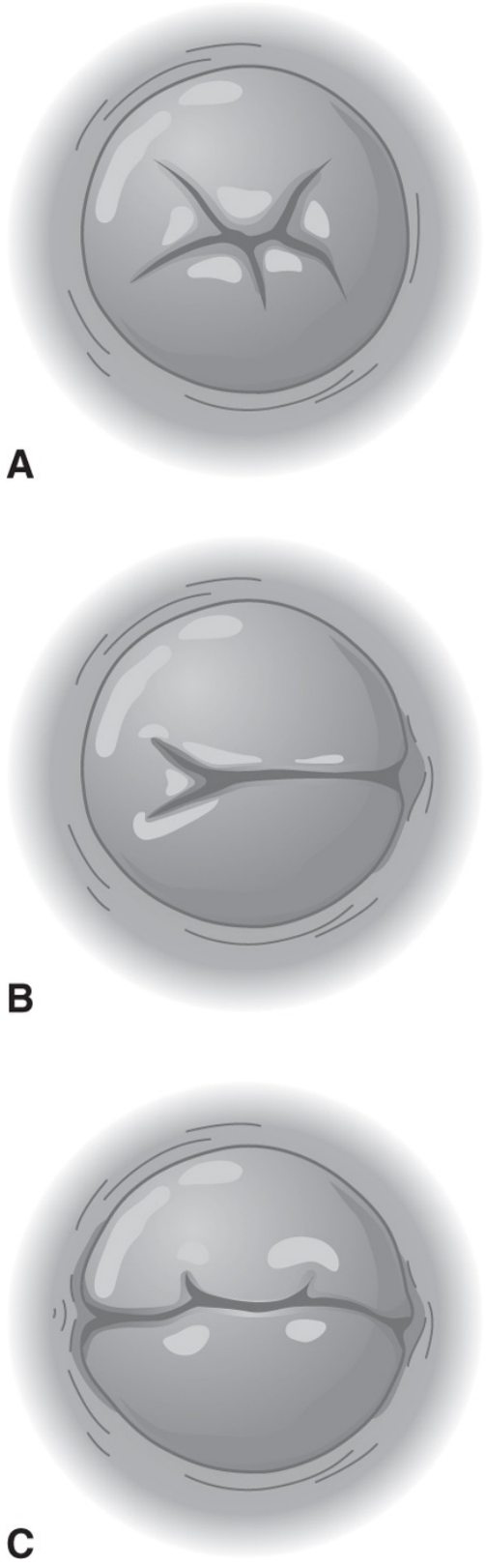


Figure 12-4 Cervical lacerations. **A.** Stellate. **B.** Unilateral transverse. **C.** Bilateral transverse.

Nabothian Cysts

Nabothian cysts, also referred to as mucus cysts or epithelial cysts, are often seen on the cervix

during routine examination. They are a benign and normal feature of the adult cervix. Nabothian cysts arise from glandular, mucus-producing tissue, and are then superficially covered by squamous epithelium. They are typically raised with a yellow or white appearance and may range from a few millimeters to 3 to 4 centimeters in diameter. Most require no treatment unless excessively large.[55] Women checking for presence of an IUD string may feel a nabothian cyst.

Cervical Ectropion (Ectopy)

Cervical ectopy may cause increased vaginal discharge and, in some cases, postcoital bleeding. While cervical ectopy is normal and is not cervical erosion, women with excessive, symptomatic discharge or postcoital bleeding require further evaluation to rule out abnormalities.

Endocervical Polyps

Endocervical polyps often are an incidental finding during the course of a regular pelvic examination. Their typical appearance is that of a small, red, tongue-like growth protruding from the endocervical canal. An endocervical polyp may frequently be the reason women report postcoital bleeding. The vast majority of polyps in reproductive-age women are benign and do not require removal unless they are a cause of bleeding, though their presence is noted in the woman's record.[55] Malignancy is more likely among perimenopausal and postmenopausal women.

Cervical Lacerations

Parous women may have sustained a cervical laceration during a birth that was not repaired. If the laceration is extensive, it can be a risk for cervical incompetence in a subsequent pregnancy. Cervical lacerations should be noted in the woman's chart and consultation obtained if they are noted during pregnancy or in a woman planning a pregnancy.

Cervicitis

An inflamed cervix typically is accompanied by discharge or bleeding, which may result from either an infectious or noninfectious cause. Presence of discharge that is mucopurulent and associated with bleeding and edema of the cervix requires further evaluation for infection such as gonorrhea. Friability and erosion of the cervix suggest malignancy.

Condylomata Acuminata

Condylomata acuminata (condylomas) are the manifestation of genital infection with HPV. When found on the cervix, condylomas typically appear as bright white growths with a warty or cauliflower-like appearance. They may be seen in the vagina and on the vulva, perineum, or anus as external genital warts. The appearance of condylomas on the cervix will most likely be

accompanied by an abnormal Pap test result. When administered prior to any HPV exposure, the current HPV vaccine will prevent at least 90% of cases of external genital warts. The *Health Promotion Across the Lifespan* chapter provides additional information regarding vaccines, and additional discussion of condylomas can be found in the *Reproductive Tract and Sexually Transmitted Infections* chapter.

Leukoplakia

Leukoplakia is a nonspecific term referring to an area on the cervix appearing as a white, adherent patch of tissue. A midwife can use a cotton-tip applicator to verify if the patch is adherent, which is characteristic of leukoplakia or cervical intraepithelial neoplasm, or not adherent, which is characteristic of candidiasis. Leukoplakia is generally the result of chronic irritation from, for example, the presence of an IUD string. The white color denotes keratin formation over the surface of squamous epithelium. Because it is not possible to distinguish leukoplakia from CIN with the naked eye, further evaluation such as a Pap test always is indicated.

Disorders of the Vulva

Conditions that affect the vulva include cysts, dermatoses, vulvodynia, and other infections.

Bartholin Gland Cysts

Bartholin glands, also called the greater vestibular glands, are two mucus-secreting, nonpalpable, structures located within the vaginal vestibule in the space between the hymenal ring and the labia minora at approximately 4 o'clock and 8 o'clock (**Figure 12-5**). Bartholin glands can develop cysts or abscesses.

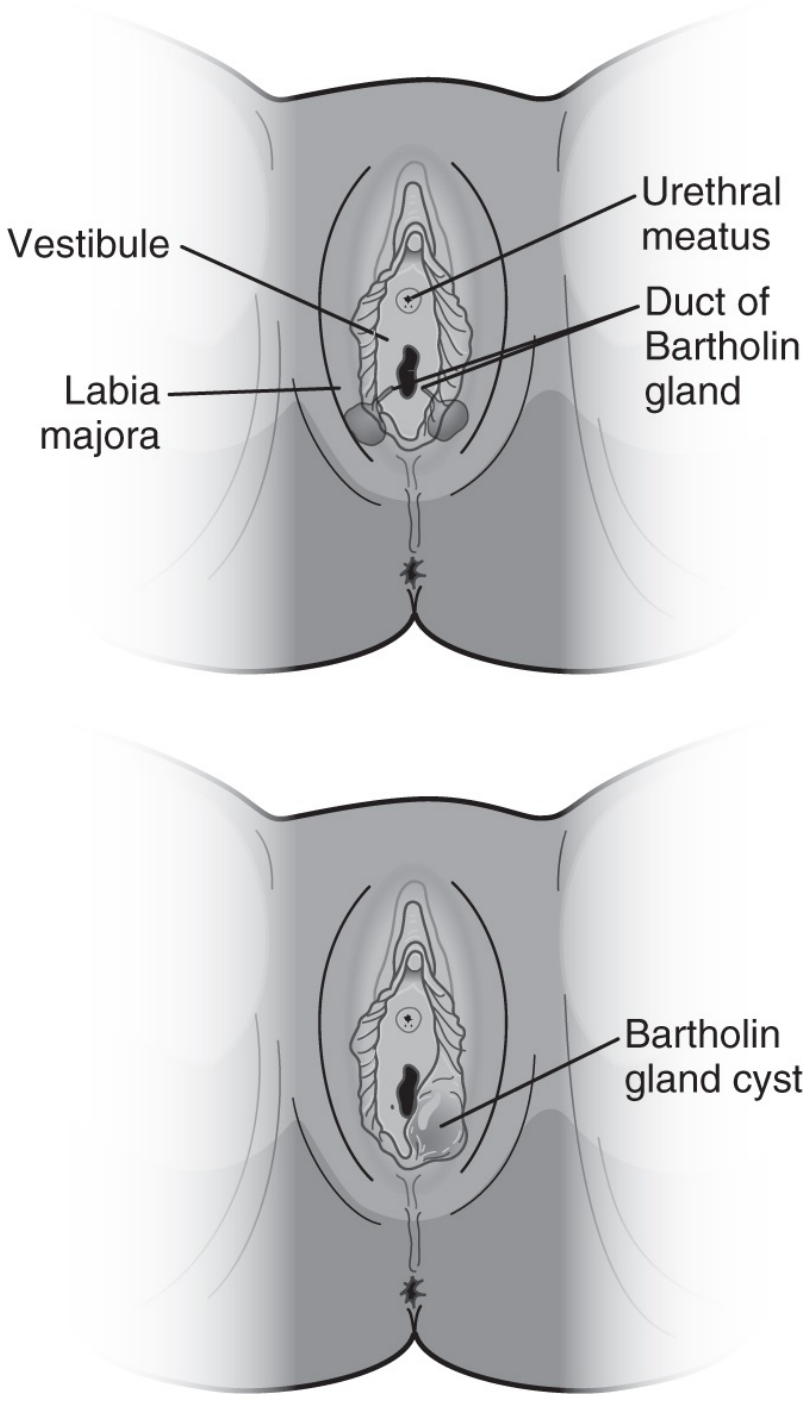


Figure 12-5 Location of Bartholin glands with Bartholin cyst on left side.

Bartholin glands normally secrete a small amount of lubricant. If the gland openings become obstructed, a cyst will develop, resulting in a mass of variable size. Most Bartholin cysts are asymptomatic and do not require treatment. However, if the fluid becomes infected, abscess formation can develop.

Infected Bartholin cysts are usually erythematous and painful. Past literature identified *Neisseria gonorrhoeae* and other STIs as the primary cause of Bartholin abscesses. However, more recent literature reports that the majority of abscesses contain multiple types of bacteria, some of which are part of the normal vaginal flora, including *Escherichia coli*.[56] Methicillin-resistant *Staphylococcus aureus* (MRSA) has been found as the predominant organism in Bartholin abscess in some studies but not all. In general, cysts that exhibit induration, but no evidence of abscess, are treated with a course of antibiotics and warm compresses. Women who are pregnant or otherwise immunocompromised require close follow-up. While the procedure for incision and drainage with placement of a Word catheter is beyond the scope of this text, midwives may add this procedure to their practices following completion of the process described in the *Standards for Practice of Midwifery*.

In 2014, the Infectious Diseases Society of America updated its guidelines for the treatment of skin and soft-tissue infections, classifying Bartholin gland cysts into three categories—mild, moderate, and severe.[57] Mild infections demonstrate only a local, mildly purulent lesion, while moderate infections involve systemic symptoms. A severe infection is one that has failed incision and drainage, has not responded to antibiotic therapy, and shows more significant systemic involvement.[58] Women with recalcitrant or recurrent infections, as well as those requiring surgical intervention, are usually best referred to a specialist.[59]

Human Papillomavirus–Associated Lesions

In addition to causing condylomata on the cervix, infection with HPV can cause condylomata acuminata on the vulva, vagina, perineum, and anus. External genital warts are commonly caused by low-risk HPV types 6 and 11, and are not associated with cancer, but they often cause physical symptoms, carry a social stigma, and lead to multiple costly visits to healthcare providers. The care and treatment of vulvar condylomata acuminata are reviewed in more detail in the *Reproductive Tract and Sexually Transmitted Infections* chapter.

Herpes Simplex Lesions

Within the herpes simplex virus (HSV) family of DNA viruses, eight are associated with human infection. Notably, HSV-1 and HSV-2 can cause both oral and genital infections.

Genital herpes is a chronic, recurrent condition of skin and mucosal surfaces caused by infection with HSV, and is transmitted by sexual contact. The initial infection with HSV typically manifests as painful blisters on the vulva, perineum, vagina, cervix, or anus, although the majority of individuals who carry and transmit genital herpes have never had an outbreak of painful blisters, are asymptomatic, and have not been diagnosed. Although both HSV-1 and HSV-2 can cause genital infection, their clinical course is generally different: Specifically,

HSV-2 causes more genital pain, and outbreaks are typically more frequent. Serologic testing capable of distinguishing HSV-1 versus HSV-2 (or the presence of both) is available. Diagnosis, counseling, and treatment of genital herpes, including its potential effect during the perinatal period, is addressed in the *Reproductive Tract and Sexually Transmitted Infections* chapter.

Dermatologic Disorders

The vulva can develop a variety of benign and cancerous dermatologic conditions. Skin disorders of the vulva may be either chronic or acute. Vulvar disorders can also be a source of sexual pain and dysfunction. Most women with skin disorders of the vulva have itching, pain, or irritation that can be a cause of significant personal distress. Care of women with chronic vulvar conditions can be challenging relative to determining the correct diagnosis, and treatments may or may not be effective. When caring for women with vulvar skin disorders, thorough knowledge of vulvar anatomy and function will guide the evaluation. Skin conditions common in other body sites, such as eczema, psoriasis, and contact dermatitis, frequently appear on the vulva. Vulvar biopsies are the gold standard to differentiate among the various vulvar conditions. Midwives trained in this procedure may incorporate vulvar biopsies in gynecologic practice depending on the practice setting and guidelines.

Folliculitis

Folliculitis of the vulva is a common condition involving hair follicles and sebaceous glands of the vulva. It occurs in hair-bearing areas of the vulva. The typical appearance is that of yellow-white- or red-colored, pinhead-sized pustules with a central hair. Most folliculitis is noninfectious, but secondary skin infections can occur mostly from surface skin bacteria and trauma, such as from shaving, waxing, or use of depilatories. Folliculitis may extend to the buttocks and upper, inner thighs. Treatment consists of avoidance of trauma and treatment with antibiotic creams or ointments.

Hidradenitis Suppurativa

Hidradenitis suppurativa is a chronic skin disease involving a recurrent, inflammatory response in areas where apocrine (sweat) glands reside.[60] Women may also report problems with frequent or recurring boils on their skin. Without a high index of suspicion, an accurate diagnosis may be inadvertently delayed, thereby allowing for progression of disease.

Hidradenitis suppurativa may first appear in adolescence, beginning as painful, nodular lesions or abscesses in the axillary or genital region. When lesions drain, sinus tracts often form, leading to scarring, pain, and deformity in severe cases. In women, the areas most commonly affected by hidradenitis suppurativa are the groin, perineum, and axilla.

Hidradenitis suppurativa is estimated to affect 1% to 4% of the population. While men may also be affected, cases among women outnumber those among men by 3 to 1.[61] The etiology of this disease has not been established, but studies suggest it is a multifocal condition—possibly an inflammatory or immune system disorder involving atrophy of sebaceous or apocrine

glands, which secrete sebum into hair follicles, and produce inflammation leading to hair follicle destruction.[62,63] Smoking and obesity have been associated with severe disease.[63]

No diagnostic testing exists for hidradenitis suppurativa. Depending on the clinical diagnosis and extent of disease, treatment may be topical or systemic. In extreme cases, immunosuppressive therapy, surgical treatments, or laser treatments are recommended. Well-controlled clinical trials have not been published comparing modalities to determine which treatments are most effective, and referral to a specialist is usually the best action.

Lichen Sclerosus

Lichen sclerosus is a common, lifelong, benign condition that can occur at any age, but has two peaks of onset: in prepuberty and during perimenopause and postmenopause.[64] While the cause of lichen sclerosus remains unclear, other conditions with an autoimmune component, such as thyroid disorders, systemic lupus erythematosus, and alopecia, are found among women with lichen sclerosus. Women with lichen sclerosus have itching and pain and may present with dyspareunia. On examination, there may be evidence of scratching and, in severe cases, changes in the appearance of the vulvar skin and loss of architecture. The skin becomes thin with white patches. Once confirmed by biopsy, treatment includes topical steroids and surveillance for signs of vulvar cancer. Referral to an expert in management of vulvar disorders is essential when diagnosis is uncertain or there is limited improvement in the woman's symptoms.

Lichen Planus

Lichen planus is an autoimmune disorder that affects the skin, mucocutaneous sites such as the mouth, the scalp, and the genitalia, most often the vulva or vagina. The etiology is unknown but the disorder is thought to occur secondary to a T-cell–mediated autoimmune response to basal keratinocytes. Like lichen sclerosus, lichen planus primarily affects perimenopausal and postmenopausal women, and is associated with other autoimmune disorders. Symptoms are similar to those of lichen sclerosus, including vulvar soreness, burning, and pruritus.[64] Dyspareunia is common. Vulvar tissue may be more reddened with an erosive appearance and extremely tender to touch.[64] Architectural changes involving the vulva and vagina may be present.

Women may try a number of self-treatments prior to seeking professional care. Once biopsy confirms lichen planus, referral to a provider who specializes in the management of vulvar disorders is advisable, as lichen planus can be refractory to treatment. Lichen planus is associated with a small increase in the woman's risk of vulvar cancer.[64]

Lichen Simplex Chronicus

Lichen simplex chronicus is another vulvar skin condition that can involve the entire perianal region. The most common symptom associated with lichen simplex chronicus is severe itching, leading to a cycle of itch–scratch–itch, with the itching often being severe enough to interrupt

sleep.[64] A thickened and leathery-like skin texture results after long-term scratching, which ultimately damages the protective skin barrier, exposing the woman to secondary infection. Diagnosis is generally based on clinical appearance, as biopsy may be nonspecific. Treatment is directed at reducing the itch–scratch–itch cycle with topical steroids, restoring the normal protective skin barrier, and reducing or managing recurrences.[64]

Complications of Genital Piercing

Though not a disorder, the practice of genital piercing has increased dramatically among all segments of the population. Women may choose genital piercing as a means of personal and sexual expression, and in some cases, it is perceived by women as a form of psychological healing following a traumatic event such as abuse or sexual assault.[65,66] When problems arise as a consequence of piercing, women may initially seek answers from the Internet rather than from their healthcare provider.[67]

Although genital piercing is usually safe when performed by a licensed provider whose practice is regulated by state laws, no national safety guidelines exist. Unless the individual performing the procedure is a licensed health professional, no anesthesia is used when performing piercings.

Health risks of genital piercings include local and systemic infection, scarring, infertility due to infection complications, and urethral scar formation.[65,66] Women considering pregnancy or who are pregnant are recommended to defer genital piercing to avoid risk of blood-borne complications such as hepatitis.[65] The Association of Professional Piercers (listed in the Resources section at the end of this chapter) publishes a quarterly journal on piercing techniques, safety, anthropology, and legislative issues relating to body piercing.

Vulvodynia

Vulvodynia is a chronic genital pain syndrome that is estimated to affect as many as 28% of reproductive-age women during their lifetimes.[58] In 2015, the International Society for the Study of Vulvovaginal Disease (ISSVD), the International Society for the Study of Women's Sexual Health (ISSWSH), and the International Pelvic Pain Society (IPPS) agreed to reexamine and revise terminology related to vulvar pain which now subdivides vulvar pain into one of two categories: vulvar pain that is associated with a specific disorder or vulvodynia which refers to persistent vulvar pain without an identified etiology. The updated terminology appears in **Table 12-8**. The intent of the new terminology was to reflect the complexity of the disease, to distinguish vulvodynia from other pain or infectious syndromes with *known* causes, and to better guide diagnosis and treatment.[68] The current definition of vulvodynia describes vulvar pain that has lasted at least 3 months and has no identifiable etiology.[68]

Table 12-8	2015 Consensus Terminology and Classification of Persistent Vulvar Pain
Vulvar Pain Caused by a Specific Etiology[a]	

Infectious (e.g., recurrent candidiasis, herpes)
Inflammatory (e.g., lichen sclerosus, lichen planus, immunobullous disorders)
Neoplastic (e.g., Paget disease, squamous cell carcinoma)
Neurologic (e.g., postherpetic neuralgia, nerve compression or injury, neuroma)
Trauma (e.g., female genital cutting, obstetric)
Iatrogenic (e.g., postoperative, chemotherapy, radiation)
Hormonal deficiencies (e.g., genitourinary syndrome of menopause [vulvovaginal atrophy], lactational amenorrhea)
Subcategories of Vulvodynia[b]
Localization: localized (e.g., vestibulodynia), generalized, or mixed
Provocation: provoked (e.g., insertional contact), spontaneous or mixed
Onset: Primary or secondary (e.g., symptoms that develop after a period of normal function)
Temporal pattern: intermittent, persistent, constant, immediate, delayed

[a] Women may have both another specific disorder (e.g., lichen sclerosus) and vulvodynia.

[b] Vulvar pain of at least 3 months' duration, without clear identifiable cause, which may have potential associated factors and following descriptors.

Reproduced with permission from Bornstein J, Goldstein AT, Stockdale CK, et al., on behalf of Consensus Vulvar Pain Terminology Committee of the International Society for the Study of Vulvovaginal Disease (ISSVD), International Society for the Study of Women's Sexual Health (ISSWSH), and International Pelvic Pain Society (IPPS). 2015 ISSVD, ISSWSH and IPPS consensus terminology and classification of persistent vulvar pain and vulvodynia. *Obstet Gynecol.* 2016;127:745-751.[68]

The cause of vulvodynia is believed to be multifactorial, and several elements—including pathophysiologic factors, psychological factors, and social and relational factors[69]—may influence either the development or subsequent consequences of this disorder. The presentation of vulvodynia includes symptoms that are either chronic and unrelenting or cyclic in nature which makes the diagnosis of vulvodynia difficult and generally one of exclusion. Various algorithms have been published to aid the clinician in ruling out other potential conditions; **Figure 12-6** shows one example.[69]

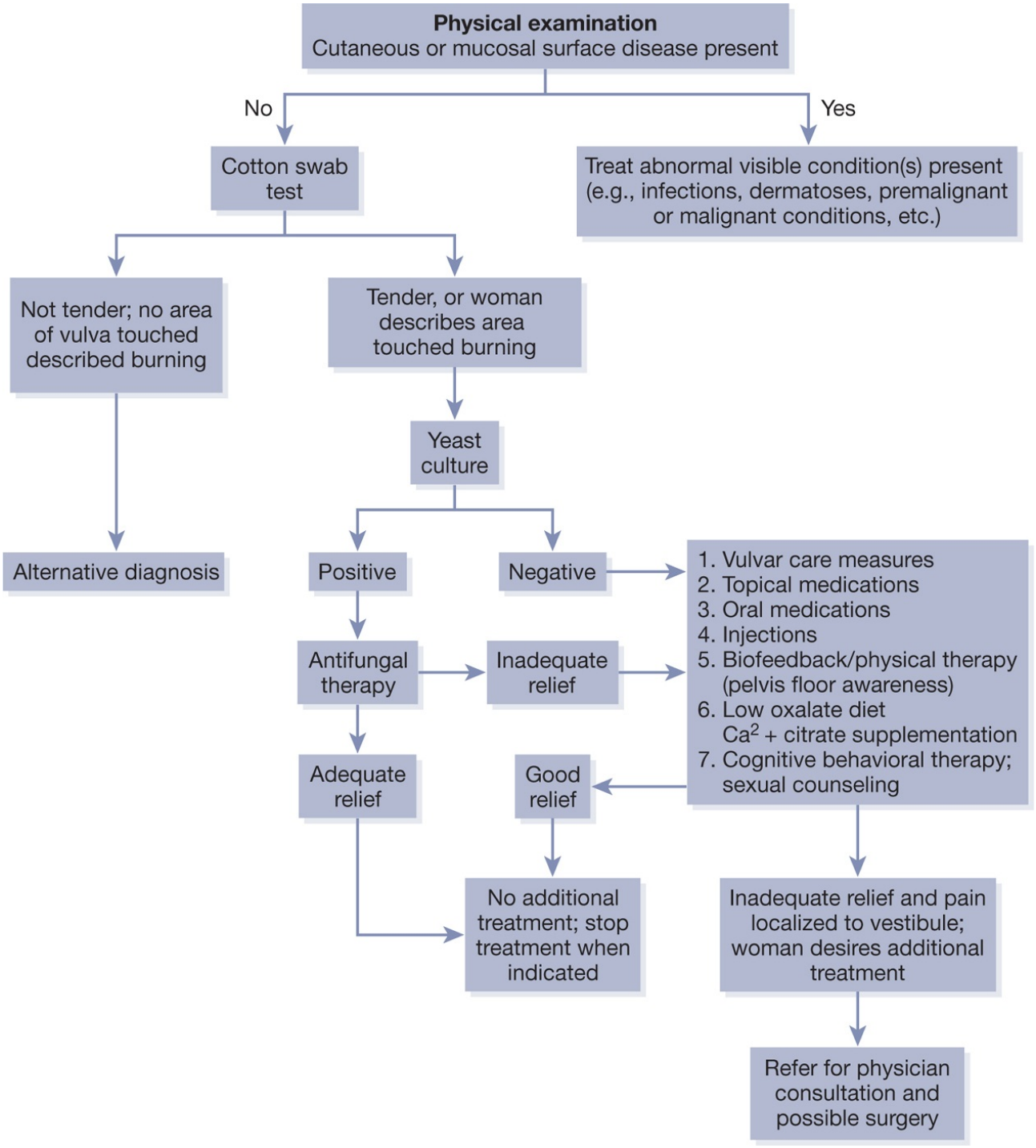


Figure 12-6 Algorithm for the diagnosis and management of vulvodynia.

Modified with permission from Haefner HK, Collins ME, Davis GD, et al. The vulvodynia guideline. *J* **Low Genit Tract Dis**. 2005;9:40-51.[69]

Evaluation of Vulvodynia

The evaluation for vulvodynia starts with a comprehensive history of the onset and duration of symptoms using the OLD CARTS mnemonic (see **Table 12-5**). Other comorbid symptoms, such as interstitial cystitis, fibromyalgia, or irritable bowel, are frequently found among women

with vulvodynia.[70] Physical and sexual abuse in childhood have been identified as possible risk factors for development of vulvodynia.[70]

Examination of the vulva includes evaluation of skin changes, vaginal secretions, presence of lesions, and changes in the vulvar architecture. It is useful to perform an evaluation of point-specific pain and sensitivity using a cotton swab and gently proceeding in a clockwise fashion on the vulva. Particular areas of sensitivity may be noted, especially at the 11 to 1 o'clock points and the 5 to 7 o'clock points (**Figure 12-7**).[71,72]

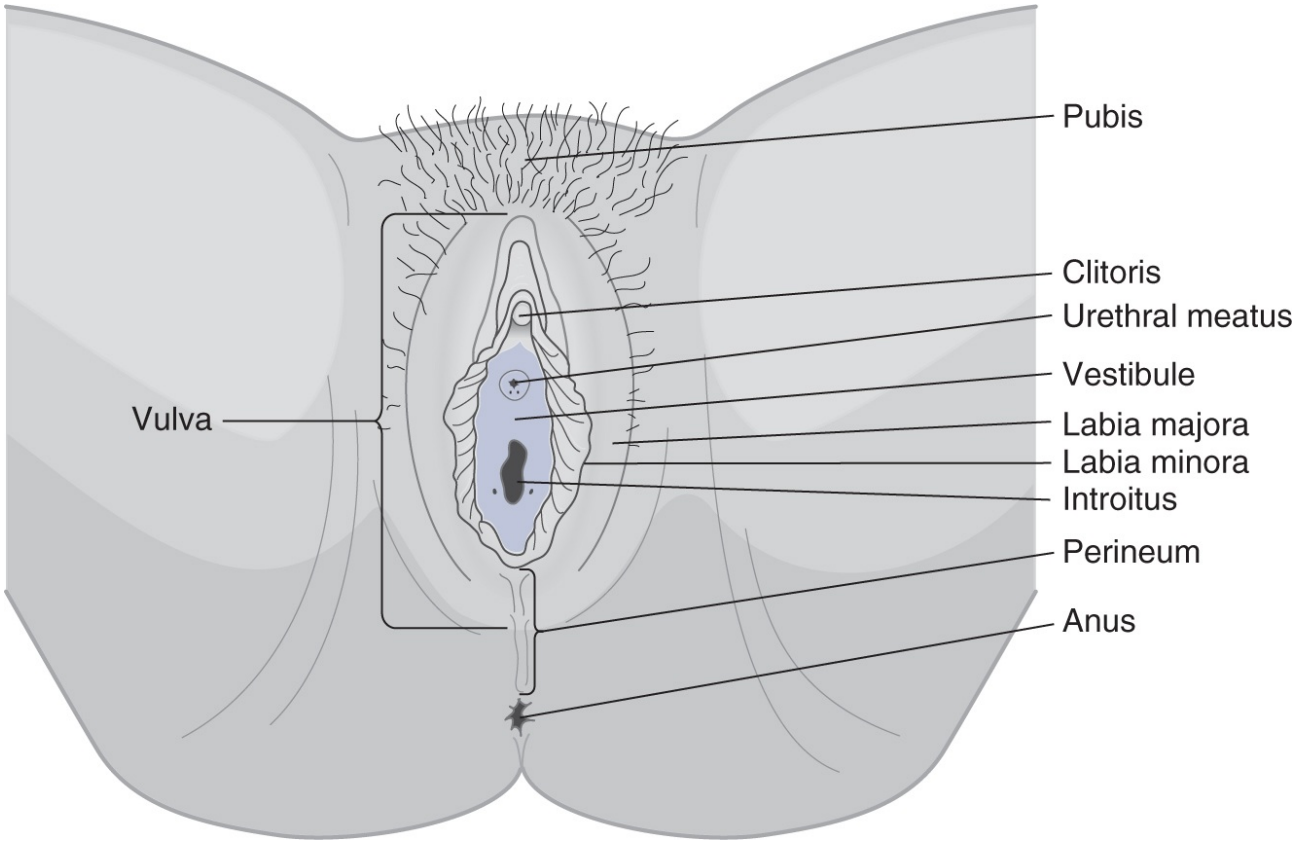


Figure 12-7 Cotton swab test for specific location of vulvodynia.

Treatment of Vulvodynia

Treatments for vulvodynia are wide-ranging in scope, are highly individualized, and usually begin with avoidance of potential irritants. A woman experiencing vulvar pain may have tried numerous over-the-counter treatments, including antifungal and topical steroid preparations. These treatments in themselves may have caused a dermatitis that must be minimized before further evaluation can occur.[71,73] Use of unscented toilet tissue, loose cotton clothing, unscented soaps, and showers instead of baths is recommended.[74] Avoidance of feminine hygiene products, fragranced baths, baby/personal wipes, over-the-counter anti-itch products, detergent additives, fabric softeners, dryer sheets, and latex products has been found to reduce vulvar pain for some women.[71,73,74] Following these initial steps, recommendations for treatment may include psychological interventions, physical therapy, and pelvic floor physical therapy.[74] Antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs) or serotonin–

norepinephrine reuptake inhibitors (SNRIs), also have been used and appear to have some value for relief of pelvic neuropathies.[71,73,74] A variety of alternative or complementary therapies have shown varying degrees of success, including biofeedback, pelvic floor exercises, cognitive group therapy, cognitive-behavioral therapy (CBT), acupuncture, transcutaneous nerve stimulation, and hypnotherapy, although none of these is considered a first-line treatment.[71-74]

When most or all of the aforementioned modalities are unsuccessful, clinicians may recommend surgery, including excision of nerve pathways or nerve injuries associated with specific locations of pain.[75] A pelvic examination presents the perfect opportunity to educate women about care and hygiene practices that protect the vulva, as outlined in **Table 12-9**.[74]

Table 12-9	**Measures to Minimize Vulvar Irritation**
Wear 100% cotton underwear (no underwear at night)	
Avoid vulvar irritants (perfumes, dyes, shampoos, detergents) and douching	
Use mild soaps for bathing, avoiding applying the soap to the vulva	
Clean the vulva only with warm (not hot) water	
Avoid the use of hair dryers on the vulvar area	
Pat the area dry after bathing, and apply a preservative-free emollient (such as vegetable oil or plain petrolatum) topically to maintain moisture in the skin and improve the barrier function	
Switch to 100% cotton menstrual pads as needed (if regular pads are irritating)	
Use adequate lubrication for intercourse	
Consider cool gel packs to the vulvar area	
Rinse and pat the vulva dry after urination	

Modified with permission from American College of Obstetricians and Gynecologists. Committee Opinion No. 673: persistent vulvar pain. *Obstet Gynecol.* 2016;128:e78-e84.[74]

Pelvic Floor Dysfunction Disorders

The two most common findings related to pelvic floor changes are pelvic organ prolapse and urinary incontinence.[76] Both of these conditions can occur secondary to a number of structural abnormalities, physical trauma, or pathophysiologic conditions. Risk factors for both conditions include pregnancy (regardless of the mode of birth), older age, increasing parity, family history, obesity, smoking, constipation, a history of heavy lifting, prior lower abdominal or pelvic surgeries, a lower abdominal or pelvic mass, a history of chronic lung disease, and a history of a connective tissue disorder (e.g., Marfan syndrome).[77-80]

Pelvic Organ Prolapse

Pelvic organ prolapse refers to herniation of pelvic organs to or beyond the vaginal walls. Common forms of pelvic organ prolapse include *cystocele* or *urethrocele* (bladder or urethral descent into the vagina), *rectocele* or *enterocele* (protrusion of the rectum into the vagina), and *uterine prolapse* (descent of the uterus into the vagina); see **Table 12-10**. Each of these conditions, or a combination of them, can cause abnormal bladder or rectal function, pelvic or lower back pain, and/or decreased pleasure with sexual activity.[81]

Table 12-10 Pelvic Organ Prolapse Conditions

Term	Description and Findings
Urethrocele	Prolapse of the urethra, evident as descent of the anterior vaginal wall
Uterine/cervical prolapse	Prolapse of the uterus, evident as descent of the uterus or uterine cervix into the vagina
Vaginal vault (cuff scar) prolapse	Descent of the vaginal vault or cuff scar into the vagina after hysterectomy
Cystocele	Prolapse of the bladder, evident as descent of the anterior vaginal wall
Rectocele	Prolapse of the rectum, evident as descent of the posterior vaginal wall
Enterocele	Hernia of peritoneal sac and small intestine into the rectovaginal space between the vagina and rectum

Although several instruments have been developed to describe the nature and extent of pelvic organ prolapse, the most widely supported is the Pelvic Organ Prolapse Quantification (POP-Q) system, which is endorsed by professional organizations.[82] In using the POP-Q system, the hymen acts as a fixed point of reference and the prolapse is measured with regard to six anatomic locations. The results define one of four stages of prolapse as described in **Table 12-11**.

Table 12-11 Pelvic Organ Prolapse Quantification System (POP-Q)

	Description
Point	

Aa	Anterior vaginal wall 3 cm proximal to the hymen −3 cm to +3 cm
Ba	Most distal portion of the remaining upper anterior vaginal wall −3 cm to +3 cm
C	Most distal edge of the cervix or vaginal cuff scar
D	Posterior fornix (not applicable if posthysterectomy)
Ap	Posterior vaginal wall 3 cm proximal to the hymen −3 cm to +3 cm
Bp	Most distal position of the remaining upper posterior vaginal wall −3 cm to +3 cm
Point POP-Q Staging Criteria	
Stage 0	Aa, Ap, Ba, Bp = −3 cm and C or D ≤ − (total vaginal length − 2) cm
Stage I	Most distal portion of the prolapse −1 cm (above the level of hymen)
Stage II	Most distal portion of the prolapse ≥ −1 cm but ≤ +1 cm (≤ 1 cm above or below the hymen)
Stage III	Most distal portion of the prolapse > +1 cm but < + (total vaginal length − 2) cm (beyond the hymen; protrudes no farther than 2 cm less than the total vaginal length)
Stage IV	Complete eversion; most distal portion of the prolapse ≥ + (total vaginal length − 2) cm

Based on Persu C, Chapple C, Cauni V, Gutue S, Geavlete P. Pelvic organ prolapse quantification system (POP–Q): a new era in pelvic prolapse staging. *J Med Life.* 2011;4:75-81.[82]

Evaluation of Pelvic Floor Disorders

Evaluation of pelvic floor disorders includes obtaining a comprehensive history and visualizing the woman's external genitalia, vagina, and cervix for signs of urinary leakage or organ displacement/prolapse. Asking the woman to perform a Valsalva maneuver or cough allows the midwife to visually assess the level of vaginal wall support. Uterine prolapse may also be observed with the woman in an upright position. In severe cases, the cervix may protrude through the vulva simply by the pull of gravity. A follow-up bimanual examination during which the woman tightens vaginal muscles around the clinician's fingers allows the midwife to gauge the strength of the pelvic muscles, and presents an opportunity to teach the correct method for performing muscle tightening exercises. Two short survey instruments to help clinicians determine the nature and extent of prolapse—the Pelvic Floor Impact Questionnaire and the Pelvic Floor Distress Inventory—are also widely available.

Treatments for Pelvic Floor Disorders

In the absence of pelvic masses, treatment for pelvic floor relaxation may include the initiation of pelvic floor muscle training. While some evidence indicates that pelvic floor muscle training alone can improve pelvic organ prolapse, women often require specific instruction and feedback during a vaginal examination to learn how to perform pelvic floor muscle training correctly, and referral to a physiotherapist specializing in such training may be warranted.[83,84]

A variety of support devices or pessaries can be employed in the case of significant uterine prolapse.[85] In general, there are two types of pessaries (described in **Appendix 12A**) that are used: those that offer support (e.g., ring pessary) and those that fill space in the vagina (e.g., cube pessary). The former are suited for women at POP-Q Stage 1 or 2, while the latter are

designed for women at Stage 3 or 4. After the initial examination and placement of the pessary, the woman returns for a follow-up visit in 1 to 2 weeks to assess comfort and normal urinary function. She then returns again in 3 to 6 months and at least semi-annually thereafter to assess proper use and effectiveness of the pessary. The midwife should teach the woman to remove and clean the pessary weekly with soap and warm water. Women unable to perform self-care should be seen as needed to remove and clean the pessary and ensure proper fitting.[86] Vaginal evaluation includes visual inspection to assess for lesions or bleeding due to tissue erosion caused by pessary use. A woman with a pessary in place should be cautioned to report vaginal bleeding promptly, as this may be a sign of tissue damage or intrauterine bleeding indicative of the growth of a malignancy.

Several surgical procedures have been introduced in an attempt to repair or restore the lost muscle tone associated with pelvic organ prolapse. Despite these efforts, the long-term success rates of different surgical approaches are variable and can result in complications, such as urinary incontinence.[87]

Urinary Incontinence

Urinary incontinence is underreported for many reasons, including personal embarrassment, limited access to health care, and lack of proper screening by clinicians. Underreporting has made it difficult to identify how many women experience urinary incontinence, but reviews of the prevalence, identification, and treatment of urinary incontinence suggest that between 25% and 55% of adult females experience urinary incontinence at some point in their lives. The occurrence can be as high as 75% in older age groups.[88]

Urinary incontinence is often associated with significant diminishment of women's quality of life. Effects of urinary incontinence can include impaired sleep, limitations on the ability to travel far from home or to engage in social activities, and difficulty in maintaining good hygiene to avoid skin irritation or breakdown. The role of the midwife in assessing and treating urinary incontinence is most often accomplished in consultation with colleagues who specialize in urogynecologic care.

Urinary incontinence is classified based on the presenting symptoms and signs, as summarized in **Table 12-12**. The most common types of urinary incontinence are stress, urgency, and overflow incontinence. *Stress urinary incontinence*, which involves involuntary leakage of urine, may co-occur with detrusor overactivity (overactive bladder), defined as involuntary bladder contractions during the filling phase of the bladder. *Urgency urinary incontinence* is less common than stress urinary incontinence; it causes the woman to experience a sudden urge to urinate and subsequent involuntary loss of urine if she is unable to empty her bladder almost immediately. This form of urinary incontinence can be due to a variety of neuropathies. *Overflow urinary incontinence* presents with continuous urine leakage and incomplete bladder emptying. A woman with overflow urinary incontinence may also have frequency, nocturia, hesitancy, and/or a weak intermittent urinary stream. If the bladder is very full, stress leakage may cause symptoms that are similar to those of stress or urgency incontinence. The combination of stress and urgency urinary incontinence is termed *mixed urinary incontinence*.[88,89]

Table 12-12	Types of Urinary Incontinence
Type of Incontinence	**Definition and Description**
Stress urinary incontinence	Involuntary leakage caused by increases in abdominal pressure, effort, or exertion (e.g., sneezing or coughing) but no urge to urinate prior to leakage. Caused by urethral hypermobility, which causes the bladder neck and urethra to lose the ability to close. Can occur secondary to insufficient support from pelvic floor muscles, trauma from childbirth, or chronic pressure from obesity or chronic cough.
Urgency urinary incontinence	Urge to urinate is sudden, with involuntary leakage and frequent small-volume voids. The result of detrusor overactivity. Can occur secondary to bladder abnormalities or neurologic disorders.
Neurogenic detrusor overactivity (overactive bladder)	Involuntary bladder contractions during the filling phase of the bladder, due to a defined neurologic condition. A form of urgency urinary incontinence.
Mixed urinary incontinence	Involuntary leakage associated with both urgency and stress incontinence.
Overflow incontinence	Frequent or constant dribbling or stress incontinence. Typically caused by an underactive detrusor muscle or outlet obstruction. Can occur secondary to detrusor underactivity, which may stem from a low estrogen state, peripheral neuropathy (e.g., diabetes, alcoholism), or spinal cord damage (e.g., muscular sclerosis). Urinary retention can also occur secondary to drug therapy.
Nongenitourinary incontinence	Variable leaking of urine caused by functional problems (e.g., neurologic, psychologic, cognitive), pharmacologic agents, environmental problems, or metabolic disorders.

The need to void once or twice during the night is not necessarily indicative of urinary dysfunction, and an occasional episode of leaking urine does not meet the criteria for incontinence treatment. If the urinary incontinence is a side effect of a pharmaceutical agent, change in mental status, mobility, or new onset of constipation, the condition may be temporary. Increasing age, obesity, smoking, pregnancy, and increased abdominal pressure due to constipation and occupational lifting also have been shown to contribute to urinary incontinence.

Certain disease states are associated with increased urination and incontinence, including diabetes, urinary tract infection or other renal disease, bladder mass, and pelvic masses such as fibroids or ovarian cancer. Surgery or pelvic trauma also can result in involuntary loss of urine. Women who have a vaginal birth do not appear to have a higher risk of urinary incontinence when compared with those having a cesarean birth. A genetic disposition may increase the risk of urinary incontinence even if the woman has never been pregnant.

Evaluation of Urinary Incontinence

In the absence of a urinary tract infection, a pelvic mass, pelvic organ prolapse, or some other extrinsic cause of incontinence, the diagnosis of incontinence is made using a combination of the woman's history and a variety of urinary function tests.

Several clinical assessment instruments are available to aid making a diagnosis of urinary

incontinence, including the shortened versions of the Pelvic Floor Impact Questionnaire and the Pelvic Floor Distress Inventory used to assess pelvic organ prolapse, as well as the often-used Questionnaire for Urinary Incontinence Diagnosis (QUID).[90,91] Women can offer a significant amount of diagnostic information by completing a daily urinary diary that includes the time and amount of oral intake, urine output, any urine leakage, and necessary pad changes. These validated questionnaires are often used to help establish a diagnosis and can be readily obtained from the sites listed in the Resources section at the end of this chapter.[90,91]

Urinary function tests can range from the simple, such as the cough stress test, which can be performed in the office by asking the woman to cough with a full bladder and subsequently observing for leaking from the urethra; to the complex, such as urodynamic testing, which involves the use of equipment that can evaluate urethral function, bladder capacity, bladder stability, and the woman's ability to control voiding. The latter form of assessment can be expensive and is usually reserved as a second line of testing, typically performed by a urologist or urogynecologist.

Treatment of Urinary Incontinence

For women with stress urinary incontinence, initial treatment includes pelvic floor muscle exercises, behavioral modifications, and dietary changes. Weight loss may improve symptoms of stress urinary incontinence.[92] Some clinicians advocate use of Knack exercises, which are similar to Kegel exercises but focus more on strengthening urethral muscles. Knack exercises are a form of Kegel exercise that involves contracting the pelvic muscles as deep into the vagina as possible and holding the contraction while doing other activities during which the woman usually experiences incontinence, such as coughing or exercising in general.[93]

The successful use of pelvic floor muscle training can be enhanced by using provider *feedback* (i.e., giving the woman instructions while performing a bimanual examination) or *biofeedback* (i.e., using a vaginal insertion device that offers feedback on the success of a woman's attempt with the exercises). Similarly, the promotion of healthy dietary changes, especially in women with obesity, may help to reduce incontinence related to obesity.

Pharmaceutical treatments for stress urinary incontinence are generally considered ineffective, and there are no FDA-approved drugs for treatment of stress urinary incontinence.[88] If a woman is diagnosed with stress urinary incontinence combined with urgency or detrusor overactivity (overactive bladder), bladder training is recommended. This training involves either encouraging the woman to delay any initial urges to void by using the Knack technique and simultaneously distracting herself with mental exercises, or teaching her to empty her bladder before an urge occurs.

Two classes of drugs are used to treat urgency urinary incontinence: beta-adrenergic drugs and antimuscarinic agents. Medications approved by the FDA to treat detrusor overactivity are listed in **Table 12-13**.[94-96] Several antimuscarinic agents are available, and they are generally the first-line agents even though they have only modest effectiveness.[95] These drugs increase bladder capacity by blocking muscarinic receptor stimulation by acetylcholine in the bladder during filling. Unfortunately, all these drugs have peripheral anticholinergic effects that limit women's tolerance of them.

Table 12-13 **U.S. Food and Drug Administration–Approved Medications for Treatment of Overactive Bladder**

Medication: Generic (Brand)	Action	Dose	Clinical Considerations
Beta-Agonist Drugs			
Mirabegron (Myribetriq)	Beta-agonist that causes muscle relaxation and increased bladder capacity	25–50 mg per day	Contraindicated for persons with uncontrolled hypertension, liver impairment, or renal disease.
Antimuscarinic Drugs[a]			
Darifenacin (Enablex ER)[a]	Antimuscarinic	7.5–15 mg daily	Metabolized in the liver. Advantages and disadvantages of M3 selectivity are unknown due to the lack of head-to-head trials. Can be taken with food. Half-life is 12 hours.
Oxybutynin (Ditropan IR)[a]	Antimuscarinic Mild antispasmodic Peak action: 1 hour Duration: 6–10 hours	2.5 mg or 5 mg daily, up to 5 mg 4 times a day	Has an active metabolite. Gastrointestinal absorption/liver metabolism leads to increased potential for side effects and drug interactions. Available in liquid formula (5 mg/5 mL). Women with cardiac conditions should take oxybutynin with caution due to its tachycardic side effects. Crosses the blood–brain barrier. Excretion in breast milk is unstudied; lactation suppression has been reported. Use with osteoporosis medications such as alendronate (Fosamax) with caution.

Fesoterodine ER[a] (Toviaz)	Targets M1–M3	4–8 mg once daily	Gastrointestinal absorption/liver metabolism leads to increased potential for side effects and drug interactions. Contraindicated for persons with liver impairment.
Oxybutynin (Ditropan) XL[a]	Antimuscarinic Peak action: 4–6 hours Duration: > 24 hours	5, 10, and 15 mg daily, up to 30 mg daily	Start with 10 mg and titrate; can increase to 30 mg or decrease to 5 mg. Gastrointestinal absorption; similar efficacy and side-effect profile as the immediate-release version. Cannot be chewed or crushed due to the internal dispensing mechanism.
Oxybutynin transdermal (Oxytrol Patch)[a]	Antimuscarinic Steady dose	Change biweekly	Avoids the liver first-pass effect and active metabolite production, which decreases side effects. Discontinuation may occur due to skin site reaction.
Tolterodine IR (Detrol)[a]	Antimuscarinic Peak: 1–3 hours	2 mg twice a day (1 mg and 2 mg tablets)	Gastrointestinal absorption/liver metabolism. Less lipophilic, so it decreases CNS effects, though they have been reported. Pill form only. Start with 4 mg daily, decrease to 2 mg as needed.
Tolterodine (Detrol LA)[a]	Antimuscarinic Peak: 2–6 hours	2–44 mg daily (2 mg and 4 mg tablets)	Gastrointestinal absorption/liver metabolism. Less lipophilic, so it decreases CNS effects, though they have been reported. Pill form only. Start with 4 mg daily, decrease to 2 mg as needed.
Trospium (Sanctura IR)[a]	Antimuscarinic Peak: 4–6 hours	20 mg twice daily	Minimal liver metabolism, with decreased drug–drug interaction possible. Water-soluble, larger molecule allows for possibly decreased crossing of the lipid blood–brain barrier. Cognitive side-effect profile in elderly may be improved, but is not eliminated. Due to water solubility, take on an empty stomach. In elderly, start with one dose daily.
Solifenacin (VESIcare ER)[a]	Antimuscarinic	5–10 mg daily	Metabolized in the liver. Long half-life (approximately 50 hours); no head-to-head comparisons relating to effect. Can be taken with food.

Abbreviations: CNS, central nervous system; ER, extended release; IR, immediate release; LA, long acting; XL, extended release.

[a] All antimuscarinic drugs are contraindicated for persons with myasthenia gravis, gastric retention, and narrow angle-closure glaucoma. In addition, these drugs are associated with a risk for urinary retention, and have side effects of dry mouth, constipation, tachycardia, drowsiness, and decreased cognitive function.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 155: urinary incontinence in women. *Obstet Gynecol.* 2015;126:e66-e81[88]; Sanford M. Mirabegron: a review of its use in patients with overactive bladder syndrome. *Drugs.* 2013;73:1213-1225[94]; O'Dell KK, Labin LC. Common problems of urination in nonpregnant women: causes, current management and prevention strategies. *J Midwifery Womens Health.* 2006;51:159-173.[96]

Injections of botulinum toxin A (Botox A) into the bladder wall also are effective treatments for some women with detrusor overactivity or urgency urinary incontinence. This therapy appears to offer only short-term relief, rendering possible repeat injections necessary for continence to be maintained.[88]

Estrogen was once thought to be a promising treatment for women with urinary incontinence, especially after menopause. While some evidence suggests that topical or vaginal application of estrogen can improve incontinence, oral systemic use of this agent has been associated with worsening symptoms.[88]

Mechanical devices for control of urinary incontinence include vaginal cones and pessaries in various sizes and shapes, which have been used with some reports of effectiveness. Nevertheless, in population studies, their use has not been demonstrated to make a significant difference in bladder control,[97] and they appear to be most effective in conjunction with other treatments, such as pelvic floor muscle exercises and behavioral modifications.

When other treatments have been ineffective, the final approach to treatment of a woman with urinary incontinence is surgery. This approach is limited to stress urinary incontinence, however—it is not applicable to urgency urinary incontinence.[88] Women with stress urinary incontinence considering surgery should be informed of the multiple surgical procedures available and the related side effects associated with each respective approach, and referred to appropriate specialists.

Fertility/Infertility

Infertility is the inability to conceive during 1 year of unprotected, appropriately timed acts of penile–vaginal intercourse or donor insemination.[98] Infertility can be present in a male or female partner. In women, infertility is often associated with, or directly caused by, gynecologic conditions such as uterine anomalies, pelvic masses, or history of pelvic inflammatory disease. Categorizing infertility leads to one of two diagnoses: *primary infertility* for women with no prior pregnancy, and *secondary infertility* for women who have conceived at least once in the past.

While several of the gynecologic conditions discussed in this chapter can contribute to infertility (e.g., endometriosis, uterine fibroids), the cause is often unknown. One-third of the time, infertility is believed to result from conditions affecting the female, one-third of the time from conditions affecting the male, and one-third of the time from conditions affecting a combination of both the female and male. Thus, when discussing infertility with a woman, it is important to consider both partners.[99]

Known biological causes of female infertility include obstruction of the fallopian tubes, endometriosis, pelvic adhesions, hyperprolactinemia, endocrine disorders such as hypothyroidism, and advancing age. Any woman 35 years or older who has been trying to conceive should seek care after 6 months of attempting to become pregnant instead of waiting 1 year.[99] Causes of male infertility are usually secondary to sperm abnormalities, including those related to the quality of sperm and the number of sperm per ejaculation as well as genetic mutations that adversely affect sperm function. The prevalence of chromosomal abnormalities is increased in men with infertility.

Focused History

The initial evaluation for a woman who has been unsuccessful at becoming pregnant begins with a detailed history to ascertain whether the woman's sexual, menstrual, and social history matches the definition of infertility.[99,100] Once it has been determined that a couple's history matches the accepted definition of infertility, a detailed history exploring potential factors related to fertility status should occur. These factors can be related to female anatomy/physiology/psyche, male anatomy/physiology/psyche, or a combination (**Table 12-14**).[99,101]

Table 12-14	Female and Male Factors Associated with Infertility	
History for Both Partners	**Additional Female Medical History**	**Additional Male Medical History**
Current Nature of Intercourse		
Frequency of intercourse Duration of infertility		
Medical History		

History of any prior pregnancies	Prior contraception use	History of varicocele
History of sexually transmitted infections	Dyspareunia	History of mumps
History of genitourinary infection	Abnormal Pap smears or treatment	History of testicular torsion
Symptoms or known diagnosis of endocrine disorders	Pelvic or abdominal surgeries	
Prior chemotherapy or treatments for cancer	Obesity	
Current prescribed or over-the-counter medications	Menstrual history[a]	
History of sexual dysfunction		
Social History		
Smoking, recreational drugs or alcohol use		History or current use of anabolic steroids
Occupational History		
Regular exposure to toxic environmental or chemical substances such as dry-cleaning solvents, pesticides		Regular exposure to high levels of heat (e.g., steel factory workers)
Family History		
Family history of infertility	Family history of early menopause	
Family history of birth defects, developmental delays, cystic fibrosis	Galactorrhea, hirsutism	

[a] Menstrual history includes age of menarche, interval, regular or irregular, amount of flow, history of oligomenorrhea or amenorrhea, and dysmenorrhea.

Based on Koroma L, Stewart L. Infertility: Evaluation and initial management. *J Midwifery Womens Health.* 2012;57:614-621[99]; Lindsay TJ, Vitrikas KR. Evaluation and treatment of infertility. *Am Fam Physician.* 2015;91(5):308-314.[101]

Physical Examination

In addition to assessing vital signs and establishing body mass index (BMI), particular attention should be given to any signs of potential genetic or hormonal abnormalities such as excess androgen. For example, short stature could indicate Turner syndrome [45,XO]; acne, alopecia, or hirsutism should raise suspicion of elevated androgen levels; and galactorrhea could be a sign of hyperprolactinemia. A pelvic examination is performed to assess for conditions related to anatomy (e.g., fibroids, bicornuate uterus), infections (e.g., STIs), and hormonal states (e.g., hypoestrogenic signs, such as a lack of vaginal moisture and a cervix deficient in mucus) that could contribute to infertility.[99,100]

The biological male partner also should be examined, either by the midwife experienced with conducting male examinations, a reproductive specialist, or a urologist. In general, as with the female examination, the clinician looks for signs of anatomic conditions (e.g., enlarged testicular veins causing a varicocele or absent vans deferens), infectious conditions (e.g., prostatitis), and hormonal conditions (e.g., gynecomastia that could be an indication of hyperthyroidism or Klinefelter syndrome [47,XXY]) that can cause infertility.[99,100] Any

suspicious findings, in either the female or the male, indicate a need for referral to a reproductive specialist for further evaluation and care.

The midwife can initiate the assessment for infertility by first reviewing basic sexual knowledge and techniques that optimize the possibility of conception. Topics to review are listed in **Table 12-15**. The midwife can teach the person who is attempting conception to identify signs and symptoms of ovulation using one of the many methods available for this purpose, including charting of daily basal body temperature (BBT), self-evaluation of cervical mucus, ovulation over-the-counter tests, and smartphone apps. Details about using these methods can be found in the *Nonhormonal Contraception* chapter.

Table 12-15 Techniques for Optimizing Natural Fertility

Topic	Content
Timing of intercourse	Intercourse every 1–2 days results in the highest pregnancy rates, but should be based on couple's comfort.
Fertile window	The fertile window varies considerably even in women with regular cycles, but is usually considered to last 6 days. Conception is most likely when intercourse occurs within 3 days before ovulation.
	Three ways of determining the fertile window are available:
	BBT recorded at rest at the same time each day before rising. Fertility is highest 2 days after BBT increases by at least 0.4°F.
	The woman can be taught to self-monitor her cervical mucus. Fertility is highest when the mucus is slippery and clear and intercourse occurs on the day when cervical mucus is most profuse.
	Ovulation detection kits are based on the surge of LH that is associated with ovulation. These kits may underestimate the fertile window, as ovulation can occur up to 2 days after the LH surge.
Coital practices	There is no evidence that coital position improves fertility. Sperm can be found in fallopian tubes within 2 minutes after ejaculation.
	Vaginal lubricants may decrease sperm motility.
Lifestyle considerations	Tobacco and excessive caffeine consumption (> 5 cups of coffee per day) are associated with decreased fertility. The effects of alcohol or marijuana have not been determined.
	Sauna use does not decrease fertility.

Abbreviations: BBT, basal body temperature; LH, luteinizing hormone.

Diagnostic Testing

The initial laboratory testing includes ordering thyroid-stimulating hormone (TSH), free thyroxine (T_4), and prolactin levels for the female desiring conception to rule out thyroid conditions or prolactinemia, which could be associated with infertility. Simultaneously, the male partner may be assessed for thyroid conditions by testing the levels of TSH and T_4, as well as undergo evaluation of testosterone levels for evidence of diminished endocrine production.[99] Male partners initially need a semen analysis to gauge the number, concentration, motility, morphology, form, and structure of sperm per ejaculate. Since conditions associated with biological male partners have been shown to contribute significantly to infertility, and further testing of the female partner can be invasive and costly, an assessment of the male partner's fertility status is conducted early in the process of evaluation for infertility.

Once the basic assessments have been completed, a series of more invasive and expensive procedures are undertaken. Generally, these further steps are conducted under the guidance and care of a reproductive specialist, and can include the use of ultrasonography, magnetic resonance imaging, and laparoscopy.

Treatment of the Woman/Couple with Infertility

A detailed presentation of the treatments available for infertility is beyond the scope of this text. Nevertheless, midwives counseling women and couples diagnosed with infertility should familiarize themselves with the choices available for treatment. These include the use of medications (e.g., clomiphene citrate [Clomid]), surgery (e.g., fimbrioplasty to remove adhesions from fallopian tubes), insemination procedures, and assisted reproductive technologies (ART), such as in vitro fertilization (IVF). Complementary approaches that have been assessed include the use of acupuncture, chiropractic, prayer, antioxidant supplements (e.g., selenium intake to improve the properties of sperm), and psychotherapy, although evidence for the success rates of these approaches is weak and conflicting.[99]

Overall, it is very difficult to predict the success rates of completed pregnancies and live births resulting from treatment for infertility.[101] Many factors contribute to the cause of infertility as well as to the subsequent success of available treatment. Midwives caring for individuals with infertility who are undergoing treatment can provide emotional support in recognition of the physical, emotional, and social pressures that infertile couples face. **Table 12-16** provides a sample of critical elements included in a transfer note after a midwife has performed initial evaluation and determines that more specialized care is needed by a woman/couple with infertility.

Table 12-16 Sample Critical Elements for a Transfer Note for a Woman/Couple with Infertility

Risk factors, if any, that are identified
Current medications
Menstrual history
Past health history of note
Length of time attempting pregnancy (> 1 year if the woman is < 35 years; > 6 months if she is ≥ 35 years)
Care provided: procedures and results for woman/couple; laboratory results or if pending
Summary of rationale for transfer
Request for information about the treatment plan for follow-up

One important aspect with which midwives should be familiar is the prevention of infertility at the outset. Although many causes of infertility involve chromosomal, anatomic, or physiologic factors that are beyond a couple's control, assisting women in leading healthy lifestyles that include safe sex practices, nutritious diets, exercise, and avoidance of smoking or excessive alcohol intake can facilitate desired fertility. Educating clients to avoid exposure to potentially harmful environmental toxins is also important.

Alterations in Women's Sexual Function

Sexuality is an essential component of the assessment of a woman's well-being and gynecologic health. Alterations in women's sexual function may occur with any of the gynecologic conditions described in this chapter, or they may occur independently of them. Women and their healthcare providers often find it difficult to speak about sexuality. The topic can be embarrassing for some, and does not easily fit into a typical 15-minute ambulatory site appointment schedule. Lack of desire may be a major concern for some women, whereas it seems to be a minor consideration or a nonissue for others. Stereotypes abound that can inhibit communication between the provider and the woman and/or between the woman and her sexual partner(s). Even a standard definition of *normal* sexual function is elusive.

Alterations in sexual function are reported more commonly by older persons those with chronic diseases such as cardiac, kidney, and obstructive pulmonary disease, and those who are post cancer treatment.[103-106] Women with endometriosis and infertility are reported to have a higher prevalence of sexual concerns than women in the general population.[107,108] Psychotropic medications, antihypertensive drugs, lipid-lowering agents, and hormones may have negative implications related to sexual function.[109,110]

Sexual Dysfunction Disorders

Four categories of female sexual dysfunction currently are generally accepted among healthcare providers; **Table 12-17** provides an overview of these disorders.[110-113] Regardless of the type or category of disorder, the initial step for assessment includes a thorough history and physical examination. Behavioral counseling is a major intervention for sexual dysfunction disorders since it is considered effective and the presumed harm is low. Other strategies for treatment include mindfulness, as well as creams, gels, and pelvic floor exercises, although the results with these modalities have been inconsistent.[110,111] The vast majority of studies are limited by small numbers of participants, short duration, variation in how alterations in sexual function are defined, and variation in how sexual dysfunction is measured.

Table 12-17 Categories of Female Sexual Dysfunction

Category	Description	Common Management Strategies
Sexual interest/arousal disorder	Includes HSSD, which involves distressing low desire for sexual activity or persistent deficient sexual fantasies. Previously known by the value-laden term of "frigidity," this category also encompasses women who are unable to experience arousal by genital stimulation or nongenital stimulation (e.g., kissing, experiencing erotic situations). Associated with lack of vaginal lubrication. Perimenopausal and postmenopausal women may report secondary arousal problems.	Few studies have found effectiveness of sildenafil (Viagra) in women, but a newer agent, flibanserin (Addyi; 5-HT1a) is FDA approved for this indication. Some evidence of effectiveness for estrogen with or without progesterone therapy has been found for this indication among older women, although the research is not strong.
Substance/medication-	Prominent among the drugs associated with	Changes in types, dose, and other aspects

induced disorder	female sexual problems are antidepressants, especially SSRIs and SNRIs.	of the offending medication are indicated. Determining the optimal antidepressant may take time; care is best managed by a specialist.
Orgasmic disorder	Defined as a persistent or recurrent problem in attaining sexual arousal or the inability to maintain arousal. May be associated with use of antidepressants as well as diseases influencing neurologic health (e.g., diabetes).	Although no FDA-approved drug exists for treating women with this disorder, many providers may prescribe testosterone products on an off-label use with some success. However, type, route, and dose are uncertain.
Genito-pelvic pain disorders	This category includes dyspareunia and vaginismus. Etiology varies, but symptoms often include vestibulodynia, interstitial cystitis, vaginal infections, and vaginal atrophy.	Treatment usually is based on the etiological agent.

Abbreviations: FDA, U.S. Food and Drug Administration; HSSD, hypoactive sexual desire disorder; SNRIs, serotonin–norepinephrine reuptake inhibitors; SSRIs, selective seroton in reuptake inhibitors.

[a] Behavioral counseling is a relatively low-risk intervention that has been used as a treatment modality or in conjunction with other treatments for any woman with a disorder in one of these categories.

Based on Jayne CJ, Heard MJ, Zubair S, Johnson DL. New developments in the treatment of hypoactive sexual desire disorder: a focus on flibanserin. *Int J Womens Health*. 2017;9:171-178[110]; Nastri CO, Lara LA, Ferriani RA, Rosa-e-Silva ACJS, Figueiredo JBP, Martins WP. Hormone therapy for sexual function in perimenopausal and postmenopausal women. *Cochrane Database Syst Rev.* 2013;6:CD009672. doi:10.1002/14651858.CD009672.pub2[111]; Parish SJ, Hahn SR. Hypoactive sexual desire disorder: a review of epidemiology, biopsychology, diagnosis, and treatment. *Sex Med Rev*. 2016;4(2):103-120[112]; Schmidt HM, Hagen M, Kriston L, Soares-Weiser K, Maayan N, Berner MM. Management of sexual dysfunction due to antipsychotic drug therapy. *Cochrane Database Syst Rev.* 2012;11:CD003546. doi:10.1002/14651858.CD003546.pub3.[113]

As female sexuality and alterations in female sexual function become more recognized as an integral component of health, clinicians need research that is robust and meaningful to guide counseling and practice. Healthcare providers who specialize in this area are few in number, but referral is indicated for persons with sexual dysfunction disorders. Midwives may consider additional education and preparation to provide this specialized care so that they can increase women's access to such services and reduce the need for outside referral.

Cancer of the Uterus, Ovary, Vulva, and Vagina

Cervical cancer screening is a well-known primary prevention strategy. By comparison, endometrial cancer is the most common gynecologic cancer in the United States and the second most common gynecologic cancer after cervical cancer in low-resource nations.[114] Perhaps the most troubling of gynecologic cancers is ovarian cancer: It is the second most common gynecologic cancer in the United States but accounts for a greater ratio of deaths per diagnosis than does endometrial cancer or breast cancer.

Cancer of the Uterine Endometrium

While a variety of carcinomas may be found in the uterus, including sarcomas and gestational trophoblastic tumors (e.g., hydatidiform mole), the endometrium is the source of approximately 90% of the cancers involving the corpus of the uterus.[115] When detected early in the disease process, the cure rate for endometrial cancer is high because tumors tend to be localized and well defined. Even so, the incidence of endometrial cancer results in more than 10,000 deaths per year, a mortality rate that, based on the ratio of deaths to new diagnoses (1 to 5.4), is comparable to that of breast cancer (1 to 5.2).[115,116]

Conditions associated with a prolonged exposure to unopposed estrogen are risk factors for development of endometrial cancer. Examples include early menarche or late menopause, polycystic ovary syndrome with anovulation, nulliparity, and use of unopposed estrogens or tamoxifen (Nolvadex). Factors that suggest an underlying prolonged exposure to estrogen may have occurred include unexplained vaginal bleeding in postmenopausal woman, Lynch syndrome, infertility, obesity, and history of breast or ovarian cancer. The most common sign of endometrial cancer, which always requires diagnostic follow-up, is unexplained vaginal bleeding, especially when it is experienced by a postmenopausal woman. Thus, the most widely recommended screening approach for endometrial cancer is education of women about the signs, symptoms, and risk factors associated with the disease.

Endometrial cancer is categorized into two types. Type I, commonly referred to as endometrioid adenocarcinoma, is the most common form, accounting for approximately three-fourths of all cases. Endometrioid adenocarcinoma usually occurs in the perimenopausal years, and if discovered in its early stages, is treatable. Type II, which is unrelated to the presence of estrogen, is less common and has a more dire prognosis than Type I. Some oncologists include a third subclassification, which is hereditary in nature and rarer than Types I and II. The most common cause of this third type of endometrial tumor is Lynch syndrome, which is also called hereditary nonpolyposis colorectal cancer. Lynch syndrome is caused by a genetic mutation that causes the individual to have a high risk for developing colorectal cancer as well as other cancers such as endometrial, ovary, and gastrointestinal cancers.[116]

The first *diagnostic* test for endometrial cancer is endometrial biopsy (EMB), which will reveal normal tissue, hyperplasia, or possibly endometrial cancer depending on the sample obtained. The procedure for performing an endometrial biopsy is presented in the *Endometrial Biopsy* appendix in the *Menstrual Cycle Abnormalities* chapter. Traditionally, the other standard method used for preliminary diagnosis of endometrial cancer has been transvaginal

ultrasound. The most effective method to definitively diagnose endometrial cancer following an EMB or transvaginal ultrasound is to perform hysteroscopy accompanied by a dilatation and curettage (D & C).[116]

For a postmenopausal woman, *any* vaginal bleeding, except that experienced as part of an expected withdrawal bleeding while on hormone therapy, is considered a possible sign of cancer requiring further evaluation. Other signs or symptoms of endometrial cancer, such as pain, usually occur during late-stage disease, when the cancer has spread beyond the uterus.

Other Uterine Cancers

While endometrial cancer is by far the most prevalent form of uterine cancer, two additional forms of abnormal growth are important to note: gestational trophoblastic tumors (e.g., hydatidiform mole) and uterine sarcoma. The most common of these tumors is hydatidiform mole, which consists of abnormal growth of placental tissue. In its beginning stages, this tumor is a benign growth. Women with hydatidiform mole present with a positive pregnancy test, usually with excessively elevated human chorionic gonadotropin (hCG) levels, and abnormal bleeding. The uterus commonly will be found to be larger than expected based on the woman's gestational dates, and confirmation of diagnosis is accomplished through ultrasonography. Treatment most frequently involves the use of suction curettage with the goal of preventing the development of metastatic disease.[117] Additional information on hydatidiform mole can be found in the *Pregnancy-Related Conditions* chapter.

Uterine sarcomas are a rare form of gynecologic cancer (< 1% of these cancers) that account for approximately 3% to 7% of all uterine carcinomas.[118] Generally occurring after menopause, these anomalies originate in the endometrium, the myometrial muscle, or the endometrial stroma. Risk factors include prior therapeutic radiation to the pelvic region. Prolonged use of tamoxifen, and African Caribbean ancestry.[118] There is no recommended form of regular screening, other than the assessment of uterine size and shape that occurs during a gynecologic examination. The most common symptom/sign of this form of cancer is abnormal uterine bleeding. While sarcomas cannot be easily distinguished on physical examination from benign leiomyomas, it is important to remember that sarcomas usually are found in postmenopausal women, whereas uterine fibroids more commonly manifest themselves prior to menopause, and begin to regress in size after menopause when estrogen stimulation diminishes.

If sarcoma is suspected, further assessment may include an endometrial biopsy or a hysteroscopic-guided D & C. However, these diagnostic instruments are less sensitive for sarcomas than they are for endometrial carcinomas. By comparison, more sensitive assessment is achieved with imaging studies, such as computed tomographic scans used to guide needle biopsy or MRI. Employing ultrasound for the diagnosis of sarcoma is not very useful.

Generally, the midwife refers the woman in whom uterine sarcoma is suspected to an appropriate specialist. Prognosis depends on the extent of the disease when diagnosis is made. If the cancer is confined to the uterine corpus at the time of discovery, the 5-year survival rate is 50%, whereas the 5-year survival rate for women with sarcoma that has metastasized ranges from 0% to 20%.[118] Clearly, early discovery is vital to curtailing the spread of this disease—one reason why the significance of uterine bleeding in postmenopausal women should be

stressed.

Ovarian Cancer

Ovarian cancer can occur in epithelial, germ cell, or sex cord stromal cell tissue. The majority of ovarian cancers are epithelial. The 5-year survival rate for women with ovarian cancer is markedly less than that for women with other cancers, primarily because ovarian cancer is often first diagnosed at a late stage. The lifetime risk of being diagnosed with breast cancer is 1 in 8 women whereas the comparable lifetime risk of being diagnosed with ovarian cancer is 1–2 per 100 women.[119] However, this risk varies among different populations. For example, the risk of developing ovarian cancer is greatly increased in women who have a mutation in breast cancer gene 1 (*BRCA1*) or breast cancer gene 2 (*BRCA2*). As with most cancers, the risk of developing ovarian cancer increases with age: It is most prevalent after the age of 45 years; the median age of diagnosis is 63 years.

Known and possible risk factors for ovarian cancer include a history of breast, uterine, or colorectal cancer; postmenopausal status; evidence of mutations in the *BRCA1* or *BRCA2* genes; rarer mutations in more recently discovered genetically based cancers (e.g., the *PTEN* gene associated with Cowden disease that causes a form of thyroid cancer and breast cancer); Lynch syndrome; more than 40 years of active ovulation (e.g., nulliparous never-user of oral contraceptives); early menarche or late menopause; use of infertility drugs for longer than 1 year; and use of unopposed estrogen therapy.[120-123] Notably, repetitive stimulation of the epithelium of the ovary is theorized to result in the development of a malignant state. In contrast, high parity, use of oral contraceptives or injectable depot medroxyprogesterone acetate (DMPA), breastfeeding, hysterectomy and/or salpingectomy, and prophylactic oophorectomy are all associated with a decreased risk of developing ovarian cancer.[121-123]

Bimanual examinations during routine healthcare visits had long been the only screening method for ovarian cancer not dependent on self-report of symptoms, but the sensitivity and specificity of these clinical examinations with regard to detecting ovarian cancer are low. Because a history and physical examination have not been predictive for the detection of ovarian cancer, particularly in its earlier stages, other assessment methods have been suggested as screening mechanisms for this disease. One detection method recommended in the recent past was the presence of increased blood levels of the CA-125 tumor antigen in women with early-onset ovarian cancer. Although CA-125 levels are elevated in women with ovarian cancer, the specificity of this biomarker is limited because elevated CA-125 levels are associated with many other conditions as well. This is an area of ongoing research.

Approximately 10% to 20% of women who develop ovarian cancer have an inherited genetic mutation in *BRCA1* or *BRCA2*. Risk factors that increase the chance of having a *BRCA1* or *BRCA2* mutation are presented in the *Breast Conditions* chapter and reviewed in the *Health Promotion Across the Lifespan* chapter. The term hereditary breast and ovarian cancer syndrome (HBOC) refers to persons who have a family history of multiple cases of breast, ovarian, and/or peritoneal cancers in first-degree relatives. Criteria for offering women screening for HBOC is reviewed in the *Breast Conditions* chapter. Women who are at increased risk for having a *BRCA1* or *BRCA2* mutation should be offered genetic

counseling.[124-127] In short, screening all women for a family history that suggests they are at risk for HBOC and offering those women genetic counseling, which may include testing for *BRCA* mutations, is the current recommendation for ovarian cancer screening.

The lack of evidence regarding the exact etiology of ovarian cancer, definitive risk factors, and means of early detection make screening for this disease difficult for individual women and practitioners alike. Reminders for women about the signs and symptoms of ovarian cancer may potentially aid in detecting this carcinoma. These signs and symptoms include abdominal distension or bloating; flatulence, difficulty eating or easily feeling full when eating, and other persistent gastrointestinal disturbances, including either diarrhea or constipation; abdominal or pelvic pain; fatigue; urinary complaints, including urgency or frequency; and irregular vaginal bleeding.[128]

Cancer of the Vulva and the Vagina

Cancer of the vulva and the vagina account for approximately 6% to 7% of all gynecologic malignancies. The relative 5-year survival rates for vulvar and vaginal cancers are approximately 72% and 49%, respectively.[129,130] The lower survival rate for women with vaginal cancer is in part due to the fact that this malignancy usually presents as a result of metastatic spreading of a cancer whose primary source of development was in another gynecologic organ, such as the vulva or the cervix. When vaginal cancer is discovered prior to spreading beyond the vagina, involving lymph nodes, or metastasizing, the 5-year survival rate is approximately 84%.[131] The vast majority of both forms of cancer are squamous cell in type, and the incidence increases with age.

Both vulvar and vaginal cancer have been increasingly associated with HPV exposure.[132] Consequently, the CDC now emphasizes that receiving the HPV vaccine will protect not only against cervical cancer, but also against vulvar and vaginal cancer. Additionally, any woman with a history of infection with a high-risk HPV type, most likely identified on a Pap test, should be considered at risk for vulvar or vaginal cancer, though the incidence of these cancers is less than the incidence of cervical cancer. Other risk factors for vulvar and vaginal malignancies include smoking, multiple sex partners, other gynecologic cancers, lichen sclerosus, and immunosuppressive disorders (e.g., HIV). A rarer form of vaginal cancer, found primarily in female offspring of women who took DES during pregnancy, is clear cell adenocarcinoma. It first occurs in late adolescence and early twenties, although most of the women affected are now much older and no longer at risk for clear cell adenocarcinoma.[133]

Both screening for and diagnosis of vulvar and vaginal cancers include the identification of any growths in the genital area noted by the woman, or by the midwife during the pelvic examination. The most common site of vulvar cancer is the labia majora, while the most common location of vaginal cancer is the upper third of the vaginal wall.

Early symptoms of vulvar cancer may be persistent pruritus and the presence of condylomata, whereas vaginal disease may be accompanied by bleeding, unusual discharge, and pelvic discomfort. Depending on the extent of the disease process, pelvic examination may reveal a mass at the site of involvement, and possibly lymphadenopathy. Any suspicious lesion should be biopsied, either by a midwife with experience in collecting such samples or by the

specialist to whom the woman has been referred. Referral should occur if any of the following exist: unexplained lymphadenopathy, an HPV lesion that does not improve with topical treatment, or a lesion with an unusual presentation or continued growth over time. As with other gynecologic malignancies, early detection of a vulvar or vaginal cancer can result in a high rate of survival.

Congenital Uterine Anomalies

Congenital uterine anomalies, also referred to as Müllerian anomalies, are present in as many as 10% of all biological females, although the majority of these anomalies are minor.[133-135] These conditions often go undiagnosed until a woman presents for evaluation due to infertility, recurrent miscarriages, pelvic pain, difficulty with vaginal penetrative sexual activity, or ectopic pregnancy.[136] The diagnosis may be made by pelvic examination, by ultrasound, or during a surgical procedure.

During development of the embryo, the two Müllerian ducts differentiate to form the uterus, cervix, superior portion of the vagina, and fallopian tubes. Abnormal development results in a wide range of anatomic malformations. Classification of congenital uterine anomalies has been proposed by the American Society for Reproductive Medicine. Although attempts have been made to update this classification system with more detailed categorizations, the ASRM system remains the standard for describing uterine anomalies and is presented in **Table 12-18**.[136]

Table 12-18 Classification of Congenital Uterine Anomalies

Classification	Description
Class I Hypoplasia or Agenesis	
I A	Vagina (with or without uterine abnormality)
I B	Cervix
I C	Fundus of uterus
I D	Fallopian tube(s)
I E	Combination of above
Class II Unicornuate Uterus (One Single Horn of Uterus)	
II A	Rudimentary horn with endometrial cavity and communicating with single horn of uterus
II B	Rudimentary horn with endometrial cavity but noncommunicating with single horn of uterus
II C	Rudimentary horn without endometrial cavity
II D	No rudimentary horn
Class III Uterus Didelphys	
III	Two separate cavities with two separate cervices
Class IV Bicornuate Uterus	
IV A	Complete: Two separate uterine cavities separated by myometrial tissue and one cervix
IV B	Partial: Septum confined to fundus
Class V Septate Uterus	
V A	Complete: The septum extends into the internal cervical os with two cavities separated by avascular tissue and one cervix
V B	Partial: The septum does not reach the internal os
Class VI Arcuate Uterus	

VI	Concave fundus instead of convex or straight
Class VII Diethylstilbestrol-Related Anomalies	
VII A	T-shaped uterus
VII B	T-shaped uterus with dilated horns
VII C	Uterine hypoplasia

Based on American Fertility Society. The American Fertility Society classifications of adnexal adhesions, distal tubal occlusion, tubal occlusion secondary to tubal ligation, tubal pregnancies, Müllerian anomalies and intrauterine adhesions. *Fertil Steril.* 1988;49:944-955.[136]

Normal fertilization may not be possible for women with some Müllerian anomalies, such as agenesis of the fallopian tubes. Other anomalies such as a communicating unicornuate uterus have been found to be associated with endometriosis and dysmenorrhea. However, an arcuate uterus may have no negative impact on conception and pregnancy.

The most recent classification of congenital anomalies by ASRM relates to changes due to the use of DES, a nonsteroidal estrogen that was widely prescribed from 1948 to 1971 in an effort to prevent a number of pregnancy-related complications. Approximately 5 to 10 million pregnant women received DES during this time frame, after which DES was discovered to be not only ineffective but actually harmful to the intrauterine fetus.[133,137] A number of structural and functional abnormalities have been identified as the direct effects of in utero exposure to DES, including changes to the uterus, vagina, and cervix. Reproductive health risks in DES daughters (i.e., biological females exposed to DES in utero) include infertility, ectopic pregnancy, spontaneous abortion, and premature birth. In addition, DES daughters are at greater risk for cervical and uterine carcinomas at age 30 years or younger, and at greater risk for breast cancer at age 40 years and older.[133,137]

To date, the limited data on the outcomes of DES-exposed grandchildren have not demonstrated uterine abnormalities in these individuals. In contrast, animal studies have shown a significant increase in uterine cancers, ovarian tumors, and lymphomas in third-generation offspring of DES-exposed subjects.[133,137] Thus, when obtaining a woman's health history, it is recommended that the midwife inquire whether the woman knows if her mother was exposed to DES, especially if the woman's mother has a history of infertility, difficult pregnancies, or reproductive organ cancers.[138]

Based on the type of anomaly, the contraceptive choices of a woman may be somewhat complicated. The midwife can offer continued support and referral to local resources or national organizations that provide web-based information for women with congenital uterine anomalies. Concerns related to fertility and childbearing are important to reproductive-age women, and should be promptly addressed. Regardless of the woman's age or childbearing plans, information pertaining to an organ anomaly may have a significant emotional impact. Selected resources are listed at the end of this chapter.

Conclusion

The role of the midwife involves care for women across the lifespan. Both historically and currently, that care includes the diagnosis and management of women's gynecologic health. This chapter summarizes some of the more common gynecologic disorders and some additional conditions such as pelvic organ prolapse and alterations in sexual function, and presents the associated diagnostic and management steps. In addition, it provides an overview of gynecologic cancers.

Essential to all midwifery care, for every element of practice, and for every situation, the key to excellent care is listening to the woman and really hearing what she says about her personal symptoms or conditions. Since optimal gynecologic health is a goal for all women, it is essential that the midwife have a working knowledge of the information in this chapter regarding the diagnosis and management of common gynecologic problems, and screening for more complicated conditions. Knowing where and how to access resources and expert care is a lifelong skill for practicing midwives.

Resources

Organization	Description	Webpage
Agency for Healthcare Research and Quality (AHRQ)	Federal agency that publishes multiple guidelines, including guidelines from other groups such as the U.S. Preventive Services Task Force. Among the publications available online are those dealing with cervical screening, management of urinary incontinence, cancer and contraception, and vulvar carcinoma.	https://www.ahrq.gov
American College of Nurse-Midwives (ACNM)	Midwifery organization that includes information about standards and scope of practice as well as a number of position statements. These position statements include *Collaborative Management in Midwifery Practice for Medical, Gynecologic, and Obstetric Conditions* and *Joint Statement of Practice Relations Between Obstetrician-Gynecologists and Certified Nurse-Midwives/Certified Midwives* as well as *Standards for the Practice of Midwifery* that address how to incorporate new procedures into practice.	http://www.midwife.org
American College of Obstetricians and Gynecologists (ACOG)	Organization that issues multiple guidelines and recommendations regarding women's health care. Although large parts of the website are behind a firewall, Committee Opinions and Practice Bulletins can usually be found published in the journal *Obstetrics & Gynecology.* Toolkits on female sexual dysfunction and human papillomavirus vaccinations also are available.	https://www.acog.org
American Society for Reproductive Medicine (ASRM)	Practice guidelines on this website include recommended algorithms for evaluating female and male infertility.	http://www.asrm.org/news-and-publications/practice-committee-documents/
Association of Professional Piercers (APP)	An international nonprofit organization dedicated to the dissemination of vital health and safety information about body piercing to piercers, healthcare professionals, legislators, and the general public.	https://safepiercing.org
Centers for Disease Control and Prevention (CDC)	Consumer-focused page on selected bibliographies related to diethylstilbestrol exposure.	https://www.cdc.gov/des/bibliographies/index.html
National	Federal agency that includes professional	https://www.cancer.gov

Cancer Institute (NCI)	information about a variety of cancers, including cervical, ovarian, peritoneal, vaginal, and vulvar cancer. Clinical databases about research in the area as well as clinical trials are available.	
Our Bodies Ourselves (OBOS)	Provides information about DES exposure, health risks, and other gynecologic conditions.	http://www.ourbodiesourselves.org/health-info/des/
Practicing Physician Education in Geriatrics Project (PPE)	Urinary incontinence toolkit.	http://www.gericareonline.net/tools/eng/urinary/index.html
	These sites have the Questionnaire for female Urinary Incontinence Diagnosis (QUID) and other bladder diary tools as well as professional resources.	http://bronchiectasis.com.au/wp-content/uploads/2015/09/The-Questionnaire-for-female-Urinary-Incontinence-Diagnosis.pdf
Point-of-Care Apps		
American Society for Colposcopy and Cervical Pathology (ASCCP)	Guidelines for management of a woman with an abnormal Pap test. More than a dozen algorithms are included in this site, covering a large number of Pap test results and situations stratified by age and human papillomavirus status, among other factors. A downloadable mobile app is also available from the website.	http://www.asccp.org/Assets/51b17a58-7af9-4667-879a-3ff48472d6dc/635912165077730000/asccp-management-guidelines-august-2014-pdf http://www.asccp.org
International Society for the Study of Vulvovaginal Disease (ISSVD)	Document in outline form describing vulvar lesions by color and shape, to help with diagnosis and treatment. A downloadable app is available for a nominal fee.	https://issvd.org

References

1. American College of Nurse-Midwives. *Definition of Midwifery and Scope of Practice of Certified Nurse-Midwives and Certified Midwives*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
2. Fullerton J, Sipe T, Hastings-Tolsma M, et al. The midwifery workforce: ACNM 2012 and AMCB 2013 core data. *J Midwifery Womens Health* 2015;60:751-761.
3. Deneris A, Wheeler C, Salmon S. Development and pilot outcome data of a midlife women's health assessment clinic: a comprehensive and multidisciplinary approach to health care. *J Midwifery Womens Health*. 2013;58:328-332.
4. Phillippi JC, Barger MK. Midwives as primary care providers for women. *J Midwifery Womens Health* 2015;60:250-257.
5. U.S. Preventive Services Task Force. Gynecological conditions: periodic screening with the pelvic examination. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/gynecological-conditions-screening-with-the-pelvic-examination. Accessed September 16, 2017.
6. Centers for Disease Control and Prevention. Cancer among women. Available at: https://www.cdc.gov/cancer/dcpc/data/women.htm. Accessed September 16, 2017.
7. Collins M, Fantasia H. Sexually transmitted infections. In: Schuiling K, Likis F, eds. *Women's Gynecologic Health*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017:465-511.
8. Liu Z, Rashid T, Nyitray AG. Penises not required: a systematic review of the potential for human papillomavirus horizontal transmission that is non-sexual or does not include penile penetration. *Sexual Health*. 2015;13:10-21.
9. Doorbara J, Quint W, Banks L, et al. The biology and life-cycle of human papillomaviruses. *Vaccine*. 2012;30S:F55-F70.
10. Gibb RK, Martens MG. The impact of liquid-based cytology in decreasing the incidence of cervical cancer. *Rev Obstet Gynecol*. 2011;4(suppl 1):s2-s11.
11. Wright TC, Stoler MH, Behrens CM, Sharma A, Zhang G, Wright TL. Primary cervical cancer screening with human papillomavirus: end of study results from the ATHENA study using HPV as the first-line screening test. *Gynecol Oncol*. 2015;136(2):189-197.
12. Huh W, Ault K, Chelmow D, et al. Use of primary high-risk human papillomavirus testing for cervical cancer screening: Interim clinical guidance. *J Lower Genit Tract Dis*. 2015;19:91-96.
13. Saslow D, Solomon D, Lawson H, et al. American Cancer Society, American Society for Colposcopy and Cervical Pathology, and American Society for Clinical Pathology screening guidelines for the prevention and early detection of cervical cancer. *J Low Genit Tract Dis*. 2012;16:175-204.
14. American College of Obstetricians and Gynecologists. Practice Bulletin No. 168: cervical cancer screening and prevention: interim update. *Obstet Gynecol*. 2016;128:e111-e130.
15. Moyer V. Screening for cervical cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2012;156:880-891.
16. American College of Obstetricians and Gynecologists. Practice Bulletin No. 140: management of abnormal cervical cancer screening test results and cervical cancer precursors. *Obstet Gynecol*. 2013;122:1338-1367. [Reaffirmed 2016].
17. Massad L, Einstein M, Huh K, et al., for 2012 ASCCP Consensus Guidelines Conference. 2012 updated consensus guidelines for the management of abnormal cervical cancer screening tests and cancer. *J Low Genit Tract Dis*. 2013;17:S1-S27.
18. Nayar R, Wilbur DC. The Pap test and Bethesda 2014. *Cancer Cytopathol*. 2015;123:271-281.
19. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines 2015. *MMWR Recomm Rep*. 2015;64(RR-03):1-137. Available at: https://www.cdc.gov/std/tg2015/default.htm. Accessed October 1, 2017.
20. American College of Obstetricians and Gynecologists. Committee Opinion No. 672: clinical challenges of long-acting reversible contraceptive methods. *Obstet Gynecol*. 2016;128:e69-e77.
21. Kim YJ, Youm J, Jee BC. *Actinomyces*-like organisms in cervical smears: the association with intrauterine device and pelvic inflammatory disease. *Obstet Gynecol Sci*. 2014;57(5):393-396.
22. Carr KC, Sellors JW. Cervical cancer screening in low resource settings using visual inspection with acetic acid. *J Midwifery Womens Health*. 2004;49(4):329-337.
23. Poli U, Bidinger P, Gowrishankar S. Visual inspection with acetic acid (VIA) screening program: 7 years experience in early detection of cervical cancer and pre-cancers in rural south India. *Indian J Community Med*. 2015;40:203-207.
24. Kim JH, Kim I-W, Kim Y-W, et al. Comparison of single-, double- and triple-combined testing, including Pap test, HPV DNA test and cervicography, as screening methods for the detection of uterine cervical cancer. *Oncol Rep*. 2013;29:1645-1651.
25. American College of Nurse-Midwives. Standards for the practice of midwifery. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000051/Standards_for_Practice_ Accessed November 10, 2017.
26. Wentzensen N, Massad S, Mayeaux EJ, et al. Evidence-based consensus recommendations for colposcopy practice for cervical cancer prevention in the United States. *J Lower Genit Tract Dis*. 2017;21(4):216-222.

27. Ocque R, Austin M. Follow-up of women with negative Pap test results and abnormal clinical signs or symptoms. *Am J Clin Pathol*. 2016;145:560-567.
28. Ayorinde AA, Macfarlane GJ, Saraswat L, Bhattacharya S. Chronic pelvic pain in women: an epidemiological perspective. *Womens Health* 2015;11:851-864.
29. Steege JF, Siedhoff MT. Chronic pelvic pain. *Obstet Gynecol*. 2014;124:616-629.
30. Cheong YC, Smotra G, Williams ACDC. Non-surgical interventions for the management of chronic pelvic pain. *Cochrane Database Syst Rev*. 2014;3:CD008797. doi:10.1002/14651858.CD008797.pub2.
31. Zielinski RE. Assessment of women's sexual health using a holistic, patient-centered approach. *J Midwifery Womens Health*. 2013;58:321-327.
32. Ferguson C. Providing quality care to the sexual assault survivor: education and training for medical professionals. *J Midwifery Womens Health*. 2006;51(6):486-492.
33. Bhavsar AK, Gelner EJ, Shorma T. Common questions about the evaluation of acute pelvic pain. *Am Fam Physician*. 2016;93(1):41-48.
34. Ball J, Dains J, Flynn J, Solomon BS, Stewart RW. *Seidel's Guide to Physical Examination*. 8th ed. St. Louis, MO: Mosby Elsevier; 2015.
35. Fauconnier A, Dallongeville E, Huchon C, Ville Y, Falissard B. Measurement of acute pelvic pain intensity in gynecology: a comparison of five methods. *Obstet Gynecol*. 2009;113:260-269.
36. Ness RB, Soper DE, Holley RL, et al. Effectiveness of inpatient and outpatient treatment strategies for women with pelvic inflammatory disease: results from the Pelvic Inflammatory Disease Evaluation and Clinical Health (PEACH) randomized trial. *Am J Obstet Gynecol*. 2002;186:929-937.
37. Ross EK, Kebria M. Incidental ovarian cysts: when to reassure, when to reassess, when to refer. *Cleveland Clin J Med*. 2013;80(8):503-514.
38. Timmerman D, Van Calster B, Testa A, et al. Predicting the risk of malignancy in adnexal masses based on the Simple Rules from the International Ovarian Tumor Analysis group. *Am J Obstet Gynecol*. 2016;214:424-437.
39. Levine D, Brown DL, Andreotti RF, et al. Management of asymptomatic ovarian and other adnexal cysts imaged at US Society of Radiologists in Ultrasound consensus conference statement. *Ultrasound Qtly*. 2010;26:121-131.
40. Sasaki K J, Miller C E. Adnexal torsion: review of the literature. *J Minim Invasive Gynecol*. 2014;21:196-202.
41. Parasar P, Ozcan P, Terry KL. Endometriosis: epidemiology, diagnosis and clinical management. *Curr Obstet Gynecol Rep*. 2017;6(1):34-44.
42. Revised American Society for Reproductive Medicine classification of endometriosis: 1996. *Fertil Steril*. 1997;67(5):817-821.
43. Schliep KC, Stanford JB, Chen Z, et al. Interrater and intrarater reliability in the diagnosis and staging of endometriosis. *Obstet Gynecol*. 2012;120(1):104-112.
44. Brown J, Farquhar C. Endometriosis: an overview of Cochrane reviews. *Cochrane Database Syst Rev*. 2014;3:CD009590. doi:10.1002/14651858.CD009590.pub2.
45. Burney RO, Giudice LC. Pathogenesis and pathophysiology of endometriosis. *Fertil Steril*. 2012; 98:511-519.
46. Rahmioglu N, Nyholt DR, Morris AP, Missmer SA, Montgomery GW, Zondervan KT. Genetic variants underlying risk of endometriosis: insights from meta-analysis of eight genome-wide association and replication datasets. *Hum Reprod Update*. 2014;20:702-716.
47. Parazzini F, Esposito G, Tozzi L, Noli S, Bianchi S. Epidemiology of endometriosis and its comorbidities. *Eur J Obstet Gynecol Reprod Biol*. 2016;209:3-7.
48. American College of Obstetricians and Gynecologists. Practice Bulletin No. 114: management of endometriosis. *Obstet Gynecol*. 2010;116:223-236. [Reaffirmed 2016].
49. Rice K, Secrist J, Woodrow E, Hallock L, Neal J. Etiology, diagnosis, and management of uterine leiomyomas. *J Midwifery Womens Health*. 2012;57:241-247.
50. Vilos G, Allaire C, Laberge P-Y, Leyland N. The management of uterine leiomyomas. *J Obstet Gynaecol Can*. 2015;37:157-178.
51. Pérez-López F, Ornat L, Ceausub I, et al. EMAS position statement: management of uterine fibroids. *Maturitas*. 2014;79:106-116.
52. Abbott JA. Adenomyosis and abnormal uterine bleeding (AUB-A): pathogenesis, diagnosis, and management. *Best Pract Res Clin Obstet Gynaecol*. 2017;40:68-81.
53. Cockerham A. Adenomyosis: a challenge in clinical gynecology. *J Midwifery Womens Health*. 2012;57;212-220.
54. Hai N, Hou Q, Ding X, Dong X, Jin M. Ultrasound-guided transcervical radiofrequency ablation for symptomatic uterine adenomyosis. *Br J Radiol*. 2017;90:20160119.
55. Casey PM, Long ME, Marnach ML. Abnormal cervical appearance: what to do, when to worry? *Mayo Clin Proc*. 2011;86(2):147-151.
56. Kessous R, Aricha-Tamir B, Sheizaf B, et al. Clinical and microbiological characteristics of Bartholin gland abscesses. *Obstet Gynecol*. 2013; 122:794-799.

57. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2014;59(2):e10-e52.
58. International Society for the Study of Vulvovaginal Disease. ISSVD terminology and classification of vulvar dermatological disorders: clinical diagnosis. Updated January 23, 2016. Available at: https://www.issvd.org/tag/terminology/. Accessed September 28, 2017.
59. Ozdegirmenci O, Kayikcicioglu F, Haberal A. Prospective randomized study of marsupialization versus silver nitrate application in the management of Bartholin gland cysts and abscesses. *J Minim Invasiv Gynecol*. 2009;16(2):149-152.
60. Jemec GBE. Hidradenitis suppurativa. *N Engl J Med*. 2012;366:158-164.
61. von Laffert M, Hembold P, Wohlrab J, Fiedler E, Stadie V, Marsch WC. Hidradenitis suppurativa (acne inversa): early inflammatory events at terminal follicles and at interfollicular epidermis. *Exp Dermatol*. 2010;19:533-537.
62. Kamp S, Fiehn AM, Stenderup K, et al. Hidradenitis suppurativa: a disease of the absent sebaceous gland? Sebaceous gland number and volume are significantly reduced in uninvolved hair follicles from patients with hidradenitis suppurativa. *Br J Dermatol*. 2011;164:1017-1022.
63. Sartorius K, Emtestam L, Jemec GB, Lapins J. Objective scoring of hidradenitis suppurativa reflecting the role of tobacco and obesity. *Br J Dermatol*. 2009;161:831-839.
64. Thorstensen KA, Birenbaum DL. Recognition and management of vulvar dermatologic conditions: lichen sclerosus, lichen planus, and lichen simplex chronicus. *J Midwifery Womens Health*. 2012;57:260-275.
65. Van Hoover C, Rademayer C, Farley CL. Body piercing: motivations and implications for health. *J Midwifery Womens Health*. 2017;62:521-530.
66. Young C, Armstrong ML, Roberts AE, Mello I, Angel E. A triad of evidence for care of women with genital piercings. *J Am Acad Nurse Pract*. 2010;22(2):70-80.
67. Holbrook J, Minocha J, Lauman A. Body piercing: complications and prevention of health risks. *Am J Clin Dermatol*. 2012;13(1):1-17.
68. Bornstein J, Goldstein A, Stockdale C, et al., on behalf of Consensus Vulvar Pain Terminology Committee of International Society for the Study of Vulvovaginal Disease (ISSVD), International Society for the Study of Women's Sexual Health (ISSWSH), and International Pelvic Pain Society (IPPS). 2015 ISSVD, ISSWSH and IPPS consensus terminology and classification of persistent vulvar pain and vulvodynia. *Obstet Gynecol*. 2016;127:745-751.
69. Haefner HK, Collins ME, Davis GD, et al. The vulvodynia guideline. *J Low Genit Tract Dis*. 2005;9:40-51.
70. Pukall CF, Goldstein AT, Bergeron S, et al. Vulvodynia: definition, prevalence, impact, and pathophysiological factors. *J Sex Med*. 2016;13:291-304.
71. Cox K, Neville C. Assessment and management options for women with vulvodynia. *J Midwifery Womens Health*. 2012;57:231-240.
72. Bonham A. Vulvar vestibulodynia: strategies to meet the challenge. *Obstet Gynecol Surv*. 2015;70:274-278.
73. Nagandla K, Sivalingam N. Vulvodynia: integrating current knowledge into clinical practice. *Obstet Gynaecol*. 2014;16:259-267. Available at: http://onlinelibrary.wiley.com/doi/10.1111/tog.12130/epdf. Accessed November 26, 2017.
74. American College of Obstetricians and Gynecologists. Committee Opinion No. 673: persistent vulvar pain. *Obstet Gynecol*. 2016;128:e78-e84.
75. Goldstein AT, Pukall CF, Brown C, Bergeron S, Stein A, Kellogg-Spadt S. Vulvodynia: assessment and treatment. *J Sex Med*. 2016;13:572-590.
76. Haylen BT, de Ridder D, Freeman RM, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. *Neurourol Urodyn*. 2010;29(1):4-20.
77. Patel DA, Xu X, Thomason AD, Ransom SB, Ivy JS, DeLancey JO. Childbirth and pelvic floor dysfunction: an epidemiologic approach to the assessment of prevention opportunities at delivery. *Am J Obstet Gynecol*. 2006;195:23-28.
78. Tinelli A, Malvasi A, Rahimi S, et al. Age-related pelvic floor modifications and prolapse risk factors in postmenopausal women. *Menopause*. 2010;17:204.
79. Swift S, Woodman P, O'Boyle A, et al. Pelvic Organ Support Study (POSST): the distribution, clinical definition, and epidemiologic condition of pelvic organ support defects. *Am J Obstet Gynecol*. 2005;192:795.
80. Giri A, Hartmann KE, Hellwege JN, Velez Edwards DR, Edwards TL. Obesity and pelvic organ prolapse: a systematic review and meta-analysis of observational studies. *Am J Obstet Gynecol*. 2017;217(1):11-26.
81. O'Dell K, Morse AN. It's not all about birth: biomechanics applied to pelvic organ prolapse prevention. *J Midwifery Womens Health*. 2008;53:28-36.
82. Persu C, Chapple C, Cauni V, Gutue S, Geavlete P. Pelvic Organ Prolapse Quantification system (POP–Q): a new era in pelvic prolapse staging. *J Med Life*. 2011;4:75-81.
83. Hagen S, Stark D. Conservative prevention and management of pelvic organ prolapse in women. *Cochrane Database Syst Rev*. 2011;12:CD003882. doi:10.1002/14651858.CD003882.pub4.
84. Hagen S, Stark D, Glazener C, et al. Individualised pelvic floor muscle training in women with pelvic organ prolapse (POPPY): a multicentre randomised controlled trial. *Lancet*. 2014;383(9919):796-806.

85. Bugge C, Adams E, Gopinath D, Reid F. Pessaries (mechanical devices) for pelvic organ prolapse in women. *Cochrane Database Syst Rev.* 2013;2:CD004010. doi:10.1002/14651858.CD004010.pub3.
86. Magali R, Schulz J, Harvey, M-A. Technical update on pessary use. *J Obstet Gynaecol Can*. 2013;35(7 e-suppl):S1-S11.
87. Maher C, Feiner B, Baessler K, Christmann-Schmid C, Haya N, Marjoribanks J. Transvaginal mesh or grafts compared with native tissue repair for vaginal prolapse. *Cochrane Database Syst Rev.* 2016;2:CD012079. doi:10.1002/14651858.CD012079.
88. American College of Obstetricians and Gynecologists. Practice Bulletin No. 155: urinary incontinence in women. *Obstet Gynecol.* 2015;126:e66-e81.
89. Ayeleke R, Hay-Smith E, Omar M. Pelvic floor muscle training added to another active treatment versus the same active treatment alone for urinary incontinence in women. *Cochrane Database Syst Rev.* 2015;11:CD010551. doi:10.1002/14651858.CD010551.pub3.
90. Bradley C, Rahn D, Nygaard I, et al. The Questionnaire for Urinary Incontinence Diagnosis (QUID): validity and responsiveness to change in women undergoing non-surgical therapies for treatment of stress predominant urinary incontinence. *Neurourol Urodyn*. 2010;29:727-734.
91. Farrell S, Bent A, Amir-Khalkhali B, et al. Women's ability to assess their urinary incontinence type using the QUID as an educational tool. *Int Urogynecol J*. 2013;24:759-762.
92. Imamura M, Williams K, Wells M, McGrother C. Lifestyle interventions for the treatment of urinary incontinence in adults. *Cochrane Database Syst Rev.* 2015;12:CD003505. doi:10.1002/14651858.CD003505.pub5.
93. Dumoulin C, Hay-Smith J. Pelvic floor muscle training versus no treatment, or inactive control treatments, for urinary incontinence in women. *Cochrane Database Syst Rev.* 2014;5:CD005654. doi:10.1002/14651858.CD005654.pub3.
94. Sanford M. Mirabegron: a review of its use in patients with overactive bladder syndrome. *Drugs.* 2013;73:1213-1225.
95. Rai BP, Cody JD, Alhasso A, Stewart L. Anticholinergic drugs versus non-drug active therapies for non-neurogenic overactive bladder syndrome in adults. *Cochrane Database Syst Rev.* 2012;12:CD003193. doi:10.1002/14651858.CD003193.pub4.
96. O'Dell KK, Labin LC. Common problems of urination in nonpregnant women: causes, current management and prevention strategies. *J Midwifery Womens Health*. 2006;51:159-173.
97. Lipp A, Shaw C, Glavind K. Mechanical devices for urinary incontinence in women. *Cochrane Database Syst Rev.* 2014;12:CD001756. doi:10.1002/14651858.CD001756.pub6.
98. Practice Committee of the American Society for Reproductive Medicine. Definitions of infertility and recurrent pregnancy loss: a committee opinion. *Fertil Steril*. 2013;99:63.
99. Koroma L, Stewart L. Infertility: evaluation and initial management. *J Midwifery Womens Health*. 2012;57:614-621.
100. Koroma L. Infertility. In: Schuiling K, Likis F, eds. *Women's Gynecologic Health*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017:419-441.
101. Lindsay TJ, Vitrikas KR. Evaluation and treatment of infertility. *Am Fam Physician*. 2015;91(5):308-314.
102. Pandian Z, Gibreel A, Bhattacharya S. In vitro fertilization for unexplained subfertility. *Cochrane Database Syst Rev.* 2015;11:CD003357. doi:10.1002/14651858.CD003357.pub4.
103. Byrne M, Doherty S, Fridlund BGA, et al. Sexual counseling for sexual problems in patients with cardiovascular disease. *Cochrane Database Syst Rev.* 2016;2:CD010988. doi:10.1002/14651858.CD010988.pub2.
104. Vecchio M, Navaneethan SD, Johnson DW, et al. Interventions for treating sexual dysfunction in patients with chronic kidney disease. *Cochrane Database Syst Rev.* 2010;12:CD007747. doi:10.1002/14651858.CD007747.pub2.
105. Levack WMM, Poot B, Weatherall M, Travers J. Interventions for sexual dysfunction in people with chronic obstructive pulmonary disease (COPD). *Cochrane Database Syst Rev.* 2015;9:CD011442. doi:10.1002/14651858.CD011442.pub2.
106. Candy B, Jones L, Vickerstaff V, Tookman A, King M. Interventions for sexual dysfunction following treatments for cancer in women *Cochrane Database Syst Rev.* 2016;2:CD005540. doi:10.1002/14651858.CD005540.pub3.
107. Barbera L, Zwaal C, Elterman D, et al. Interventions to Address Sexual Problems in People with Cancer Guideline Development Group: interventions to address sexual problems in people with cancer. *Curr Oncol*. 2017;24(3):192-200.
108. Fairbanks F, Abdo CH, Baracat EC, Podgaec S. Endometriosis doubles the risk of sexual dysfunction: a cross-sectional study in a large amount of patients. *Gynecol Endocrinol*. 2017;28:1-4.
109. Mendonça CR, Arruda JT, Noll M, Campoli PMO, Amaral WND. Sexual dysfunction in infertile women: a systematic review and meta-analysis. *Eur J Obstet Gynecol Reprod Biol*. 2017;215:153-163.
110. Jayne CJ, Heard MJ, Zubair S, Johnson DL. New developments in the treatment of hypoactive sexual desire disorder: a focus on flibanserin. *Int J Womens Health*. 2017;9:171-178.
111. Nastri CO, Lara LA, Ferriani RA, Rosa-e-Silva ACJS, Figueiredo JBP, Martins WP. Hormone therapy for sexual function in perimenopausal and postmenopausal women. *Cochrane Database Syst Rev.* 2013;6:CD009672. doi:10.1002/14651858.CD009672.pub2.
112. Parish SJ, Hahn SR. Hypoactive sexual desire disorder: a review of epidemiology, biopsychology, diagnosis, and treatment. *Sex Med Rev*. 2016;4(2):103-120.

113. Schmidt HM, Hagen M, Kriston L, Soares-Weiser K, Maayan N, Berner MM. Management of sexual dysfunction due to antipsychotic drug therapy. *Cochrane Database Syst Rev.* 2012;11:CD003546. doi:10.1002/14651858.CD003546.pub3.
114. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2017. *CA Cancer J Clin*. 2017;67:7.
115. Kehoe SM, Miller DS, Schorge JO. Endometrial cancer. In: Hoffman BL, Schorge JO, Bradshaw KD, Halvorson LM, Schaffer JI, Corton MM, eds. *Williams Gynecology*. 3rd ed. New York, NY: McGraw-Hill; 2016:702-721.
116. American College of Obstetricians and Gynecologists. Practice Bulletin No. 149: endometrial cancer. *Obstet Gynecol.* 2015;125:1006-1026.
117. Monchek R, Wiedaseck S. Gestational trophoblastic disease: an overview. *J Midwifery Womens Health*. 2012;57:255-259.
118. Benson C, Miah AB. Uterine sarcoma: current perspectives. *Int J Womens Health*. 2017;9:597-606.
119. Pearce CL, Stram DO, Ness RB, et al. Population distribution of lifetime risk of ovarian cancer in the United States. *Can Epidem Biomark Prev*. 2015;24(4):671-676.
120. Salehi F, Dunfield L, Phillips KP, Krewski D, Vanderhyden BC. Risk factors for ovarian cancer: an overview with emphasis on hormonal factors. *J Toxicol Environ Health B Crit Rev.* 2008;11:301-321.
121. Sueblinvong T, Carney ME. Current understanding of risk factors for ovarian cancer. *Curr Treat Options Oncol.* 2009;10:67-81.
122. Milne RL, Antoniou AC. Modifiers of breast and ovarian cancer risks for *BRCA1* and *BRCA2* mutation carriers. *Endocrine-Related Cancer.* 2016;23(10):T69-T84.
123. Iversen L, Sivasubramaniam S, Lee AJ, Fielding S, Hannaford PC. Lifetime cancer risk and combined oral contraceptives: the Royal College of General Practitioners' Oral Contraception Study. *Am J Obstet Gynecol.* 2017;216:580.e1-580.e9.
124. American College of Obstetricians and Gynecologists. Practice Bulletin No. 182: hereditary breast and ovarian cancer syndrome. *Obstet Gynecol.* 2017;130:e110-e116.
125. Smith EC. An overview of hereditary breast and ovarian cancer syndrome. *J Midwifery Womens Health*. 2012;57:577-584.
126. Moyer VA, on behalf of U.S. Preventive Services Task Force. Risk assessment, genetic counseling, and genetic testing for BRCA-related cancer in women: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2014;160:271-281.
127. National Comprehensive Cancer Network. *Genetic/Familial High Risk Assessment: Breast and Ovarian*. Version 2.2017. NCCN Clinical Practice Guidelines in Oncology. Fort Washington, PA: NCCN; 2016.
128. Bankhead CR, Kehoe ST, Austoker J. Symptoms associated with diagnosis of ovarian cancer: a systematic review. *BJOG*. 2005;112:857-865.
129. Centers for Disease Control and Prevention. Vaginal and vulvar cancers. December 2016. Available at: https://www.cdc.gov/cancer/vagvulv/pdf/vagvulv_facts.pdf. Accessed May 20, 2017.
130. Howlader N, Noone AM, Krapcho M, et al., eds. *SEER Cancer Statistics Review, 1975–2014*. Bethesda, MD: National Cancer Institute; June 28, 2017. Available at: https://seer.cancer.gov/csr/1975_2014/. Accessed December 4, 2017.
131. American Cancer Society. Vaginal cancer: early detection, diagnosis, and staging. February 2016. Available at: https://www.cancer.org/cancer/vaginal-cancer/detection-diagnosis-staging/survival-rates.html. Accessed May 20, 2017.
132. Lee L, Garland SM. Human papillomavirus vaccination: the population impact. *F1000Research*. 2017;6:866. doi:10.12688/f1000research.10691.1.
133. Goodman A, Schorge J, Greene M. The long-term effects of in utero exposures: the DES story. *N Engl J Med*. 2011;364:2083-2084.
134. Venetis C, Papadopoulos S, Campo R, Gordts S, Tarlatzis B, Grimbizis G. Clinical implications of congenital uterine anomalies: a meta-analysis of comparative studies. *Reprod Biomed Online*. 2014;29:665-683.
135. Dreisler E, Stampe Sørensen S. Müllerian duct anomalies diagnosed by saline contrast sonohysterography: prevalence in a general population. *Fertil Steril*. 2014;102:525-529.
136. American Fertility Society. The American Fertility Society classifications of adnexal adhesions, distal tubal occlusion, tubal occlusion secondary to tubal ligation, tubal pregnancies, Müllerian anomalies and intrauterine adhesions. *Fertil Steril*. 1988;49:944-955.
137. Reed C, Fenton S. Exposure to diethylstilbestrol during sensitive life stages: a legacy of heritable health effects. *Birth Defects Res C: Embryo Today.* 2013;99:134-146.
138. Centers for Disease Control and Prevention. Information to identify and manage DES patients. February 2017. Available at: https://www.cdc.gov/DES/hcp/information/daughters/risks_daughters.html#cca. Accessed May 20, 2017.

APPENDIX

12A

Fitting a Pessary

KATHRYN OSBORNE

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Pelvic organ prolapse (POP) occurs with the descent of one or more of the pelvic organs and resulting herniation of the vaginal wall. This herniation can occur in the anterior vaginal wall (cystocele and urethrocele), the vaginal apex (uterine prolapse), or the posterior vaginal wall (rectocele or enterocele).[1] The most common type of POP is cystocele, which results from descent of the bladder.[1] The peak prevalence of POP is found in women age 60–69 years.[2] An estimated 25% to 50% of women in the United States report some degree of POP.[2,3]

The symptoms of POP differ according to the type of prolapse, but often result in reduced health-related quality of life as a result of bulging in the vagina, sexual dysfunction, and varying degrees of urinary and fecal incontinence.[1] Current recommendations are to consider treatment for women with discomfort associated with vaginal pressure or bulging, sexual dysfunction, or alterations in urinary or defecatory function, and to offer vaginal pessary as a first-line treatment alternative to surgery.[4] Evaluation and management of pelvic organ prolapse is described in the *Gynecologic Disorders* chapter; fitting a ring pessary is described in this appendix.

Types of Pessaries

After a woman and a midwife have engaged in shared decision making and determined that the best approach to treating the woman's pelvic organ prolapse is with a pessary, the first step is to determine which type of pessary to use. Two types of pessaries are available: *space-occupying pessaries* (such as the Gellhorn or cube) and *support pessaries* (such as the ring or ring with support) (**Figure 12A-1).** The choice of pessary type has traditionally been made based on the degree of prolapse; support pessaries have been used to treat Stage I and Stage II prolapse, whereas Stage III and IV prolapse have typically been treated with space-occupying pessaries.[1,5] However, recent research has identified that women with Stage III and IV prolapse can achieve adequate symptom relief with pelvic floor exercises in conjunction with

support pessaries, which are easier for women to remove and replace, and which are associated with less discharge and irritation compared to space-occupying pessaries.[1,5,6] The most commonly used pessary is the ring pessary with support, which is also associated with high rates of symptom relief.[1,5,6]

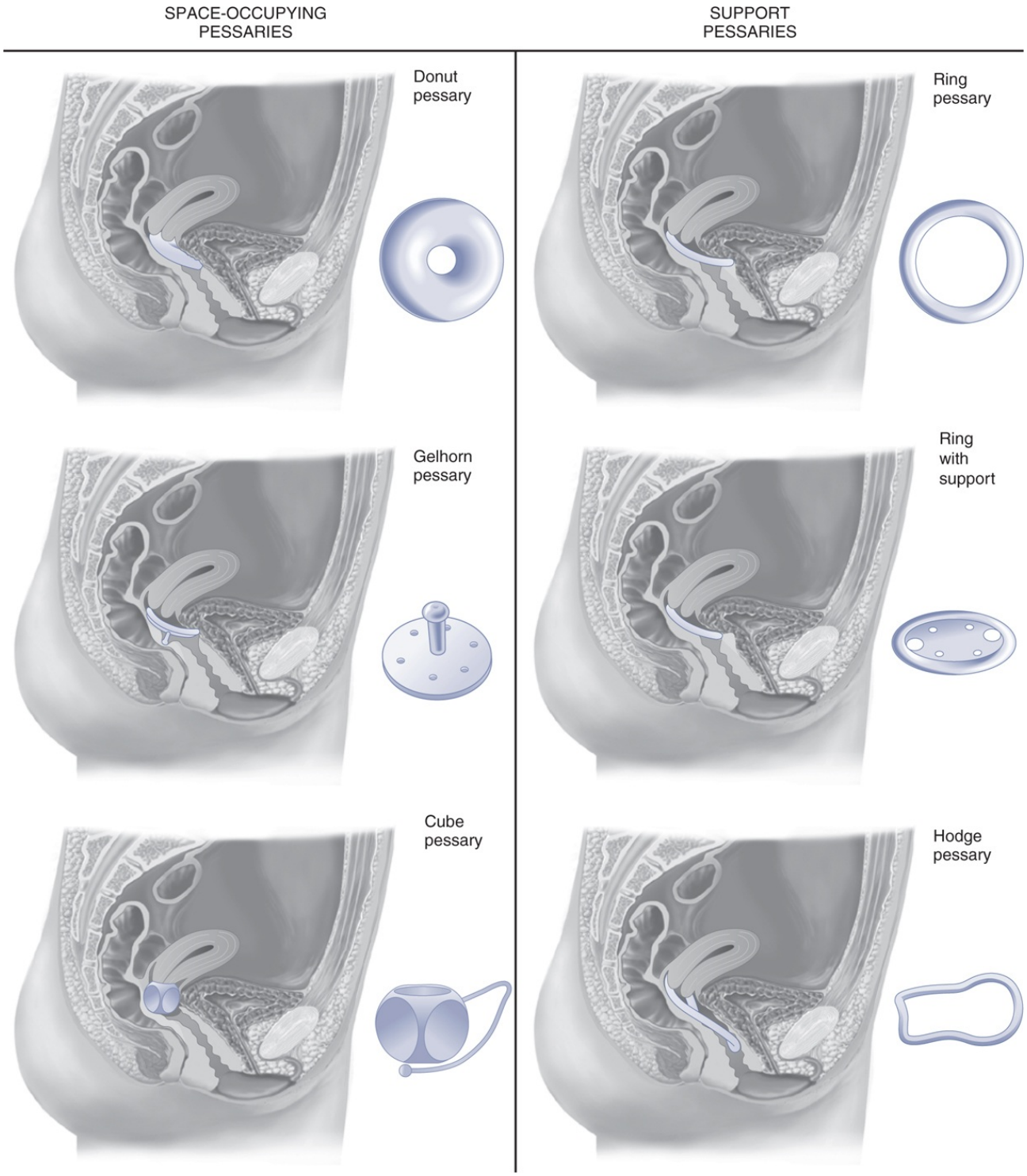


Figure 12A-1 Types of pessaries.

Fitting the Pessary

1. Obtain the necessary supplies: nonsterile gloves, set of fitting rings, and lubricant.
2. Offer the woman an opportunity to empty her bladder.
3. Assist the woman onto an examination table and into the same position used for a pelvic examination; provide draping for privacy and comfort.
4. Inform the woman that you will be conducting a pelvic examination to obtain the internal measurements necessary to fit the pessary, and that she should let you know if at any time she becomes uncomfortable. After obtaining her consent, proceed with the examination.
5. Don gloves. Insert a middle finger and forefinger into the vagina. Place your middle finger behind the cervix in the posterior fornix and place your index finger against the posterior pubic notch. You will use the distance between your index and middle fingers as the initial measurement of the distance between the posterior fornix and the symphysis pubis to size the pessary.
6. Remove your hand and choose the fitting ring with the diameter that most closely approximates the distance between your middle and index fingers.
7. Fold the ring in half and apply lubricant to the end that will be inserted; insert the ring into the vagina so that the ring rests behind the cervix posteriorly and behind the pubic notch anteriorly.
8. Sweep your finger around the diameter of the ring to ensure that the ring is fully unfolded and comfortably in place without undue pressure against the posterior, anterior, or lateral vaginal walls. Ask the woman to bear down and check for movement of the ring as she bears down. If the ring slides over the cervix or is visible at the introitus, repeat this test with a larger ring. A smaller ring will be necessary if the pessary cannot unfold completely or if you are unable to fit your finger between the vaginal wall and the ring; use the largest pessary that retains a comfortable fit.
9. Ask the woman to get up and walk around for several minutes, encourage her to void or defecate if possible. Remove gloves and perform hand hygiene while the woman is moving around.
10. When she returns, assist the woman onto the examination table and reglove. Reassess the placement of the pessary. If it remained in place, remove and discard your gloves before teaching about self-care. Assist the woman to a sitting position.
11. Explain to the woman that she may remove and clean the pessary as often as she feels it is necessary (daily, weekly, or monthly). Also explain that you will offer her an opportunity to practice removing and replacing the pessary before she leaves the clinic.
12. Using a diagram, show the woman how to insert her index finger to locate the pessary (behind the pubic notch) and remove it. Step out of the room and provide her with a private space to remove the pessary.
13. When you return to the room, ask if she felt comfortable removing the pessary and respond to any questions or concerns she may have.
14. Demonstrate how to fold the pessary in half, apply lubricant, and (using a diagram) demonstrate how the pessary is inserted. Explain that placing one foot on the examining table's step stool may make insertion (and removal) easier. Ask if she has any questions

before you leave the room to provide a private space to practice insertion of the pessary.

15. Upon returning to the room, apply gloves and check to see that the fitting ring has been properly replaced. Remove the fitting ring.
16. Remove your gloves, perform hand hygiene, and assist the woman to a sitting position.
17. Provide the woman with cleansing tissue and exit the room so that she may get dressed for the remainder of the visit.
18. Return to the room to discuss any questions or concerns the woman has and review ongoing follow-up.

Ongoing Follow-Up and Management

Women should return to the clinic 1–2 weeks after they begin wearing the pessary to evaluate symptom improvement and satisfaction with the pessary. Most women can safely perform self-care and management of a vaginal pessary, removing and cleaning the pessary with soap and water as often as desired.[1,5,7] Those who perform self-care should return to the clinic every 6–12 months for an examination and assessment of the vaginal wall.[1,5] Women who do not engage in self-care of the pessary should return to the clinic every 3 months to have the pessary removed and cleaned and for an examination of the vaginal wall.[1]

The most common complications associated with pessary use are a malodorous vaginal discharge, vaginal irritation and ulceration, pain, and vaginal bleeding.[1] Most of these complications appear to occur less frequently in women who engage in at least monthly removal and self-care of the pessary.[7] Any woman with an intact uterus who reports vaginal bleeding should be referred for further evaluation. In addition, women who do not achieve adequate symptom relief with a pessary should be referred to a gynecologic or urologic specialist for more extensive evaluation and a discussion of treatment options.

References

1. Iglesia CB, Smithling KR. Pelvic organ prolapse. *Am Fam Physician*. 2017;96(3):179-185.
2. Wu JM, Vaughan CP, Goode PS, et al. Prevalence and trends of symptomatic pelvic floor disorders in U.S. women. *Obstet Gynecol*. 2014;123(1):141-148.
3. Barber MD, Maher C. Epidemiology and outcome assessment of pelvic organ prolapse. *Int Urogynecol J*. 2013;24:1783-1790.
4. American College of Obstetricians and Gynecologists. ACOG practice bulletin summary: pelvic organ prolapse. *Obstet Gynecol*. 2017;130(5):1170-1172.
5. Ding J, Chen C, Song X, Zhang L, Deng M, Zhu L. Changes in prolapse and urinary symptoms after successful fitting of a ring pessary with support in women with advanced pelvic organ prolapse: a prospective study. *Urology*. 2016;87(C):70-75.
6. Cheung RYK, Lee JHS, Lee LL, Chung TKH, Chan SSC. Vaginal pessary in women with symptomatic pelvic organ prolapse: a randomized controlled trial. *Obstet Gynecol*. 2016;128(1):73-80.
7. Daneel L, West N, Moore KH. Does monthly self-removal of vaginal ring pessaries for stress urinary incontinence/prolapse reduce complication rates? A 5 year audit. *Austral NZ Continence J*. 2016;22(4):105-106.

CHAPTER

13

Breast Conditions

JOYCE L. KING

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Introduction

Culturally, the female breast is often intertwined with the concept of womanhood. Even though the primary function of breasts is lactation, breasts are frequently considered to be erotic and the size and shape subject to sexual description. Conditions of the breast encompass a broad range of pathology, from benign disorders such as fibroadenomas to life-threatening malignancies such as breast cancer. Injury or loss of a breast can be emotionally devastating for a woman.

This chapter reviews normal development of the breast, current recommendations for breast examination, common benign breast disorders, and an overview of breast cancer.

Breast Development

The anatomy and embryonic development of the breast is discussed in detail in the *Anatomy and Physiology of the Female Reproductive System* chapter. The process of development of secondary sexual characteristics takes place in an orderly and predictable sequence. Tanner's classification of sexual maturity is a scale used to evaluate breast and pubic hair growth as a way of assessing normal growth and development. Often the first sign of puberty is thelarche, or the onset of breast development. On average, breast development is initiated at 10 years of age in Caucasian females and 8.9 years of age in African American females.[1]

Both estrogen and progesterone influence the development of the breast, with estrogen stimulating the ductal portion of the glandular system and progesterone stimulating the alveolar or milk-producing components of the system.[2] These two hormones are not sufficient to achieve optimal growth and development; instead, stimulation of insulin, cortisol, thyroxine, prolactin, and insulin-like growth factor is also required. It is not uncommon for the breasts to develop asymmetrically, but usually by the end of puberty the breasts are approximately the same size in most women—although as many as 25% of women have persistent visible breast asymmetry. Significant asymmetry is correctable by surgery; hormone therapy is ineffective for treatment of this condition. The complete development of the alveolus into a mature milk-producing gland is accomplished with the increase in estrogen and progesterone levels that occur during pregnancy.[2]

Breast tissue changes with aging. The adolescent breast has increased density relative to the older breast, as the former consists predominantly of glandular tissue. The breast also undergoes cyclic changes in nonpregnant women due to the influence of estrogen and progesterone. These cyclic changes include increased size of breasts, fluid secretion, and premenstrual tenderness. Breast changes that occur during pregnancy and lactation (i.e., mammogenesis and lactogenesis) are discussed in the *Anatomy and Physiology of Pregnancy* and *Breastfeeding and the Mother–Newborn Dyad* chapters, respectively. In women who breastfeed, glandular tissue regresses and some tissue remodeling occurs once lactation ceases. As women age, breasts become less dense and most women progressively acquire more fat and fibrous connection tissues that gradually replace the glandular tissue.[2]

Gynecomastia, or breast enlargement in males, can be physiologic, may be associated with specific diseases (e.g., Klinefelter syndrome, hyperthyroidism, liver failure), may be a side effect of certain medications (e.g., spironolactone, digoxin), or may result from the decrease in testosterone production as men age. Fifty percent of adolescent males will experience physiologic gynecomastia during early puberty, which usually resolves within 6 months to 2 years after onset. If symptoms persist after 2 years or continue past 17 years of age, further evaluation is needed.[3]

Evaluation of Breast Symptoms

A systematic approach to breast examination is important to evaluate reports of breast concerns. Even though several benign conditions exist, because of the prevalence and fear of breast cancer, the diagnosis of breast cancer should be considered anytime a woman reports any unusual breast symptoms. For providers of care for women, a missed diagnosis of breast cancer can lead to a malpractice claim.

History

The history of presenting symptoms should include the items noted in **Table 13-1**. The healthcare professional should document these components in the record of the woman's visit as positive or negative as appropriate.

Table 13-1 Breast Symptoms That Require Evaluation and Documentation in Health Record

Change in general appearance
Changes over time, especially in relation to menstrual cycle
Skin changes such as thickening of skin (*peau d'orange*), which may indicate inflammatory cancer
Nipple inversion that is a new finding
Nipple discharge: Bilateral or unilateral? Color, timing, frequency, and spontaneous or only if elicited?
Cyclic or noncyclic pain, also known as mastalgia
Masses, including location and changes in size or tenderness with menstrual cycle

The purpose of history taking when evaluating a woman with breast symptoms is both to learn about the symptoms and to assess her risk factors for breast disorders, especially cancer.[4] Although a number of factors have been identified that increase the risk for breast cancer, the majority of women who develop breast cancer (85%) have no identifiable risk other than their gender and their age—both nonmodifiable factors.[3] When evaluating a woman with breast symptoms, factors that increase or decrease her risk for breast cancer should be identified and documented in her health record. **Table 13-2** lists the common risk factors for breast cancer to be reviewed when taking a history.[2,5,6] Factors that are known to be protective against breast cancer include breastfeeding, physical activity, and possibly a Mediterranean diet although it is not clear exactly which components of this diet are the most beneficial.

Table 13-2 Factors That Increase the Risk of Breast Cancer

Risk Factor	Description
Age, gender, race	Advancing age, female, white
Family history	First-degree relative with breast cancer Jewish ancestry Male breast cancer

	First-degree relative with ovarian cancer
	Multiple relatives with cancer, especially hereditary breast and ovarian syndrome–associated cancer (e.g., prostate, pancreatic)
	Known inherited genetic mutations
Health history	Personal history of invasive breast cancer, ductal carcinoma in situ or lobular carcinoma in situ
	Biopsy-confirmed proliferative breast lesions with atypia
	High-dose radiation to chest, usually between years 10 and 30
	Never breastfed
	Previous cancer of the endometrium, ovary, or colon
	High bone density if postmenopausal
	Known inherited genetic mutations
Reproductive history	Early menarche, before age 12 years
	Nulliparity
	Older than 30 years when first gave birth
	Onset of menopause after age 55 years
	Personal or in utero exposure to diethyl stilbestrol (DES)
Medications	Current use of oral contraceptives that contain both estrogen and progesterone
	Hormone therapy with estrogen and progesterone for 5–9 years of use[a]
Lifestyle factors	Alcohol consumption 2–5 drinks per day
	Night shift work
	Physical inactivity
	Smoking
Physical examination	High breast density if postmenopausal
	Postmenopausal obesity

[a] The increased risk of breast cancer is no longer present if there has been more than 5 years since discontinuation of menopausal hormone therapy.

Based on Fritz MA, Speroff L. The breast. In: Fritz MA, Speroff L, eds. *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2011:621-673[2]; U.S. Preventive Services Task Force. Screening for breast cancer: U.S. Preventive Services Task Force Recommendation statement. *Ann Intern Med.* 2016;164(4):279-296[5]; Hoffman B, Schorge J, Bradshaw K, Halvorson LM, Schaffer JL, Corton MM. Breast diseases. In: Hoffman B, Schorge J, Bradshaw K, Halvorson LM, Schaffer JL, Corton MM, eds. *Williams Gynecology*. 3rd ed. New York, NY: McGraw-Hill; 2016:286.[6]

Physical Examination

The clinical examination includes examination of the neck, breasts, chest wall, and axillae. The technique for examination of the breast is detailed in the *Breast Examination* appendix in the *Introduction to the Care of Women* chapter. A full physical examination may also be indicated based on the individual's needs.

Breast findings characteristic of nonmalignant breast disorders include painful or even tender, firm, mobile, well-defined masses that may fluctuate in size and tenderness with menstrual cycle changes. A breast mass is considered a dominant mass if it is a three-dimensional lesion that cannot be replicated in the same location on the other breast. The classic sign of a breast lesion suspicious for breast cancer is a hard, rocky, immobile mass with irregular or ill-defined borders.

Breast masses are to be described as dominant or nondominant and measured in centimeters when possible when noted in the woman's health record. The location, consistency, symmetry, tenderness, and mobility are noted, as are any skin changes. The location may be recorded as distance in centimeters from the areola and by comparing the breast to a clock so that the "o'clock" location can be noted (e.g., a 2 × 3 cm firm, discrete, smooth, mobile, nontender mass 3 cm from the areola at the 4 o'clock position). Negative findings also are to be recorded in a woman's health record. Physical examination cannot distinguish well between malignant and nonmalignant masses, so any palpable mass should be evaluated by the use of the breast assessment modalities discussed next.

Breast Assessment Modalities

Several instruments are available today to assess breast health. Some of these modalities are used for screening women for breast cancers, whereas others are employed for diagnostic indications. **Table 13-3** summarizes these methods, which are discussed in more detail within the subsequent sections on specific disorders, because certain methods are more appropriate than others for some women.

Table 13-3 Common Breast Assessment Methods

Method	Description	Comments
Breast awareness/breast self-examination	A woman's knowledge about her own breasts; may or may not include breast self-examination	General screening for any disorder, most often breast masses Not limited to screening for breast cancer; may discover other disorders Many studies combine with clinical breast examination for research purposes
Clinical breast examination	Examination by a provider as outlined in the *Breast Examination* appendix in the *Introduction to the Care of Women* chapter	An essential component of the physical examination Not limited to screening for breast cancer; may discover other disorders Many studies combine with breast self-examination for research purposes
Mammogram[a]	Standard mammogram uses X rays to assess abnormalities of the breast, including masses, density, and changes in architecture	Regular mammograms are the gold-standard screening technique for breast cancer Also used as part of the diagnostic evaluation indicated when breast masses are palpated
	Digital breast tomosynthesis: a newer type of mammography that provides three-dimensional views	Although FDA approved, in most areas reserved for women at high risk for breast cancer because of cost and specificity issues
Ultrasound	Sonographic assessment of the breast	May be used for screening women with dense breasts (often younger women) at high risk for breast cancer Indicated for women with palpable masses, especially those that are not identifiable on mammograms

Magnetic resonance imaging (MRI)	Method using radio waves and magnetic field to assess the breasts	Primarily used in conjunction with mammography or ultrasound to evaluate extent of cancer of the breast Can be used to screen women at high risk for breast cancer Due to the lack of ionizing radiation, MRI is the preferred method to determine whether silicon implants have ruptured
Biopsy of breast mass	Fine-needle biopsy: a common outpatient procedure to provide cells for cytological study and identification of malignancy Core biopsy, using a larger needle, and open biopsy may be indicated based on size of the mass	Performed by a physician under guided ultrasound or mammogram
	Biopsy at the time of surgery (e.g., lumpectomy, mastectomy)	Most commonly combined with biopsy of nodes Provides staging of cancer

Abbreviation: FDA, U.S. Food and Drug Administration.

[a] Mammography is not infallible; a palpable mass with a normal mammogram indicates the need for referral and additional diagnostic evaluation.

Several algorithms have been developed for evaluation of dominant breast masses.[4,7,8] Some recommend a fine-needle biopsy as the first step, whereas others recommend ultrasound or mammography first. Variations depend on local radiology, pathology, and surgical resources; the woman's age; and her preferences. Ultrasound is generally recommended for women younger than 30 years and diagnostic mammography for women older than 30 years, because the increased density of breast tissue in younger women tends to obscure abnormal findings from mammography.

Table 13-4 lists components that could be included in a note for consultation or transfer of care to a specialist in the area. In this example, the woman has a breast mass.

Table 13-4	Sample Critical Elements for a Consultation or Transfer Note for a Woman with a Breast Mass
Risk factors for breast cancer, including genetics, family history, and personal past history	
Current and past use of hormone therapy, including hormonal contraception	
Discovery of breast mass: by breast self-examination and/or clinical breast examination	
Description of breast mass: location, size, shape, mobility, tenderness, and consistency	
Any breast, nipple, and/or skin changes	
Care provided before transfer: procedures and results; laboratory results or pending status, including pregnancy test, mammogram, and/or ultrasound	
Assessment/diagnosis of the situation	
Summary of rationale for transfer and request for information about the treatment plan for follow-up	

Benign Breast Disorders

Benign breast disorders are a common diagnosis in women's health care and encompass all nonmalignant conditions of the breast, including breast pain (i.e., mastalgia), nipple discharge, and benign breast tumors. Benign breast disorders often are classified as nonproliferative, proliferative without atypia, or atypical hyperplasias. The risk of malignancy is related to the classification. Nonproliferative disorders, which include fibrocystic changes and breast cysts, are not associated with a risk of malignancy. Proliferative without atypia disorders, which include intraductal papillomas, are associated with a modest increase in risk for malignancy. Atypical ductal hyperplasia and atypical lobular hyperplasia are associated with a significant increased risk for malignancy.[7]

Mastalgia

Mastalgia (breast pain) can be either cyclic or noncyclic. Cyclic breast pain generally occurs bilaterally during the luteal phase of the menstrual cycle and resolves after the onset of menses. It is often described as sharp, shooting, or deep aching and throbbing pain. Noncyclic breast pain may be caused by mastitis, cysts, tumors, history of breast surgery, or medications, or it may be idiopathic. Noncyclic mastalgia tends to be localized, subareolar, or medial, and is characterized as tender, burning, stabbing, pulling, or pinching.

Approximately 15% of women with mastalgia require some pain-relieving therapy. Well-fitting bras may relieve some degree of cyclical and noncyclical mastalgia. Flaxseed and Vitex agnus have been evaluated for treatment of women with mastalgia with some positive results.[9] A systematic review suggested that nonsteroidal anti-inflammatory drugs (NSAIDs) were the best first-line treatment for women with mild mastalgia. Vitamin E, 600–3000 units/day, has been associated with improvement with minimal side effects in small studies.[10]

Physician consultation may be indicated for women whose pain is not controlled with NSAIDs. For women with more severe mastalgia, danazol (Danocrine), tamoxifen (Nolvadex), toremifene (Fareston), gonadotropin analogues, and gestrinone (Dimetriose) may be effective, although all have some troublesome adverse effects. Bromocriptine (Parlodel) is no longer U.S. Food and Drug Administration (FDA) approved for the treatment of women with mastalgia due to adverse effects, although it can be used on an off-label basis for this indication. Evening primrose oil has not been found to be effective for mastalgia, and its license for sale has been withdrawn in the United Kingdom due to lack of efficacy.[11] Several randomized trials failed to show any improvements in breast pain with reduction of caffeine, so women should not be counseled to decrease caffeine for this purpose.

Danazol (Danocrine) 100–200 mg/day has been found to relieve breast pain but is associated with significant side effects such as depression, acne, and hirsutism. Low-dose tamoxifen (Nolvadex) at 10 or 20 mg/daily is effective as well, and has fewer side effects than danazol. Low-dose tamoxifen should be considered the drug of first choice if NSAIDs and nonpharmacologic approaches are not sufficient, although this agent is not FDA approved for mastalgia.[2] Tamoxifen is associated with an increased risk of endometrial cancer if taken for longer than 5 years by women who have had breast cancer. This adverse effect does not appear

to be associated with short-term use (≤ 6 months) of tamoxifen.[12]

Nipple Discharge

Nipple discharge is common in reproductive-age women and generally benign, although one retrospective study observed an 11% malignancy rate in women with unilateral single-duct nipple discharge and no abnormalities on imaging.[13] The primary differential diagnosis for nipple discharge is to distinguish benign from pathologic findings. Benign discharge is generally bilateral, multiductal, and milky or green in color, and occurs with breast manipulation.[8,13] Bilateral milky nipple discharge may occur during pregnancy and lactation and may continue for as long as 1 year after childbirth or cessation of breastfeeding. Discharge that is unilateral, clear, serous, or bloody, and occurs spontaneously is more likely to be associated with cancer, especially when it occurs in conjunction with a breast mass and in women who are older than 40 years.[13]

Galactorrhea is defined as bilateral discharge that occurs in women who have not been pregnant or lactating within the last 12 months and is not caused by breast disease. Most often galactorrhea is idiopathic, but it can be associated with prolactin-secreting pituitary adenomas, medications that inhibit dopamine (e.g., some psychotropic medications, combined oral contraceptives, metoclopramide [Reglan], phenothiazines), hypothyroidism, breast stimulation, trauma, and herpes zoster.[2,8,13] More information about pituitary tumors can be found in the *Menstrual Cycle Abnormalities* chapter.

Uniductal, bloody discharge may be associated with intraductal papilloma—a benign tumor of the lactiferous ducts that is generally managed with surgical excision. With unilateral, uniductal, spontaneous, and clear, serous, or bloody discharge, a diagnostic mammogram and ultrasound are indicated and care is usually provided by an expert in the area.[2]

Fibrocystic Changes

Fibrocystic changes (previously called fibrocystic disease) are common and are associated with hormonal stimulation. Thus, fibrocystic changes are rare in postmenopausal women. These changes may be asymptomatic or associated with pain, tenderness, and bumpy areas throughout the breast tissue that fluctuate with the menstrual cycle. Clinical findings include symmetrical nodularity, with nodularity being more prominent in the upper outer region of the breast, and consistency described as like a “bag of beans.” The fibrous tissue may feel firm or rubbery but not rock hard. The cystic portion may feel like grapes.

There is no evidence that fibrocystic changes increase the risk for developing breast cancer.[2,4,7,8] Management options include expectant watchful waiting, aspiration of large or painful cysts, and prescription of combined oral contraceptives to decrease the risk of additional fibrocystic breast changes. Minimal evidence exists to suggest that change in dietary practices, the use of vitamins (e.g., vitamin E) or herbal preparations (e.g., evening primrose), or avoidance of methylxanthines decreases the symptomology associated with fibrocystic changes.

Breast Cysts

Breast cysts are smooth, round or oval, mobile, fluid-filled masses with well-described borders that develop from terminal breast lobules. Breast cysts may be single masses or present as a cluster of multiple small cysts. They may be painful, tender, or painless. These cysts are hormonally influenced and typically appear during menstrual changes in premenopausal or perimenopausal women.

Breast cysts are classified as simple, complex, or complicated based on ultrasonographic evidence of the thickness of the cyst wall and presence of echogenic material within the cyst. Malignancy in simple and complicated cysts is rare, but this risk increases in complex cysts and in cysts that have thickened walls or thick internal septations.[8]

Simple cysts can be diagnosed via ultrasound or fine-needle biopsy that reveals nonbloody fluid and results in disappearance of the cyst. Because the risk of malignancy is very low, a simple cyst generally does not need additional evaluation or follow-up, although it can be aspirated if it is very large or painful. Some experts recommend a follow-up clinical examination or ultrasound in 2 to 4 months to document that the cyst has not changed or recurred after aspiration.

The differential diagnosis for complex and complicated cysts is more extensive and includes abscess, breast cancer, hematoma, fat necrosis, and galactocele. These cysts need a fine-needle biopsy, core biopsy, or excisional biopsy to establish the diagnosis. Care for a woman with breast cysts is best provided by a specialist.

Fibroadenomas

Fibroadenomas are breast masses that most frequently occur in adolescent and young women, although they may be found among women of any age until menopause. These masses tend to be solitary findings, with multiple fibroadenomas noted in only 10% to 15% of women with such masses.[4] On examination, the tumors are nontender, with a firm or rubbery consistency, mobile, and well circumscribed. Mammogram or ultrasound can determine whether the mass is solid or fluid filled (cystic), with the diagnosis then being confirmed through either core needle or open biopsy. Diagnosis on the basis of the clinical examination only is inadequate. If the biopsy indicates that the tumor is a fibroadenoma, it does not need to be removed. The tumor can be followed clinically and removed only if it becomes enlarged or visibly distorts the breast. If the pathology is unclear, the tumor should be surgically excised.[7,8] Fibroadenomas can increase rapidly during pregnancy or estrogen therapy and may regress after menopause. They are not associated with an increased risk of breast cancer.[2,4,7,8]

Atypical Hyperplasia and Lobular Carcinoma In Situ

Atypical hyperplasia includes atypical ductal hyperplasia and atypical lobular hyperplasia. Both are often incidental findings from a core-needle biopsy that is performed as part of the evaluation of another breast mass. Atypical hyperplasia is a pathologic diagnosis that describes abnormal cells of the breast that are associated with an increased risk of breast cancer. This condition is generally treated with surgery (e.g., wide-excision biopsy or

lumpectomy) to remove all the affected tissue. More frequent screening for breast cancer (e.g., mammograms) and strategies to reduce breast cancer risk (e.g., preventive medications such as tamoxifen [Nolvadex], raloxifene [Evista], or anastrozole [Arimidex] as found in **Table 13-5**) may be recommended as well.[7]

Table 13-5 Selected Adjuvant/Preventive Therapies for Atypical Hyperplasia

Agent Generic (Brand)	Route	Side Effects and Toxicities
Antiestrogens		
Tamoxifen (Nolvadex)	Oral	The most common side effects are hot flashes, fluid retention, vaginal discharge, nausea, vomiting, and irregular vaginal bleeding. Increased risk of thromboembolic events (e.g., deep vein thrombosis, pulmonary embolism, and stroke) also exists. Tamoxifen acts as an estrogen agonist in the uterus, increasing the risk for endometrial cancer.
Raloxifene (Evista)	Oral	Side effects are similar to those for tamoxifen, although raloxifene does not increase the risk for endometrial cancer.
Aromatase inhibitors		These drugs have a low incidence of serious short-term side effects. They are not associated with an increased risk of endometrial cancer or thromboembolism.
Anastrozole (Arimidex)	Oral	The most common side effects are hot flashes, vaginal dryness, musculoskeletal pain, and headache, but they are usually mild.
Letrozole (Femara)	Oral	The most common side effects are musculoskeletal pain and nausea.
Exemestane (Aromasin)	Oral	The most common side effects are fatigue, nausea, hot flashes, depression, and weight gain.
Monoclonal Antibody		
Trastuzumab (Herceptin)	Intravenous	The major concern with this drug is cardiac damage that can lead to ventricular dysfunction and congestive heart failure. Many individuals experience flu-like symptoms during the first infusion. Infrequently, women may have a serious hypersensitivity reaction. Trastuzumab does not cause bone marrow suppression or hair loss.

Lobular carcinoma in situ (LCIS) is an area of abnormal cell growth. LCIS is a histologic diagnosis and the disorder is associated with an increased risk for breast cancer, both in the affected breast and the contralateral breast. There are no breast masses associated with LCIS. Because LCIS is not a precursor lesion for breast cancer, complete excision is not indicated. Although the name of this condition includes “carcinoma,” LCIS is not a true breast cancer. To decrease the potential for confusion, some oncologists prefer to use the term “lobular neoplasia” to describe this finding. Increased surveillance for breast cancer and risk-reduction medication should be strongly recommended when a woman is diagnosed with LCIS.[7]

Breast Cancer

Cancer may develop in any of the tissues present in the breast including epithelial, muscle, connective tissue, or fat. The majority of breast cancers are carcinomas that appear in the epithelial cells that line other tissues. Breast carcinomas are categorized as invasive (infiltrating) or non-invasive (in situ). There are several histologic subtypes of invasive breast carcinomas. Non-invasive breast carcinomas are either ductal or lobular.

Worldwide, breast cancer is the most commonly diagnosed cancer and a leading cause of death among women age 40 to 55 years in the United States. African American women have a lower incidence of breast cancer but a higher mortality rate compared to white women.[14] The causes of this striking racial disparity are complicated and include many clinical and non-clinical factors. For example, breast cancer is more biologically aggressive in African American women. Biologic factors in combination with social inequities such as less access to care may partially explain the racial disparity in breast cancer mortality. The clinical implication of this paradox is that social inequities are modifiable when they are identified.

Women have approximately a 1 in 8 chance of developing breast cancer over the course of their lifetime.[15] The risk of developing breast cancer increases as one ages. Conversely, mortality rates have decreased. Between 1989 and 2012, death rates due to breast cancer have declined by 36% overall—a trend attributed to earlier detection, improved treatment modalities, and decreasing use of hormonal replacement therapy.

Factors That Affect Breast Cancer Risk

Several personal characteristics that increase or decrease one's risk for breast cancer have been identified as noted in **Table 13-2**. For example, breastfeeding decreases a woman's risk for subsequent breast cancer whereas alcohol abuse increases the risk for breast cancer. The National Cancer Institute provides a tool on its website that calculates an individual woman's risk of developing breast cancer (see the Resources section at the end of this chapter). The variables that are used to calculate this risk include a history of ductal carcinoma in situ or lobular carcinoma in situ, presence of *BRCA1* or *BRCA2* gene mutations, current age, age at menarche, age at first live birth, number of previous breast biopsies, number of first-degree relatives with breast cancer, and the woman's race/ethnicity.[16,17] This interactive program is based on the Gail model, the most commonly accepted program to calculate risk. Other models have been suggested, though none is infallible for forecasting purposes; nevertheless, they provide a framework for identifying women at risk.[17]

A meta-analysis of prospective studies regarding soy isoflavones consumption and risk of breast cancer suggests that soy isoflavones intake is associated with a significant reduction in the risk of breast cancer in Asian populations, but not in Western populations—although further studies are needed to confirm this finding.[18] Other factors that may be associated with lowering the risk of breast cancer include breastfeeding,[19] 3 to 5 hours of moderate to vigorous physical exercise per week,[20,21] limiting alcohol intake,[22,23] and maintaining a healthy body weight.[24-26]

Hereditary Breast and Ovarian Cancer Syndrome

Although most women with breast cancer do not have a family history of the disease, approximately 5% to 10% have inherited gene mutations that place them at increased risk for developing this disease. Hereditary breast and ovarian cancer syndrome (HBOC) refers to an inherited risk for female and male breast cancer. HBOC may be present in persons who have multiple cases of breast, ovarian, pancreatic, or prostate cancer within a family. The majority of persons with HBOC have mutations in the breast cancer susceptibility gene 1 (*BRCA1*), which is located on chromosome 17q21.3 or breast cancer susceptibility gene 2 (*BRCA2*), which is located on chromosome 13q12-13. These are genes that are associated with tumor suppression.

The prevalence of *BRCA1* or *BRCA2* varies. In the general population, the prevalence is approximately 1 in every 300 to 800 persons whereas approximately 1 in 40 individuals who are of Ashkenazi Jewish background carry the mutation. Mutations of these genes are inherited in an autosomal dominant pattern and therefore offspring have a 50% chance of being a mutation carrier.

When one of these gene mutations is present, the risk for developing breast and/or ovarian cancer is greatly increased. Overall, approximately 4.5% of breast cancer cases occur in women with a genetic mutation and 80% of those are *BRCA1* or *BRCA2*.[27] The cumulative lifetime risk of developing breast or ovarian cancer in women with *BRCA1* is approximately 72% and 44%, respectively. For women with *BRCA2* mutation, the risk of developing breast and ovarian cancer is approximately 69% and 17%, respectively.[27] BRCA mutations are also associated with an increased risk for pancreatic cancer and melanoma.

Screening for Hereditary Breast and Ovarian Cancer Syndrome

It is recommended that all women be screened for possible inherited cancer gene mutations and if risk factors are present (**Table 13-6**) they should receive genetic counseling and be offered genetic testing.[28-30] Knowing her genetic status may allow the woman to seek out preventive measures such as prophylactic removal of the breasts and/or ovaries or the use of medications to lower her risk for breast cancer. Several screening tools approved by the U.S. Preventive Services Task Force are listed in the Resources section at the end of this chapter.

Table 13-6 **Risk Factors for Hereditary Breast and Ovarian Cancer Syndrome**

Woman's Medical History
Breast cancer diagnosis at ≤ 45 years (female)
Breast cancer at any age and Ashkenazi Jewish ancestry
Breast cancer and close relative[a] with breast cancer that developed at age ≤ 50 years or close relative with epithelial ovarian, tubal, or peritoneal cancer at any age.
Breast cancer diagnosis with two or more close relatives[a] with breast cancer at any age
Breast cancer diagnosis at ≤ 50 years with limited or unknown family history
Triple-negative breast cancer diagnosed at ≤ 60 years

Two or more primary breast cancers with first diagnosed ≤ 50 years
Epithelial ovarian, tubal, or peritoneal cancer
Pancreatic cancer and two or more close relatives[a] with breast, ovarian, tubal or peritoneal, pancreatic or aggressive prostate cancer
Family History
Close relative who meets any of the above criteria
Close relative known carrier for *BRCA1* or *BRCA2*
Close relative[a] with male breast cancer

[a] Close relative refers to first, second, or third degree relative.

Data from American College of Obstetricians and Gynecologists. Practice Bulletin No. 182: hereditary breast and ovarian cancer syndrome. *Obstet Gynecol*. 2017;130e110-130e116[28]; Smith EC. An overview of hereditary breast and ovarian cancer syndrome. *J Midwifery Womens Health*. 2012;57:577-584[29]; Moyer VA, on behalf of the U.S. Preventive Services Task Force. Risk assessment, genetic counseling, and genetic testing for BRCA-related cancer in women: U.S. Preventive Services Task Force Recommendation Statement. *Ann Intern Med*. 2014;160:271-281.[30]

Women who are BRCA mutation carriers should practice self-breast examination monthly; receive a clinical breast examination, mammography, and breast MRI annually, and be evaluated with a pelvic examination, transvaginal ultrasound, and test for CA-125 every 6 months.[29]

Screening for Women at Low or Average Risk for Breast Cancer

Multiple low- and high-technological interventions have been used over the years for screening asymptomatic, low-risk women as well as for diagnostic purposes, especially when a breast mass is identified by a clinician. In recent years, widespread use of breast cancer screening modalities as well as advances in treatment have significantly reduced mortality due to breast cancer. A systematic review of randomized clinical trials, and observational and modeling studies published after January 1, 2000, demonstrated that for women of all ages at average risk, screening reduced breast cancer mortality by approximately 20%.[31]

Regular breast screening for cancer has become standard practice in much of the United States. Nevertheless, controversies regarding the best type of screening and the ideal frequency of screening persist. Organizations focused on cancer prevention, such as the American Cancer Society, tend to advocate more frequent screening. In contrast, other population-based groups, such as the U.S. Preventive Services Task Force, have published guidelines urging later initiation of screening and less frequent screening. Although all the groups have access to the same evidence, interpretation of that evidence depends to some degree on the lens through which the organization views it.

Breast Self-Examination for Screening

Traditionally, after menarche all women have been encouraged to perform monthly breast self-examination (BSE) as an instrument for early diagnosis of cancerous tumors and, therefore, as a means to ensure early treatment and decrease the mortality associated with breast cancer. Controversy now surrounds the recommendations regarding BSE, as several randomized trials conducted in the 1990s found no reduction in breast cancer mortality in women who practiced monthly BSE.

Not all of the research agreed on this point, particularly because some studies used differing methodologies and populations. For example, a retrospective study published in 2012 by the Mayo Clinic, reported that while the majority of women with breast cancer presented with image-detected breast cancer, 36% of women presented with palpable disease that was found on either BSE or clinical breast examination (CBE). Those with palpable disease tended to present symptomatically between screening mammograms. Such women also tended to be younger and have more aggressive tumor characteristics (e.g., larger tumor size, axillary node involvement, high grade, and estrogen receptor–negative tumors) compared to women whose tumors are detected via mammogram. The researchers concluded that BSE and CBE can remain important components in breast cancer diagnosis.[32]

With regard to women at high risk for breast cancer, a prospective study reported that 43% of new breast cancers were detected by BSE in 147 women who were at high risk for breast cancer and undergoing intensive breast cancer screening (e.g., yearly mammograms and yearly magnetic resonance imaging), and who are more likely to present with screening-interval cancers.[33] These studies suggested that BSE can be an important surveillance tool especially for women who have a high risk for developing breast cancer and that BSE education should

be a component of the follow-up of this population.[33,34]

Today, several organizations, including the Canadian Task Force on Preventive Health Care, the U.S. Preventive Services Task Force, and the World Health Organization, recommend against teaching women how to perform BSE. This recommendation is based on research that found BSE may actually be harmful because it increases both the number of imaging procedures and the number of breast biopsies without a concomitant decrease in mortality.[28] The American Cancer Society no longer recommends monthly BSE, but does state that BSE can be an option for women older than 20 years. The American Cancer Society recommends that women should be told about the benefits, limitations, and potential harms (the possibility of a false-positive result) of this examination technique.[35]

Currently, "breast awareness" is recommended in place of BSE. No standard definition exists for breast awareness, but the general consensus is that women can be taught to be aware of the normal shape and consistency of their breasts. Any changes in how their breasts feel or look should be an impetus for a visit with a healthcare provider. Women who have breast implants should likely include regular BSE in their plan for breast awareness.

For those women who perform BSE, the technique that is recognized by the American Cancer Society includes both inspection and palpation using the vertical stripe technique described in the *Breast Examination* appendix in the *Introduction to the Care of Women* chapter. Some evidence indicates that the up-and-down pattern of examining the breast decreases the likelihood that breast tissue will be missed.[34]

Clinical Breast Examination

Regular examination of the breasts by a healthcare professional also has been recommended as an important part of breast cancer screening, although there have been no randomized trials that evaluated the benefits of clinical breast examination as a sole screening modality. The Canadian National Breast Screening Study compared CBE and BSE plus mammography to CBE alone in women between 40 and 49 years and between 50 and 59 years. CBE was performed by healthcare professionals who were trained in the techniques of CBE and who also had periodic evaluations of performance quality. The frequency of cancer diagnosis, stage at diagnosis, and breast cancer mortality rates 11 to 16 years after entry into the study were similar in the two groups.[36] For women who were high risk for breast cancer based on a positive family history of breast cancer, another study reported that after a normal initial evaluation, women using BSE and/or CBE found more cancers than mammography.[37]

Several studies have noted that the CBE technique may influence the accuracy of the examination. Miller et al., in a review of the literature, found that poorly performed CBE (e.g., clinicians who fail to use a systematic search pattern) is widely prevalent.[36] A recent observational study noted that using a rubbing movement (defined as repetitive movements in a circular motion or pushing the fingers back and forth or right to left in a repetitive motion) was four times more likely to yield an accurate assessment compared to vertical movement (defined as pushing the fingertips or pads into and out of the breast tissue in a repetitive motion) or piano fingers (defined as the use of individual fingers, in series, to "march" across the breast tissue in a repetitive fashion).[38] When firm pressure is used, breast masses are more likely to

be identified.[39]

Mammography

Mammography, which uses X rays to image breast tissue, can be done as either a screening modality or a diagnostic test. A standard screening mammogram involves four images that are evaluated for changes suspicious of cancer, microcalcifications (a benign finding), distortions of the normal architecture of the breast, and nonpalpable lesions. A diagnostic mammogram is used to further examine areas of concern that are seen on the screening mammogram. Mammography can often detect early-stage breast cancers for which treatment may be more effective, with a subsequent increased likelihood of a cure. Numerous studies have shown that routine mammography may reduce breast cancer mortality by 30% overall and by 20% in women age 40 to 49 years.[40] Mammography can detect an estimated 80% to 90% of breast cancers in asymptomatic women.[40] Similar to research about BSE and CBE, some controversies exist, especially regarding the use of routine screening.

A Cochrane systematic review published in 2013 evaluated seven randomized trials that compared women screened by mammograms with women who were not screened using this technique. The trials included 600,000 women age 39 to 74 years. The three trials with adequate randomization failed to find a statistically significant reduction in breast cancer mortality at 13 years. The other four trials, all of which had suboptimal randomization, demonstrated approximately a 25% reduction in breast cancer mortality at the same time point (relative risk [RR], 0.75; 95% confidence interval [CI], 0.67–0.83). In their analysis, the authors noted that if one assumes mammograms lead to an overdiagnosis and overtreatment rate of 30% and a reduction of breast cancer mortality by 15%, then for every 2000 women screened over 10 years, 1 will avoid death from breast cancer and 10 will be treated unnecessarily. The women in the false-positive group may undergo unnecessary lumpectomies and mastectomies. Of the women who receive a false-positive result, the psychological distress may last for years and the cost is difficult to assess. Based on these results, the researchers did not recommend discontinuation of mammography, but strongly suggested that women be fully informed before entering into routine screening.[41]

Abnormal Mammogram

Mammographic findings are reported using the Breast Imaging Reporting and Data System (BI-RADS), as described in **Table 13-7**. Based on these findings, further imaging studies or a tissue diagnosis may be indicated.

Table 13-7	Breast Imaging Reporting and Data System Classification
BI-RADS Assessment Category	**Likelihood of Cancer**
Category 0: Incomplete—need additional imaging evaluation	
Category 1: Negative	Essentially 0% likelihood of malignancy

Category 2: Benign	Essentially 0% likelihood of malignancy (may be affected by breast density)
Category 3: Probably benign	> 0% but ≤ 2% likelihood of malignancy
Category 4: Suspicious	> 2% but 95% likelihood of malignancy
Category 5: Highly suggestive of malignancy	≥ 95% likelihood of malignancy

Abbreviation: BI-RADS, Breast Imaging Reporting and Data System.

Initiation and Frequency of Mammography

Debate exists particularly regarding the point at which to initiate mammography screening, the frequency of such screening, and the age at which it can be discontinued for women who are not at high risk for breast cancer (**Table 13-8**).*5,42-46* Studies have indicated that the sensitivity of mammography is highest among women 50 years and older, since they tend to have reduced breast density due to increased fatty tissue within the breast. Conversely, sensitivity is lowest among women younger than 50 years due to their increased breast density, though rapid tumor growth is more likely to occur in this younger population.[42] Based on this information, the U.S. Preventive Services Task Force issued new guidelines in 2009 recommending against routine mammography screening in women between 40 and 49 years; it called for biennial mammography screening for women between the ages of 50 and 74 years.[5] In contrast, the updated American Cancer Society screening guidelines recommend that women 40 to 44 years be given the choice of undergoing annual mammography; those 45 to 54 years have annual mammography; and those 55 years and older have biennial or annual mammography as long as their overall health is good and they have a life expectancy of 10 or more years.[43]

Table 13-8 **Breast Screening Recommendations for Women Not at High Risk of Breast Cancer from Selected Organizations**

Age	USPSTF (2016)	ACOG (2017)	ACS (2015)	NCCN (2016)
Clinical Breast Examination				
25–39 years	Insufficient evidence to recommend for or against	May offer every 1–3 years	No recommendation	Recommend every 1–3 years
≥ 40 years	Insufficient evidence to recommend for or against	Recommend annually	No recommendation	Recommend annually
Mammogram Initiation Age and Frequency				
40–44 years	Screening at age 40 years is made on individual basis	Offer annual or biennial starting at 40 years	Offer annual	Recommend annual at age ≥ 40 years
45–49 years	Screening at age 40 years is made on individual basis	Initiate annual or biennial after counseling if woman desires screening	Offer annual	Recommend annual at age ≥ 40 years
≥ 50 years	Every 2 years until age 75 years.	Recommend annual or biennial if not already initiated	Recommend biennial with option to continue annual for women ≥ 55 years	Recommend annual at age ≥ 40 years
Mammogram Discontinuation				
	Insufficient evidence to recommend for or against at 75 years. Decision to be based on shared decision making.	Insufficient evidence to recommend for or against at 75 years. Decision to be based on shared decision making.	When life-expectancy is < 10 years	When severe co-morbidities limit life expectancy to ≤ 10 years

Abbreviations: ACOG, American College of Obstetricians and Gynecologists; ACS, American Cancer Society; NCCN, National Comprehensive Cancer Network; USPSTF, U.S. Preventive Services Task Force.

Based on U.S. Preventive Services Task Force. Screening for breast cancer: U.S. Preventive Services Task Force Recommendation statement. *Ann Intern Med.* 2016;164(4):279-296[5]; Oeffinger KC, Fontham ET, Etzioni R, et al. Breast cancer screening for women at average risk: 2015 guideline update from the American Cancer Society. *JAMA.* 2015;314(15):1599-1614[43]; American College of Obstetricians and Gynecologists. Practice Bulletin No. 179: breast cancer risk assessment and screening in average-risk women. *Obstet Gynecol.* 2017;130(1):241-243[44]; Gradishar WJ, Anderson BO, Balassanian R, et al. NCCN guidelines insights: breast cancer, version 1.2017. *J Natl Compr Canc Netw.* 2017;15(4):433-451.[46]

In 2017, the American College of Obstetricians and Gynecologists (ACOG) issued a position paper regarding screening for women at average risk.[44] Similar to the American Cancer Society, ACOG now recommends offering a woman in her 40s the choice of a screening mammogram, with annual or biennial examinations to follow at least by age 50.

The Canadian Task Force for Preventive Care guidelines for screening of women at average risk were last issued in 2011, though newer guidelines may be available in the near future.[45] In 2011, the Canadian group stated that the evidence for making recommendations was weak, and suggested no screening for a woman in her 40s, with mammograms occurring every 2 to 3 years for women age 50 to 74 years.

The National Comprehensive Cancer Network (NCCN) is a consortium of almost 30 cancer centers in the United States. Most of these centers are designated by the National Cancer

Institute (one of the National Institutes of Health) as comprehensive cancer centers. This group also has issued separate screening guidelines that have small variations from those proposed by the other groups.[46]

Discontinuation of Screening. Similar to the controversy about when to initiate screening and at what frequency, there is debate about the appropriate age at which to discontinue screening mammograms. The U.S. Preventive Services Task Force has stated there is insufficient evidence when to discontinue such screening, although several groups recommend discontinuation if life expectancy is 10 years or less.

Table 13-8 provides a brief summary of the current recommendations of various groups for screening of women who are not at high risk for breast cancer. The variations in these guidelines present a challenge for a midwife in practice. Ideally, healthcare providers should choose one guideline instead of alternating among them.[28,43,44,46]

Digital Breast Tomosynthesis

A new mammographic technique, known as digital breast tomosynthesis, provides a three-dimensional picture of the breast using X rays. This more detailed mammographic technique is approved by the U.S. Food and Drug Administration, but is not yet considered the standard of care for breast cancer screening. Two prospective population-based studies showed that digital breast tomosynthesis significantly increased breast cancer detection rates among women screened with both the standard digital mammography and digital breast tomosynthesis performed at the same screening episode.[47,48]

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is the most sensitive test for breast cancer. Nevertheless, due to its high expense, it is recommended only for screening of women who are at very high risk for breast cancer, such as those who are positive for the *BRCA* gene mutation. MRI is recommended for use in conjunction with mammography as indicated.[49]

Ultrasonography

Ultrasonography is employed to screen the breast tissue of younger women at increased risk for breast cancer, diagnostically to differentiate between solid and cystic breast masses, and to guide fine-needle biopsies. The performance of this test is not affected by breast density. The American College of Radiology Imaging Network's randomized trial showed that adding ultrasound to mammography screening in high-risk women with dense breasts improved the sensitivity of the screening, but also increased the rate of false-positive examinations.[50] Women who are at high risk for breast cancer most likely are more fearful of a delayed diagnosis of breast cancer than of a false-positive result.[2]

Screening for Women at High Risk for Breast Cancer

In contrast to the multiple guidelines found in **Table 13-8**, recommendations for screening women who are at high risk for breast cancer are lacking.[51] Although there is a general consensus that women with the *BRCA1* or *BRCA2* genes should have early screening, some sources recommend starting at age 30 years and others at age 40 years. No evidence exists that the X rays in mammograms increase cancer risk among these women. Because MRI tends to have a higher degree of specificity compared to mammograms, there appears to be a movement toward annual MRI for this population. The use of MRI as a screening modality is also suggested for women who have a lifetime risk of 20% or more for breast cancer.[52,53]

Since care of women at high risk of breast cancer varies regionally, it is wise for midwives to establish lines of collaboration with experts in the area so that screening can be accomplished in a standardized manner. In addition, such collaboration enables the midwife to provide appropriate anticipatory guidance for women with increased risk of developing such malignancies.

Diagnosis of Breast Cancer

The majority of screening modalities report normal findings. When abnormalities are found, however, diagnostic testing is the next step. In many situations, these tests are performed by healthcare professionals in the local area.

The definitive diagnosis of breast cancer is generally made through tissue sampling. This can be accomplished through fine-needle aspiration, mammography- or ultrasound-guided core-needle biopsy, or excisional breast biopsy. The tissue sample obtained through the selected modality is then sent for histologic examination. If cancer is present, the histology report will state whether the tumor is ductal or lobular in origin. When the diagnosis of cancer has been made, it is also necessary to assess the lungs, abdomen, brain, and bone for metastasis.

A commonly used diagnostic test is fine-needle aspiration, also called fine-needle aspiration biopsy or fine-needle aspiration cytology. With this test, a physician inserts a thin (23–25 gauge), hollow needle into the breast mass and uses it to withdraw cells. Placement of the needle is performed under ultrasonic guidance and as an outpatient procedure. The cells obtained via fine-needle aspiration are subsequently evaluated specifically for malignancies.

Once breast cancer has been diagnosed, the malignancy is staged using the TNM system, which describes characteristics of the tumor (T), involvement of regional lymph nodes (N), and presence or absence of distant metastasis (M). Each cancer type (e.g., breast, brain, colon) has its own classification system, so letters and numbers do not always mean the same thing for every type of cancer; thus, the specific lexicon for breast cancer is used in this situation.[53] The TNM system is used in conjunction with staging, which is helpful as a guide to determine appropriate therapy options.[53]

An essential component of staging is investigation of the axillary nodes. In women with Stage I or II breast cancer, sentinel lymph node biopsy is performed. This is accomplished by injection of radioisotope dye into the breast tissue surrounding the cancer site so as to locate

the axillary node to which the dye initially spreads. The tissue from this node is then examined intraoperatively for cancer cells. If the sentinel node is positive for breast cancer, additional axillary nodes will be removed for evaluation. If the staging indicates greater than Stage II cancer, an axillary node dissection of at least 10 nodes from the first two levels of lymph drainage in the breast is indicated.

In addition to staging, the receptor status of the tumor is assessed. This evaluation provides information for treatment options and serves as an indicator of breast cancer prognosis. For example, the presence of estrogen and progesterone receptors improves prognosis, whereas over-expression of the growth factor receptor coded by the oncogene *Her2/neu* confers a poorer prognosis.[54,55]

Paget Disease and Inflammatory Breast Cancer

Although most breast cancer is associated with breast masses, Paget disease of the breast, also termed Paget disease of the nipple, is a rare cancer that involves changes of the nipple and areola. Women may report burning or itching of the nipple as a reason for a visit to a healthcare provider. The skin may have the appearance of eczema. Paget cells are malignant cells that are pathognomonic for the cancer. Most women with this disorder will be found to have ductal carcinoma in situ or invasive breast cancer.[56] Several other diseases bear similar names, such as Paget disease of the bone, but are distinct diseases—they share only the name of the physician who first described them.

Inflammatory breast cancer affects only a small number of women with breast cancer and without discrete masses. This condition often presents with a clinical picture similar to mastitis. This aggressive carcinoma involves the lymphatic system, and care for women with this cancer is best managed by a specialist in the area.

Treatment of Breast Cancer

Breast cancer treatment usually involves surgery, which may be followed with radiation therapy, chemotherapy, hormonal therapy, or immunotherapy. Radiation therapy or systemic therapies can be administered prior to surgery (neoadjuvant) with the goal of shrinking the tumor’s size, or they may be administered after surgery (adjuvant) with the goal of preventing cancer recurrence.

Most women with Stage I and Stage II breast cancers can be managed with breast-conserving surgery (lumpectomy) and sentinel node dissection, which is generally followed by radiation therapy. Breast-conserving surgery is contraindicated for women who have more advanced disease, women who have received previous radiation therapy, women who are pregnant (radiation is contraindicated in pregnancy), and women who have two or more primary tumors in separate quadrants of the breast or diffuse malignant-appearing microcalcifications. A history of collagen vascular disease (e.g., scleroderma or lupus) is a relative contraindication for breast-conservation treatment, as reports indicate that women with these diseases do not tolerate radiation.

Mastectomy involves removal of the breast tissue and the nipple/areolar complex, with

conservation of the pectoralis muscle, as well as sentinel lymph node dissection. Many women who are diagnosed with breast cancer are concerned about developing cancer in the contralateral breast and, therefore, request contralateral prophylactic mastectomy. The actual risk of developing contralateral breast cancer in the general population is extremely low (annual risk is 0.1% to 0.75%), so current breast cancer treatment guidelines strongly discourage this practice. However, women who are carriers of the *BRCA1*/*BRCA2* gene mutations have a 40% estimated 10-year risk of contralateral breast cancer; thus, contralateral prophylactic mastectomy may be recommended for these women, although the benefit of this preventive treatment when compared to conventional annual screening in this population is currently unclear.[57]

Radiation therapy after breast-conserving surgery reduces the recurrence rate for breast cancer by 50% and the death rate for breast cancer by 16.7%.[58] Radiation is used in conjunction with mastectomy only for late-stage breast cancers. In women with breast cancer who have axillary lymph node involvement, irradiation of the regional lymph nodes increases both disease-free years and survival years. Women need information about both the short-term (skin rashes and redness) and long-term side effects of radiation therapy (damage to the heart, lungs, or blood vessels in the chest; development of lung cancer; osteoradionecrosis).

Adjuvant (systemic) therapy is used to treat all stages of breast cancer and includes chemotherapeutic agents, hormonal therapies that are estrogen antagonists (e.g., tamoxifen, aromatase inhibitors) for cancer that is estrogen receptor positive, and immunotherapy (e.g., trastuzumab [Herceptin], a monoclonal antibody) for cancer that is found to overexpress the Her2/neu protein. All of these therapies are associated with a number of side effects. When considering the use of chemotherapeutic agents, it is important that the woman understands the associated side effects as well as her treatment options (**Table 13-9**).[53,55] For example, approximately 4% of women who take trastuzumab will develop ventricular dysfunction and congestive heart failure.[2]

Table 13-9 **Management of Common Side Effects of Chemotherapy for Breast Cancer**[a]

Side Effect	Management
Fatigue	Methylphenidate (Ritalin)
	Erythropoietin
Anemia	Iron supplementation
	Blood transfusion
	Erythropoiesis-stimulating drug (Procrit, Epogen)[b]
Neutropenia	Avoid contact with individuals who are sick or who are at risk for infection (e.g., schoolchildren)
	Agents that stimulate the production of neutrophils (Neupogen, Neulasta)
Nausea and vomiting	Eat small, frequent meals
	Stay well hydrated
	Avoid nausea triggers (unpleasant smells)
	Rest after eating
	Relaxation techniques
	Antiemetic drugs (e.g., ondansetron [Zofran], metoclopramide [Reglan], dexamethasone

	[Decadron])
Mucositis	Cold liquids and ice chips
	2% viscous lidocaine
	50/50 attapulgite/diphenhydramine (Kaopectate/Benadryl)
	Systemic analgesics
Diarrhea	Loperamide (Imodium)
	Octreotide (Sandostatin)
Constipation	Psyllium (Metamucil)
	Senna (SenoKot)
	Docusate sodium (Colace)
	Bisacodyl (Dulcolax)

[a] Many nonpharmaceutical treatments for breast cancer are taken based on anecdotal reports and lack evidence of effectiveness.

[b] The U.S. Food and Drug Administration has issued a black-box warning noting that studies have shown these drugs increase the risk of thromboembolic disease and may promote tumor growth.

Modified with permission from Carlson N, King J. Overview of breast cancer treatment and reconstruction for primary care providers. *J Midwifery Womens Health.* 2012;57(6):558-568.[55] © 2012, with permission from Wiley.

Breast reconstruction is an option for all women who undergo surgery. The reconstruction can be performed immediately post mastectomy or at a later time. Reconstructive surgery does not influence recurrence of the breast cancer or overall survival.

Follow-up

During the first 2 years after breast cancer treatment is completed, follow-up appointments generally occur every 3 to 6 months and include physical examination and mammography. Routine laboratory evaluation for metastasis (e.g., liver function tests, bone scans) is not recommended unless specifically indicated clinically.[59,60] Ovarian dysfunction is common in women of reproductive age who are treated for breast cancer, and reflects their age, ovarian function at the time of treatment, and the specific chemotherapy agents used. One study observed that 83.1% of women between the ages of 18 and 34 years resumed menstruating, on average, 3.5 months following breast cancer treatment; therefore, a discussion regarding contraception is merited.[61]

Contraception Following Breast Cancer

Breast cancer is a hormonally sensitive tumor and therefore, hormonal contraception is contraindicated for women with current or past history of breast cancer. Copper-containing intrauterine devices, tubal ligation, and vasectomy are considered to be better contraceptive options for women with a history of breast cancer.[62]

The safety of estrogen-containing hormone therapy for menopausal symptoms in women with a history of breast cancer is not well established. Nonhormonal treatments, such as selective serotonin reuptake inhibitors or gabapentin (Neurontin), are effective alternative options for the treatment of women with vasomotor symptoms. Vaginal estrogens are effective for the

treatment of women with atrophic vaginitis, although few data have been published on the safety of these medications in women with a history of breast cancer.[63,64]

Breast Conditions During Pregnancy and Lactation

The same breast disorders that occur in nonpregnant women can also present during pregnancy.[65] Notably, the breast examination can be more difficult when examining a pregnant woman secondary to engorgement and proliferation of breast tissue. Thus, small breast masses are more difficult to detect. Fibroadenomas can grow during pregnancy, and occasionally they may infarct and become painful. In addition, a few types of benign lesions are unique in pregnancy and lactation. The most common of these are lactational adenomas, galactoceles, mastitis, and breast abscess. The diagnosis and management of mastitis and breast abscess are reviewed in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

Lactational adenomas usually occur during the third trimester or during lactation.[66] These breast masses are similar to fibroadenomas in that they are painless, soft mobile masses. A lactational adenoma may appear as a fibroadenoma on ultrasound. These masses are not associated with an increased risk for malignancy.

A galactocele is a distended mammary duct with milk retention (milk retention cyst). Galactoceles usually present after cessation of breastfeeding.[66] They are usually round, soft, painless masses that may be associated with nipple discharge. Risk factors for galactocele include poor latch and abrupt cessation of breastfeeding. They usually regress spontaneously and do not require aspiration.

Breast Cancer

Treatment options for breast cancer during pregnancy are the same as for nonpregnant women, except for radiation therapy, which is considered to be unsafe for the embryo/fetus throughout all stages of gestation. It is also recommended that chemotherapy be delayed until after the first trimester of pregnancy due to an increase in spontaneous miscarriages as well as a possible increase in the risk for major fetal malformations secondary to exposure to chemotherapeutic agents. Chemotherapy in the second and third trimesters, while not associated with major malformations, may cause fetal growth restriction. Termination of pregnancy does not improve survival rates and is generally not recommended.[59,65]

Breast Cancer in Transgender Individuals

Transgender individuals may choose to undergo treatment to alter their breasts as they go through a transition from male to female/trans woman (e.g., hormone therapy, breast augmentation) or from female to male/trans man (e.g., mastectomy).

Trans women most often choose to take estrogen hormone therapy to create female secondary sex characteristics. Breast development in trans women on hormone therapy is rarely as complete as it is in genetic females, so many trans women will consider breast augmentation to add to breast volume. It is important when using estrogen that the risks versus benefits tradeoff be carefully evaluated for each individual. The standard mammography guidelines are recommended for this population given the lack of research regarding breast cancer risk in trans women.[67,68]

Trans men often perceive that breasts are a constant reminder of their biologic sex and may choose to either use a breast binder or pursue a mastectomy. If a mastectomy has not been performed, continued mammography screening is indicated. The risk for breast cancer after a mastectomy for this indication is rare, although the American Cancer Society recommends that women who have had mastectomies undergo CBE annually to evaluate the chest wall, skin, and incision. These guidelines can be extrapolated to transgender individuals even though the recommendations do not specifically identify their applicability.[67,68]

Conclusion

The midwife may be the first healthcare provider a woman consults for breast-related concerns. Breast cancer is one of the most frequently diagnosed cancers in women throughout the world. Screening and early detection are essential components of primary care services provided by midwives. The midwife is also an important member of the multidisciplinary team caring for a woman with the diagnosis of breast cancer, and fulfills this role by providing accurate information and support as the woman is making decisions regarding breast cancer treatment, and management of symptoms.

Resources

Organization	Description	Webpage
American Cancer Society (ACS)	Nonprofit organization with updated statistics regarding all types of cancers, including breast cancer	https://www.cancer.org
Cochrane Nordic Mammogram Leaflet	Pamphlet available in multiple languages discussing components of shared decision making for a woman considering mammogram screening	http://nordic.cochrane.org/mammography-screening-leaflet
Susan G. Komen Breast Cancer Foundation	Nonprofit organization named after an individual who died of breast cancer; site includes support resources for women with the disease	http://ww5.komen.org
Point-of-Care Apps		
Touch Surgery	Free surgical simulator app; full-color illustrations of the breast, including surgical procedures, which can be used when educating women about expected care	https://www.breastcancercare.org.uk/information-support/vita-magazine/touch-sur surgery-app
Breast Cancer Screening Tools		
National Cancer Institute (NCI)	Breast Care Risk Tool is an interactive risk calculator based on the Gail	http://www.cancer.gov/bcrisktool

	model, which is used to estimate an individual's risk of developing breast cancer	
National Comprehensive Cancer Network (NCCN)	Guidelines for breast cancer screening, breast cancer risk reduction, and guidelines for women with hereditary breast and ovarian cancer syndrome	www.nccn.org
U.S. Preventive Services Task Force (USPSTF)	Final recommendation statement BRCA-Related Cancer: Risk assessment, genetic counseling, and genetic testing	https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/brca-related-cancer-risk-assessment-genetic-counseling-and-genetic-testing
	This summary document includes several validated screening tools for familial cancer risk including the Ontario Family History Assessment Tool, Manchester Scoring System, and Referral Screening Tool	

References

1. Carswell JM, Stafford DEJ. Normal physical growth and development. In: Neinstein LS, ed. *Adolescent Health Care*. 6th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2016:30-32.
2. Fritz MA, Speroff L. The breast. In: Fritz MA, Speroff L, eds. *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2011:621-673.
3. Dickson G. Gynecomastia. *Am Fam Physician*. 2012;85(7):716-722.
4. Pearlman MD, Griffin JL. Benign breast disease. *Obstet Gynecol*. 2010;116(3):747-758.
5. U.S. Preventive Services Task Force. Screening for breast cancer: U.S. Preventive Services Task Force Recommendation statement. *Ann Intern Med*. 2016;164(4):279-296.
6. Hoffman B, Schorge J, Bradshaw K, Halvorson LM, Schaffer JL, Corton MM. Breast diseases. In: Hoffman B, Schorge J, Bradshaw K, Halvorson LM, Schaffer JL, Corton MM, eds. *Williams Gynecology*. 3rd ed. New York, NY: McGraw-Hill; 2016:275-286.
7. American College of Obstetricians and Gynecologists. Practice Bulletin No. 164: diagnosis and management of benign breast disorders. *Obstet Gynecol*. 2016;127(6):e141-e156.
8. Amin AL, Purdy AC, Mattingly JD, Kong AL. Benign breast disease. *Surg Clin North Am*. 2013;93:299-308.
9. Mirghafourvand M, Mohammad-Alizadeh-Charandabi S, Ahmadpour P, Javadzadeh Y. Effects of Vitex agnus and flaxseed on cyclic mastalgia: a randomized controlled trial. *Complement Ther Med*. 2016;24:90-95.
10. Pruthi S, Wahner-Roedler DL, Torkelson CJ, et al. Vitamin E and evening primrose oil for management of cyclical mastalgia: a randomized pilot study. *Altern Med Rev*. 2010;15(1):59-67.
11. Goyal A. Breast pain. *BMJ Clin Evid*. 2011;0812.
12. Lazzeroni M, Serrano D, Dunn BK, et al. Oral low dose and topical tamoxifen for breast cancer prevention: modern approaches for an old drug. *Breast Cancer Res*. 2012;14(5):214.
13. Wong Chung J, Jeuriens-van de Ven S, van Helmond N, Wauters CA, Duijm LE, Strobbe LJ. Does nipple discharge color predict (pre-) malignant breast pathology? *Breast J*. 2016;22(2):202-208.
14. Yedjou CG, Tchounwou PB, Payton M, et al. Assessing the racial and ethnic disparities in breast cancer mortality in the United States. Chakraborty J, ed. *International Journal of Environmental Research and Public Health*. 2017;14(5):486.
15. Akram M, Iqbal M, Daniyal M, Khan AU. Awareness and current knowledge of breast cancer. *Biological Research*. 2017;50:33. doi:10.1186/s40659-017-0140-9.
16. Gail MH, Brinton LA, Byar DP, et al. Projecting individualized probabilities of developing breast cancer for white females who are being examined annually. *J Natl Cancer Inst*. 1989;81:1879-1886.
17. Gail MH, Costantino JP, Pee D, et al. Projecting individualized absolute invasive breast cancer risk in African American women. *J Natl Cancer Inst*. 2007;99(23):1782-1791.
18. Dong JY, Qin L. Soy isoflavones consumption and risk of breast cancer incidence or recurrence: a meta-analysis of prospective studies. *Breast Cancer Res Treat*. 2011;25:315-323.
19. Islami F, Liu Y, Jemal A, et al. Breastfeeding and breast cancer risk by receptor status: a systematic review and meta-analysis. *Ann Oncol*. 2015;26(12):2398-2407.
20. Fournier A, Dos Santos G, Guillas G, et al. Recent recreational physical activity and breast cancer risk in postmenopausal women in the E3N cohort. *Cancer Epidemiol Biomarker*. 2014;23(9):1893-1902.
21. Magné N, Melis A, Chargari C, et al. Recommendations for a lifestyle which could prevent breast cancer and its relapse: physical activity and dietetic aspects. *Crit Rev Oncol Hematol*. 2011;80:450-459.
22. Dam MK, Hvidtfeldt UA, Tjønneland A, et al. Five year change in alcohol intake and risk of breast cancer and coronary heart disease among postmenopausal women: prospective cohort study. *BMJ*. 2016;353:i2314.
23. Chen WY, Rosner B, Hankinson SE, Colditz GA, Willett WC. Moderate alcohol consumption during adult life, drinking patterns, and breast cancer risk. *JAMA*. 2011;306(17):1884-1890.
24. Yung RL, Ligibel JA. Obesity and breast cancer: risk, outcomes, and future considerations. *Clin Adv Hematol Oncol*. 2016;14(10):790-797.
25. Eichholzer RA, Rohrmann S, Schmid S, Guth U. Overweight, obesity, and breast cancer screening: results from the 2012 Swiss Health Survey. *Eur J Cancer Prev*. 2016;24:130-136.
26. Patterson RE, Cadmus LA, Emond JA, Pierce JP. Physical activity, diet, adiposity and female breast cancer prognosis: a review of the epidemiologic literature. *Maturitas*. 2010;66:5-15.
27. Kuchenbaecker KB, Hopper JL, Barnes DR, et al. Risks of breast, ovarian, and contralateral breast cancer for BRCA1 and BRCA2 mutation carriers. *JAMA*. 2017; 317:2402.
28. American College of Obstetricians and Gynecologists. Practice Bulletin No. 182: hereditary breast and ovarian cancer syndrome. *Obstet Gynecol*. 2017;130e110-130e116.
29. Smith EC. An overview of hereditary breast and ovarian cancer syndrome. *J Midwifery Womens Health*. 2012;57:577-584.

30. Moyer VA, on behalf of the U.S. Preventive Services Task Force. Risk assessment, genetic counseling, and genetic testing for BRCA-related cancer in women: U.S. Preventive Services Task Force Recommendation Statement. *Ann Intern Med*. 2014;160:271-281.
31. Myers ER, Moorman P, Gierisch JM, et al. Benefits and harms of breast cancer screening: a systematic review. *JAMA*. 2015;314(15):1615-1634.
32. Ma I, Dueck A, Gray R, et al. Clinical and self breast examination remain important in the era of modern screening. *Ann Surg Oncol*. 2012;19:1484-1490.
33. Wilke LG, Broadwater G, Rabiner S, et al. Breast self-examination: defining a cohort still in need. *Am J Surg*. 2009;198(4):575-579.
34. Visser A, Box W, Prins JB, Hoogerbrugge N, Van Laarhoven H. Breast self-examination education for *BRCA* mutation carriers by clinical nurse specialists. *Clin Nurse Spec*. 2015;29(3):E1-E7.
35. American Cancer Society. Breast cancer: early detection. Available at: https://www.cancer.org/cancer/breast-cancer/screening-tests-and-early-detection/american-cancer-society-recommendations-for-the-early-detection-of-breast-cancer.html Accessed October 17, 2017.
36. Miller AB, Baines CJ. The role of clinical breast examination and breast self-examination. *Prevent Med*. 2011;3:118-120.
37. Gui GPH, Hogben RKF, Walsh G, A'Hern R, Eeles R. The incidence of breast cancer from screening women according to predicted family history risk: does annual clinical examination add to mammography? *Eur J Cancer*. 2001;37:1668-1673.
38. Humphrey LL, Helfand M, Chan BK, Woolfe SH. Breast cancer screening: a summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2002;137(5 pt 1):347-360.
39. Laufer S, Cohen ER, Maag AD, Kwan C, Vanveen B, Pugh CM. Multimodality approach to classifying hand utilization for the clinical breast examination. *Stud Health Technol Inform*. 2014;196:238-244.
40. Welch HG, Prorok PC, O'Malley AJ, Kramer BS. Breast-cancer tumor size, overdiagnosis, and mammography screening effectiveness. *N Engl J Med*. 2016;375:1438-1447.
41. Gøtzsche PC, Jørgensen KJ. Screening for breast cancer with mammography. *Cochrane Database Syst Rev*. 2013;6:CD001877. doi:10.1002/14651858.CD001877.pub5.
42. Yi M, Hunt KK. Optimizing mammography screening intervals. *JAMA*. 2015;314(15):1635-1636.
43. Oeffinger KC, Fontham ET, Etzioni R, et al. Breast cancer screening for women at average risk: 2015 guideline update from the American Cancer Society. *JAMA*. 2015;314(15):1599-1614.
44. American College of Obstetricians and Gynecologists. Practice Bulletin No. 179: breast cancer risk assessment and screening in average-risk women. *Obstet Gynecol*. 2017;130(1):241-243.
45. Canadian Task Force on Preventive Health Care. Recommendations on screening for breast cancer in average-risk women aged 40-74 years *CMAJ*. 2011;183(17):1991-2001.
46. Gradishar WJ, Anderson BO, Balassanian R, et al. NCCN guidelines insights: breast cancer, version 1.2017. *J Natl Compr Canc Netw*. 2017;15(4):433-451.
47. Vedantham S, Karellas A, Vijayaraghavan GR, Kopans DB. Digital breast tomosynthesis: state of the art. *Radiology*. 2015;277(3):663-684.
48. Houssami N, Miglioretti DL. Digital breast tomosynthesis: a brave new world of mammography screening. *JAMA Oncol*. 2016;2(6):725-726.
49. Narayan AK, Visvanathan K, Harvey SC. Comparative effectiveness of breast MRI and mammography in screening young women with elevated risk of developing breast cancer: a retrospective cohort study. *Breast Cancer Res Treat*. 2016;158(3):583-589.
50. Gundry KR. Breast ultrasound: indications and findings. *Clin Obstet Gynecol*. 2016;59(3):380-393.
51. Wellings E, Vassiliages L, Abdalla R. Breast cancer screening for high-risk patients of different ages and risk: which modality is most effective? *Cureus*. 2016;8(12):e945.
52. Fuller MS, Lee CI, Elmosre JG. Breast cancer screening: an evidence-based update. *Med Clin North Am*. 2015;99(3):451-468.
53. Bond-Bero S. Filling the gap for early-stage breast cancer follow-up: An overview for primary care providers. *J Midwifery Womens Health*. 2016;61:166-176.
54. Hernadez-Blanquisett A, Touya D, Strasser-Weippl K, Ruiz R, St Louis J, Goss P. Current and emerging therapies of *HER2*-positive metastatic breast cancer. *Breast*. 2016;20:170-177.
55. Carlson N, King J. Overview of breast cancer treatment and reconstruction for primary care providers. *J Midwifery Womens Health*. 2012;57(6):558-568.
56. Sandoval-Leon AC, Drews-Elger K, Gomez-Fernandez CR, Yepes MM, Lippman ME. Paget's disease of the nipple. *Breast Cancer Res Treat*. 2013;141(1):1-12.
57. Barry M, Sacchini V. When is contralateral mastectomy warranted in unilateral breast cancer? *Expert Rev Anticancer Ther*. 2011;11(8):1209-1214.
58. Early Breast Cancer Trialists' Collaborative Group, Darby S, McGale P, et al. Effect of radiotherapy after breast-

conserving surgery on 10-year recurrence and 15-year breast cancer death: meta-analysis of individual patient data for 10,801 women in 17 randomised trials. *Lancet*. 2011;378(9804):1707-1716.
59. Becker S. Breast cancer in pregnancy: a brief clinical review. *Best Pract Res Clin Ob*. 2016;33:79-85.
60. Sisler J, Chaput G, Sussman J, Ozokwelu E. Follow-up after treatment for breast cancer: practical guide to survivorship care for family physicians. *Can Fam Physician*. 2016;62:805-811.
61. Hickey M, Peate M, Saunders CM, Friedlander M. Breast cancer in young women and its impact on reproductive function. *Human Reprod Update*. 2009;15(3):323-339.
62. Centers for Disease Control and Prevention. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR*. 2016;65(15). Available at: https://www.cdc.gov/mmwr/volumes/65/rr/rr6503a1_appendix.htm. Accessed April 24, 2017.
63. King J. Cancer. In: King TL, Brucker MC, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:837-860.
64. Biglia N, Peano E, Sgandurra P, et al. Low-dose vaginal estrogens or vaginal moisturizer in breast cancer survivors with urogenital atrophy: a preliminary study. *Gynecol Endocrinol*. 2010;26(6):404-412.
65. Ullery M, Carter L, McFarlin BL, Giurgescu C. Pregnancy-associated breast cancer: significance of early detection. *J Midwifery Womens Health*. 2009;54:357-363.
66. Vashi R, Hooley R, Butler R, Geisel J, Phipotts L. Breast imaging of the pregnant and lactating patient: physiologic changes and common benign entities. *Am J Roentgenol*. 2013;200:329-336.
67. Bond Maycock L, Powell Kennedy H. Breast care in the transgender individual. *J Midwifery Womens Health*. 2014;59:74-81.
68. Selix NW, Rowniak S. Provision of patient-centered transgender care. *J Midwifery Womens Health*. 2016;00:1-8.

CHAPTER

14

Reproductive Tract and Sexually Transmitted Infections

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Introduction

A woman's overall well-being includes sexual and reproductive components. The healthy expression of sexuality and the ability to safely engage in sexual and reproductive behaviors is supported by national and international organizations such as the United Nations,[1] the World Health Organization (WHO),[2] and the *Healthy People* initiative in the United States.[3] Unfortunately, women across the globe continue to struggle to obtain services to improve their sexual and reproductive health. Midwives are ideal care providers for women's sexual health needs, which include screening, diagnosis, and treatment of infections in women and their partners. This chapter reviews midwifery care for women with reproductive tract and sexually transmitted infections (STI).

Sexually transmitted infections are diseases that are passed from one person to another through sexual contact and sometimes via genital contact. Some STIs, such as gonorrhea, are transmitted only via exchange of body fluids during sexual contact; this chapter primarily focuses on these diseases. However, it is possible to spread a wide variety of infections and parasites through sexual contact. Some conditions that are not traditional STIs but are easily transmitted during sexual contact are included in the final section of this chapter.

A broad range of terms have been used to describe conditions that are transmitted through sexual contact. Many of these terms, including *venereal disease*, may have connotations of judgment or shame related to their use with public health campaigns. To avoid these negative stereotypes, the term *sexually transmitted disease* became more commonly used, and is the current term of choice of the Centers for Disease Control and Prevention (CDC). However, the term *sexually transmitted infection* (STI) is more precise, as not all sexually transmitted pathogens have immediate disease manifestations. The term "sexually transmitted infections" will be used throughout this chapter for precision and clarity.

Incidence of Sexually Transmitted Infections

STIs impact health across the life span, having both wide-ranging and persistent effects. According to the WHO, more than 357 million cases of curable STIs occur annually.[2] The CDC reports that 20 million new STI infections occur each year in the United States, with an immediate cost of more than $16 billion.[4] While immediate costs are easy to calculate, the long-term costs of STIs may be much higher, as such infections are associated with cancer, pelvic inflammatory disease, infertility, ectopic pregnancy, and lifelong malformations and blindness in infants born to women who have an STI during pregnancy or at the time of birth.[2]

Sexually transmitted infections vary greatly in incidence by geographic area, even within the United States. In addition, many variants or strains of an infection may occur, and the prevalence of these strains differs. If a woman or her partner is new to the geographic area or has recently traveled to another location, it can be useful to check treatment guidelines developed in the geographic area where the infection may have originated. For example, the antibiotic resistance profile of *Neisseria gonorrhoeae* varies by geographic region.[5] National and international organizations monitor the prevalence of infections and their susceptibility to treatment and produce reports to guide clinical management. However, with increasing global

travel, women may be infected with strains or subtypes not typical for their current location.

The prevalence of STIs also varies by personal risk factors. Women have a disproportionate health burden from STIs. Compared to men, women are more likely to contract an STI from a single sexual encounter and are more likely to have long-term sequelae.[2] In addition, women are often disproportionately stigmatized for having an STI due to taboos surrounding female sexuality. Furthermore, accessing appropriate and affordable services related to STI care and prevention may be difficult for some women.

The CDC monitors reportable diseases, such as chlamydia, gonorrhea, and syphilis.[4] Current information about these conditions is maintained on the agency's website and is available through its state-by-state interactive atlas.[6] The CDC also periodically updates guidelines for diagnosis and treatment of STIs; these guidelines, which were most recently updated in 2015, are extensively used in the United States.[7] The CDC guidelines can be downloaded, and paper copies can also be ordered.[7] In addition, the CDC has free apps to assist clinicians in accessing guidelines quickly at the point of care (see the Resources section at the end of this chapter). The U.S. Preventive Services Task Force (USPSTF) also releases guidelines that address STI screening based on current evidence. In addition, the Agency for Healthcare Research and Quality (AHRQ) has a mobile electronic device application that displays the USPSTF-recommended STI screenings based on the individual's age and risk factors.

While the CDC provides clear guidance for STI treatment in the United States, these guidelines are not always appropriate in other countries because treatment availability, drug names, and antibiotic resistance vary by geographic location. The WHO and country-specific health organizations are the best source of current treatment information outside the United States.

History and Physical Examination for Vaginal Symptoms and Sexually Transmitted Infections

Women may come to see the midwife with concerns about vaginitis or STIs or present for a comprehensive physical examination. Begin any visit by asking the woman in an open and inviting tone the reason for her clinic visit. If a woman is concerned about her privacy, she may have not been completely forthcoming with previous staff. Give her a chance to talk without interruption, as listening patiently establishes rapport and provides important information.

Problem-Focused Visit

Ideally, the woman will have already participated in a more comprehensive visit that included a complete history and physical examination. In these cases, a targeted but thorough history of the symptoms is important. The chief concern should be used to generate a list of differential diagnoses. It is helpful to use a systematic method to assess the problem. Many clinicians use the OLDCARTS technique to assess the Onset, Location/radiation, Duration, Character, Aggravating factors, Relieving factors, Timing, and Severity of symptoms. After exploring the woman's chief concern for the visit, ask her about additional STI-related symptoms such as fever, sore throat, enlarged lymph nodes, sores in the mouth or genital area, dysuria, discharge, and dyspareunia.

Sexual History

Obtaining an in-depth sexual history helps determine the woman's risk for infections and the need for screening and prevention during both well-woman care and problem-based visits. The woman is an essential partner in her health care, and listening to her concerns and history is one of the first steps in establishing a trusting relationship. It is important to be respectful when obtaining a thorough sexual history. While striving to put the woman at ease, the clinician should not shy away from asking personal questions. It is crucial to know the extent and location of previous and current sexual contact. For example, a vaginal test for gonorrhea would be useless if the woman has engaged in only anal or oral intercourse.

A variety of approaches may be used when obtaining a thorough sexual history. Some midwives request the woman complete a paper form as a guide for discussion, while others talk through the entire history. It is important to provide privacy in which to complete the forms and a private space for discussion. A woman may be less forthcoming when others are with her, especially her parents or children. While she may want her family or friends nearby for support, it is ideal to speak with her alone at some point during her visit. When discussing sensitive topics, it helps if the midwife and the woman are seated on equal levels without anything blocking their vision. While taking note of key items discussed, the focus should be on the woman, rather than the chart or computer.

A guide for obtaining a sexual history can be found in the appendix *Collecting a Health History*, in the *Introduction to the Care of Women* chapter. It is important to ask questions (5 P's)[8,9] to obtain needed information without making assumptions about the woman's sexuality

or beliefs. Remain open and engaged no matter what she reveals, using open- and closed-ended questions as needed. Focus on building rapport and obtaining the needed information to guide your assessment. Teaching about safe practices can take place later in the visit.

Physical Examination for Vaginitis and Sexually Transmitted Infections

The physical examination for STIs includes evaluation of all systems that may be affected. These systems are included in a complete well-woman examination but are examined individually during a targeted physical examination. **Table 14-1** provides a list of important systems to include in a targeted examination if the woman or midwife has concerns about STIs. While looking closely for STIs, take a holistic approach to the examination and avoid ruling out non-STI-related diagnoses too soon. A comprehensive and gentle physical examination can reveal many non-infection-related abnormalities that may be causing symptoms. Although laboratory data can be very valuable in the diagnosis of an STI, the physical examination is important to help determine which laboratory tests are indicated. In addition, treatment may be initiated on the basis of physical findings without waiting for laboratory confirmation.

Table 14-1 Physical Examination for Sexually Transmitted Infections

Organ/System	Abnormal Findings Related to STIs
Mouth	Lesions, pharyngitis
Cervical lymph nodes/chains	Palpable lymph nodes, possibly painful
Abdominal palpation	Abdominal tenderness
Inguinal lymph nodes	Palpable lymph nodes, which may be fluctuant or draining
Inspection of the pubis	Parasites, lesions, excoriations
Vulva, vagina, and perineum	Lesions, warts, excoriations, edema, discharge
Paraurethral (Skene's) glands	Enlarged glands, discharge on palpation
Greater vestibular (Bartholin's) glands	Enlarged glands, discharge on palpation
Urethra	Inflammation, swelling, discharge
Rectal area and anus	Lesions, warts, excoriations, signs of trauma
Speculum examination	Lesions, discharge, color, and character of vaginal walls
Vaginal mucosa	Overall appearance, lesions, discharge
Cervix	Wet mount, gonorrhea and chlamydia tests, cultures/testing of lesions, HPV testing with Pap test
Obtain needed specimens	
Bimanual examination	Enlarged uterus could indicate pregnancy; tenderness is a symptom of PID
Uterus	
Cervix	Cervical motion tenderness is a sign of PID
Adnexa	Adnexal tenderness is a sign of PID (and ectopic pregnancy)
Urine testing as indicated (nitrites, leukocytes, NAAT for gonorrhea and chlamydia)	Urinary tract infection signs include nitrites, leukocyts Urine test for chlamydia and gonorrhea is appropriate for screening and testing
Blood work as indicated (HIV, VDRL/RPR, HSV, hepatitis B)	Blood testing can confirm or enhance physical examination findings

Abbreviations: HIV, human immunodeficiency virus; HPV, human papillomavirus; HSV, herpes simplex virus; NAAT, nucleic acid amplification test; PID, pelvic inflammatory disease; RPR, rapid plasma reagin; STI, sexually transmitted infection; VDRL, Venereal Disease Research Laboratory test.

Vaginal Microbiome

The bacteria that are normally present in the vagina serve as the first line of defense against vaginal infection.[10] These bacteria can prevent infection via either competitive exclusion or direct killing. In addition, the vaginal bacteria produce agents such as lactic acid that creates a pH level, which is inhospitable for some pathologic organisms.

A healthy vaginal flora is usually dominated by *Lactobacillus* species, especially *L. crispatus*.[10,11] However, the mix of organisms that constitutes a healthy vaginal microbiome differs by race and ethnicity and many modifiable factors such as smoking, sexual behavior, intravaginal practices, and diet. In addition, the vaginal microbiome may be an important mediator explaining some of the link between socioeconomic factors and increased risk for STIs. Dysbiosis—the term used to describe an unhealthy vaginal microbiome—is a known risk factor both for acquisition of STIs and for adverse reproductive outcomes.[11,12]

A great deal remains to be learned about the vaginal microbiome and its relationship to health. The role of modifiable factors, metabolic interactions within the vaginal microbiome, and the effects of treatment of dysbiosis are largely unknown at this time. That said, health education about modifiable factors that dysregulate the vaginal microbiome should be included in all health counseling about vaginal infections and sexually transmitted infections. A review of preventive strategies can be beneficial for women of all ages; the points to be covered in such a review are summarized in **Table 14-2**.

Table 14-2 Health Education for Prevention of Vaginal Irritation and Vaginitis

General Health Measures That Support a Health Vaginal Microbiome
Avoid cigarette smoking.
Avoid foods that have high dietary fat content and glycemic load.
Cleaning the Vulva and Vagina
Clean only the outside of the vagina with gentle, unscented soap.
Do not douche, as it eliminates helpful bacteria and increases risk of infection.
Use unscented, dye-free soaps, especially on the hands, anal areas, and genital areas of the woman and her partner.
Use a clean, previously dry washcloth or towel each time the vulva is cleaned or dried.
Wipe or pat the vulva gently after urinating; excessive wiping can damage the skin.
Cleaning with warm water after urinating and defecating can help some women.
Keeping the Vulva Dry
Gently but thoroughly dry the vulva after bathing; a hair dryer is ideal for this purpose.
Sleep without underwear to increase air circulation.
Cotton underwear (with a cotton crotch) is the most breathable.
Avoid staying in a wet bathing suit.
Avoiding Irritating Substances
Use unscented and dye-free detergents for underwear of both partners.
Double-rinse underwear to eliminate soap.
Avoid pads and panty liners unless needed, and try a variety of pads to find the least irritating.

Clean and gently dry the vulva as soon as possible after incontinence, as urine and stool are caustic to skin.

Altering Sexual Practices

Only clean objects should touch or enter the vagina (e.g., fingers, penis, sex toys).

Sexual contact should be gentle and nonpainful.

Use a water-based lubricant to prevent chafing during sexual activities.

Try a variety of pH-balanced lubricants to determine which is least irritating.

Clean objects (including fingers, penis, and sex toys) with soap and water after contact with the anus before they touch the vagina.

Consider condoms or nonvaginal ejaculation with recurrent infections, as semen may affect the pH of the vagina, especially when intercourse is frequent.

Choosing Birth Control

Spermicides may irritate the vagina.

Oral contraceptives alter the vaginal flora in some women, increasing their risk of vaginitis.

Reconsider use of these birth control method(s) for recurrent, severe vaginitis.

Vaginitis

Vaginitis, which is inflammation of the vagina, refers to a disruption of the normal healthy microbial environment within the vagina. Vaginitis, which is a common problem for women from menarche through menopause, can have many causes and origins that are not fully understood. Vaginitis can be a symptom of an STI, a disruption in the normal vaginal flora, or simple transient irritation of the tissue. Recurrent vaginitis can have many causes and warrants a thorough sexual and lifestyle history that includes evaluation of the woman's overall health and well-being, including information about behaviors of the sexual partner. While partner transmission of noninfectious vaginitis is not common, female and uncircumcised male partners may act as a bacterial reservoir, reinoculating susceptible women. Common vaginitis conditions include bacterial vaginosis, vulvovaginal candidiasis, and atrophic vaginitis.

Bacterial Vaginosis

Bacterial vaginosis (BV) is the most common cause of vaginal infections in childbearing women[6] (**Figure 14-1**). Formerly known as *Gardnerella vaginalis*, *Haemophilus vaginalis*, or *Corynebacterium vaginitis*, BV is a dysbiosis of vaginal bacteria.[12] In essence, this infection represents a non-inflammatory disturbance in the vaginal microflora—which is why it is called *vaginosis*, rather than *vaginitis*. During episodes of BV, the vaginal flora is shifted toward a preponderance of anaerobic bacteria; these bacteria form a biofilm that adheres to vaginal epithelial cells.[13] In addition, production of volatile amines increases, and vaginal pH increases to more than 4.5.[14,15] Collectively, these changes result in a depletion of *Lactobacillus*.

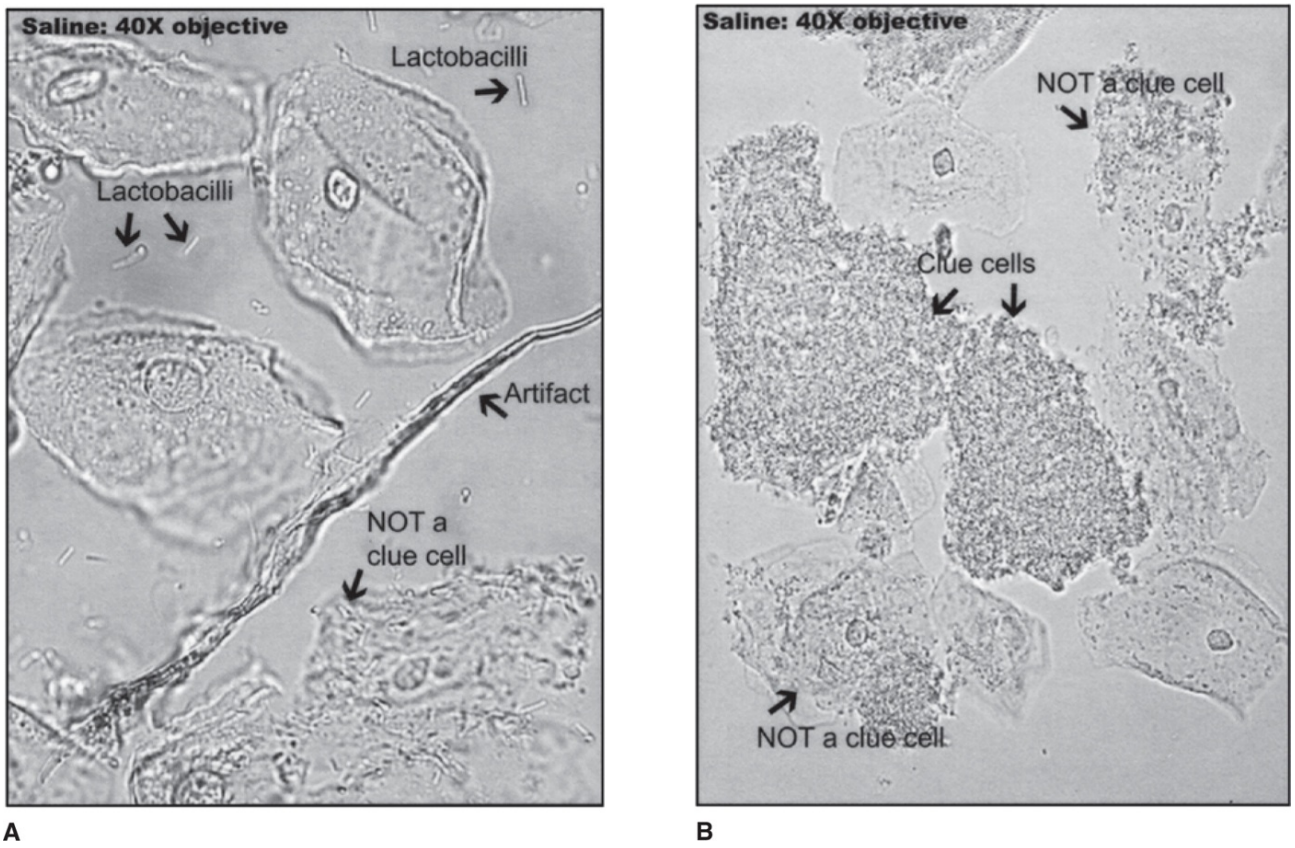


Figure 14-1 Microscopic diagnosis of bacterial vaginosis (BV). **A.** No BV; note the normal epithelial cells and presence of lactobacilli. **B.** BV; note the clue cells and lack of lactobacilli.
Reproduced with permission from Seattle STD/HIV Prevention Training Center and Cindy Fennell, MS, MT, ASCP.

Although the exact cause of bacterial vaginosis is not known, many factors are associated with its development, including smoking, menstruation, douching, sexual contact without a condom, low level of education, and engaging in oral or anal sex. This bacterial imbalance is associated with sexual contact, yet BV is not generally thought to be spread through sexual contact alone.[12-14] For example, the alkaline properties of semen may facilitate the vaginal flora's shift toward a predominance of anaerobic bacteria. Menopausal women have an increased risk for BV as a consequence of the decrease in healthy vaginal flora noted in

menopause. Women who have sex with women have increased prevalence of BV, and women of African descent have a higher prevalence of BV than women of other races, for unknown reasons.[12-14]

Bacterial vaginosis is associated with many adverse health outcomes, including preterm birth, postoperative infections, endometritis following pregnancy, and acquisition of other STIs.[15] However, this association is not fully understood, and treating BV does not prevent or mitigate the risk of associated adverse outcomes.[7,11,15]

Diagnosis of Bacterial Vaginosis

Symptoms of BV include vaginal irritation and itching, dyspareunia, gray or white discharge, and a "fishy" odor that is often most noticeable after vaginal penetration during sexual activity. Nevertheless, BV is asymptomatic in 75% to 85% of women.[15] On speculum examination, BV is usually evident as a thin white/gray homogenous discharge, irritated vaginal mucosa and introitus, and possibly cervicitis.

Gram stain with use of the Nugent scoring system is the gold standard for diagnosis of BV but is rarely available in outpatient settings. This test is time-consuming and requires laboratory equipment that is not always available in a clinic.[16-18] To make the diagnosis via clinical microscopy, a sample of the vaginal discharge is collected with a swab as described in the appendix *Collecting Urinary, Vaginal, Cervical, and Rectal Specimens for Testing and Interpretation of Saline and KOH Slides*, in the *Introduction to the Care of Women* chapter. The sample is first tested to determine the pH, and then a sample of the discharge is placed on two slides. Normal saline is added to one slide, which is covered with a slide cover and then set aside for microscopy. Finally, potassium hydroxide (KOH) is added to the other slide, which is used immediately for the "whiff test."

Studies have confirmed the sensitivity and specificity of individual components of Amsel's criteria for diagnosis of BV.[15-18] Clinically, BV is diagnosed when three of four of Amsel's criteria are present:

1. Presence of a thin homogenous discharge that adheres to vaginal walls
2. Presence of clue cells on the normal saline–prepared slide
3. pH of the vagina or vaginal discharge is 4.5 or higher
4. Positive "whiff test," which signals the release of an amine "fishy" odor when vaginal discharge contacts alkaline KOH

Alternatively, BV can be diagnosed with commercially available tests such as Affirm VP III, a DNA probe for *Gardnerella vaginalis*, or the OSOM BV Blue Test, which detects an enzyme produced by common BV-related organisms. Both of these tests are approved for use in making the diagnosis of BV.[19] While the sensitivity and specificity of these tests for diagnosing BV are better than that of Amsel's criteria alone, they are also more expensive.[19] Other commercial tests are available but have not been endorsed by the CDC due to their low sensitivity and specificity or lack of validation.[7]

Screening and Treatment of Bacterial Vaginosis

Routine screening for BV is not recommended, but all women who are symptomatic should be tested and offered treatment if the diagnosis is confirmed. **Table 14-3** outlines pharmacologic treatments for BV.[7,20] The choices for treatment include oral medications such as metronidazole (Flagyl), clindamycin (Cleocin), and tinidazole (Tindamax), as well as vaginal medications such as metronidazole gel or clindamycin cream or ovules. Selection of a specific agent should be part of the shared decision-making process, as the drugs have different side effects. For example, drinking alcohol during treatment with metronidazole and for 24 hours afterward can cause severe nausea and vomiting. Vaginal creams may be perceived as messy, and those containing clindamycin can weaken latex condoms and diaphragms. To ensure the treatment is compatible with her lifestyle, the plan of care must be developed in partnership with the affected woman.

Table 14-3 Treatment for Bacterial Vaginosis

Recommended Treatments for Nonpregnant Women Generic (Brand)	Alternative Treatments for Nonpregnant Women Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women Generic (Brand)
Metronidazole (Flagyl) 500 mg orally twice a day for 7 days	Tinidazole (Tindamax) 2 g orally for 2 days	Metronidazole (Flagyl) 500 mg orally twice a day for 7 days	Vaginal administration of metronidazole (Flagyl) is preferred for breastfeeding women.
Metronidazole gel (MetroGel) 0.75%, one applicator full per vagina at bedtime for 5 days	Tinidazole (Tindamax) 1g orally for 5 days	Metronidazole (Flagyl) 250 mg orally three times a day for 7 days	Metronidazole (Flagyl) taken orally by nursing women is excreted in breast milk and has active metabolites, increasing infants' exposure, but has not been demonstrated to be harmful. Timing the dose after nursing may decrease infant exposure, or women may discontinue breastfeeding for 12–24 hours after taking the medication.
Clindamycin (Cleocin) cream 2%, one applicator full per vagina at bedtime for 7 days	Clindamycin (Cleocin) 300 mg orally for 7 days	Clindamycin (Cleocin) 300 mg orally for 7 days	
	Clindamycin (Cleocin) ovules 100 mg intravaginally at bedtime for 3 days	Clindamycin (Cleocin) cream 2%, one applicator full per vagina at bedtime for 7 days	

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

Some studies have shown that probiotics, especially *Lactobacillus crispatus*, can be helpful

in establishing a normal vaginal flora and reducing BV recurrence.[12,21] However, the safety and effectiveness of these therapies have not been established.

Health education includes the recommendation to abstain from vaginal intercourse during treatment and wash all objects before they touch the vagina. More information related to maintaining healthy vulvar skin and the vaginal microbiome can be found in **Table 14-2**. Receptive oral and anal sex may increase the risk for BV related to microbial inoculation. Condoms may reduce the risk of BV by preventing contact with alkaline semen.

Until a normal vaginal flora is reestablished, relapses are common, especially after menses. Relapses occur in part because the anaerobic bacteria involved in BV form a biofilm that resists treatment.[12] After initial treatment, follow-up with another evaluation is recommended only if symptoms persist or reoccur. Chronic reinfection warrants further investigation and more intense treatments as outlined by the CDC.

Management of Bacterial Vaginosis During the Perinatal Period

Bacterial vaginosis is associated with an increased incidence of preterm birth.[15] However, treatment of BV during pregnancy with metronidazole (Flagyl) does not lower the incidence of preterm birth and, in fact, may increase the preterm birth rate via one of several mechanisms.[22] Therefore, as in nonpregnant women, routine screening for BV is not recommended, but diagnosis and treatment are recommended for women who are symptomatic.

Vulvovaginal Candidiasis

Candidiasis is an overgrowth of a *Candida* (yeast) species that affects the vagina, vulva, groin, and other moist areas of the body. The most common cause is *Candida albicans*, but other species may also cause this STI and are harder to treat. While candidiasis can be found in many locations on and in the body, this section focuses on infections in the female genital region. Seventy-five percent of women will have vulvovaginal candidiasis (VVC), commonly known as a yeast infection, at some point during their life.[7]

VVC is classified as uncomplicated or complicated. Uncomplicated VVC is a common condition that occurs sporadically throughout a woman's life and is associated with mild to moderate symptoms and manifestations. If infections occur more than four times per year, produce severe symptoms, or occur in women who are immunocompromised, VVC is classified as complicated and requires more intensive treatment.[7] Recurrent VVC is more likely to involve different *Candida* species, such as *C. glabrata*, that are not responsive to conventional antimycotic treatments.[7]

Several factors can make conditions favorable for *Candida* growth and increase a woman's risk of developing candidiasis, such as wearing nonbreathable clothes or being in very humid or warm living conditions. The hormonal changes that occur during pregnancy and while taking oral contraceptive pills also increase risk. In addition, diabetes is associated with increased incidence of candidiasis. Antibiotics administered to treat any infection may alter the vaginal flora, thereby facilitating proliferation of *Candida*. Immunosuppressed states, like those seen in persons with human immunodeficiency virus (HIV) and persons using corticosteroids, can also increase the risk of developing candidiasis in the vulvovaginal region and other moist areas of the body. Although species of *Candida* can be passed between sexual partners, candidiasis involves an overgrowth of normal flora and is not contagious.

Diagnosis of Vulvovaginal Candidiasis

Symptoms of candidiasis include vaginal itching, burning, irritation, dyspareunia, and increased vaginal discharge. The vulva may be erythematous, be slightly swollen, and have areas of redness with 1- to 2-mm "satellite" lesions extending from the affected area on external examination. The vagina is often red and slightly edematous. The vaginal discharge is usually thick, white, and curd-like, but can also be thin and watery, or adherent to the vaginal walls. Douching can complicate the physical examination by washing away discharge. Manifestations of severe candidiasis include widespread and severe erythema, skin fissures, edema, and excoriations.

A wet mount may reveal a lack of lactobacilli on a saline preparation or the presence of hyphae and pseudohyphae with a saline or KOH preparation (**Figure 14-2**). However, lack of hyphae does not rule out candidiasis, especially for women experiencing recurrent or severe symptoms, as the species of *Candida* implicated in recurrent and infection—*C. glabrata*—does not form hyphae. A culture can be performed in the absence of hyphae to check for non-*albicans* varieties of yeast. Treatment can begin without waiting for culture results.

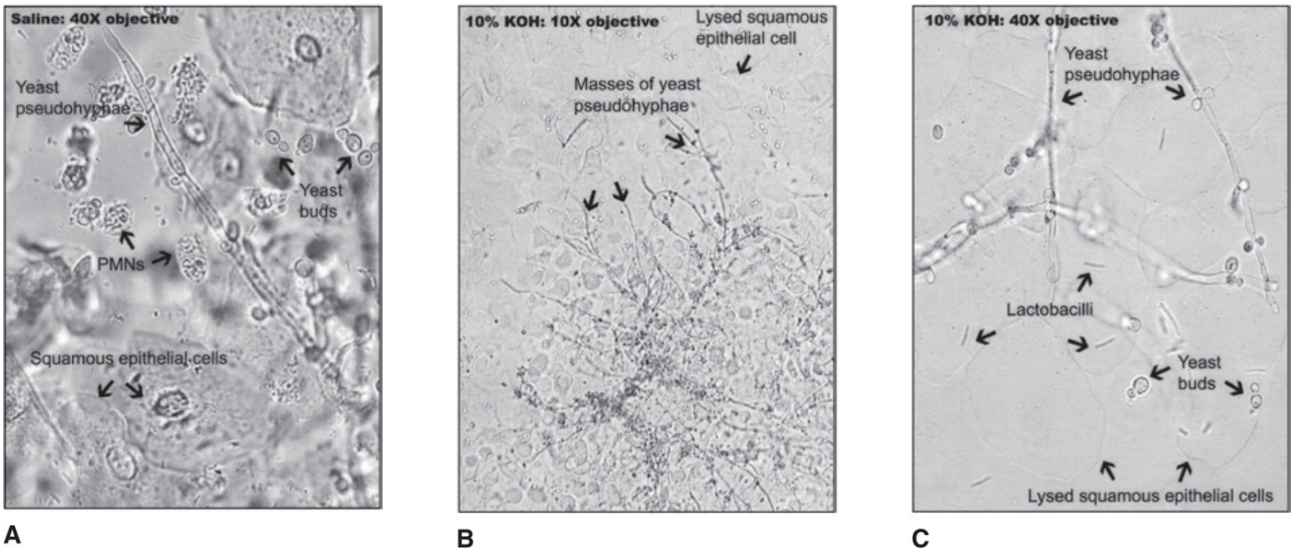


Figure 14-2 *Candida* pseudohyphae and budding spores under microscopic examination. **A.** Saline, 40×. **B.** KOH, 10×. **C.** KOH, 40×. Abbreviations: KOH, potassium hydroxide; PMNS, polymorphonuclear cells.
Reproduced with permission from Seattle STD/HIV Prevention Training Center and Cindy Fennell, MS, MT, ASCP.

Screening and Treatment of Vulvovaginal Candidiasis

Routine screening for VVC is not recommended. Treatment options for VVC are listed in **Table 14-4**. Many of these treatments are available without a prescription, and women may use the medications prior to a clinical diagnosis of candidiasis. There is no standard recommendation about being seen by a healthcare provider prior to initiating treatment. Use of over-the-counter medications can decrease costs for women if they have VVC, but can also mean that women delay seeking treatment for harder-to-treat or more serious vaginal infections.

Table 14-4 **Recommended Treatment for Uncomplicated Vulvovaginal Candidiasis**

Agents	OTC or Rx	Brand Name	Dose	Duration
Intravaginal				
Butoconazole 2% cream (single dose bioadhesive patch)	Rx	Gynazole-1	5 g intravaginally once at night	Single dose
Clotrimazole 1% cream	OTC	Gyne-Lotrimin-7	5 g intravaginally once at night	7–14 days[a]
Clotrimazole 2% cream	OTC	Gyne-Lotrimin-3 or Clotrimazole-3	5 g intravaginally	3 days
Miconazole 2% cream	OTC	Monistat-7	5 g intravaginally once at night	7 days[a]
Miconazole 4% cream	OTC	Miconazole-3	5 g intravaginally	3 days
Miconazole 100 mg suppository	OTC	Monistat-7	1 suppository intravaginally once at night	7 days
Miconazole 200 mg suppository	OTC	Monistat-3	1 suppository intravaginally once at night	3 days
Miconazole 1200 mg suppository	OTC	Monistat-1	1 suppository	Single dose
Terconazole 0.4% cream (45 g)	Rx	Terazol-7	5 g intravaginally once at night	7 days
Terconazole 0.8% cream (30 g)	Rx	Terazol-3	5 g intravaginally once at night	3 days
Terconazole 80 mg suppository	Rx	Terazol-3	1 suppository intravaginally once at night	3 days
Oral				
Fluconazole	Rx	Diflucan	150 mg oral tablet once at night	Single

Abbreviations: OTC, over-the-counter; Rx, prescription.

[a] A 7-day course is recommended during pregnancy.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017.[7]

The class of medications used to treat VVC are antifungal agents that work by interfering with the formation of fungal cell membranes. Options include oral treatments, vaginal creams and tablets, and topical preparations. The costs and side effects of these therapies vary. All locations of infection (vaginal and/or vulva) should be treated. The creams require vaginal insertion, and they may weaken condoms. The oral medication fluconazole (Diflucan) does not require handling of the genitals but has systemic side effects including alterations in hepatic function and several drug–drug interactions.[7]

Complicated VVC in nonpregnant women, which includes skin fissures, should be treated with a longer treatment course of either 7 to 14 days of a topical “azole” or two doses of 150 mg fluconazole, 72 hours apart.[7] If the vagina is very irritated, topical creams may aggravate the discomfort experienced by some women. For the midwife treating a woman with candidiasis, it is important to involve the woman in choosing the route of administration and

medication.

Probiotics, especially oral *Lactobacillus*, may help to restore normal vaginal flora, but conclusive evidence is lacking in part due to variations in probiotic composition.[12] Douching is not a recommended treatment. Health education includes methods to decrease yeast growth by reducing vulvar moisture and eliminating irritants. Additional health information is found in **Table 14-5** later in this chapter. Some complementary medicine sources recommend lifestyle changes, including decreasing consumption of refined sugars and yeast products. While these strategies have minimal potential for harm, they have not been substantiated as beneficial in otherwise healthy women. There is no need for a test of cure if symptoms resolve; women should be rescreened only if they are symptomatic.

Recurrent Candidiasis

Recurrent candidiasis is defined as four or more occurrences of candidiasis within one year.[7] If a woman has recurrent candidiasis, assess for contributing lifestyle factors, including those related to care of the vulva, and perform a culture to test for non-*albicans Candida* strains. Testing for underlying diabetes or HIV infection may be indicated depending on the woman's history and risk factors. If the woman has severe, recurrent infections, she may consider changing her sexual practices or changing her birth control method to avoid spermicides and oral contraceptive pills.

A variety of treatment regimens for recurrent candidiasis are available. For recurrence of infection with *C. albicans* strains, longer duration of topical treatment combined with sequential oral treatment may be considered.[7] For non-*albicans* yeast, pharmaceutical-grade boric acid administered as a gelatin capsule that contains 600 mg; this capsule is inserted into the vagina once daily for 2 weeks.[7,23] Alternatively, 100,000 units of Nystatin (Mycolog) administered as a suppository per vagina for 14 days followed by a maintenance regime can be therapeutic.[7,23] Suppressive therapy can be considered once the infection is controlled and should be based on the type of yeast species.[7,23]

Atrophic Vaginitis

Atrophic vaginitis is a collection of vaginal symptoms related to low estrogen levels. Lower estrogen levels can cause vaginal changes, including decreased collagen and adipose tissue, and increased pH.[24] These changes make the vagina more friable and prone to irritation, and increase the risk of vaginal infections. Low estrogen levels can occur with natural menopause or cycle cessation during breastfeeding, chemotherapy, or treatment with gonadotropin-releasing hormone (GnRH) agonists such as leuprolide (Lupron). While not an infection, these changes place women at increased risk for other infections such as BV, candidiasis, and STIs.

Vaginal changes in menopause are normal and do not require intervention unless they adversely impact the woman's functioning or quality of life. Some women may be reluctant to disclose such problems, so questions about vaginal dryness, irritation, and dyspareunia are important history components in women who are menopausal or not having regular menses. Potential symptoms of atrophic vaginitis include vaginal irritation and dryness, frequent vaginal infections, dyspareunia, and lack of lubrication with sexual activity.

Diagnosis of atrophic vaginitis is made via physical examination. Factors that suggest diagnosis include thinning vulvar skin; decreasing prominence of the inner labia; small, nonelastic vaginal introitus; few vaginal rugae; pale pink vaginal walls; shortening of the vagina; and a lack of vaginal moisture. The woman's vaginal pH may be higher than 4.5.[24]

Over-the-counter, water-based vaginal moisturizers that contain polycarbophil and are mildly acidic, such as Replens, can decrease symptoms when used daily and are appropriate for most women. Use of water-based lubricant products such as Astroglide, K-Y Jelly, or K-Y Liquibeads during sexual activity can decrease discomfort. Regular sexual arousal, and sexual intercourse, can be helpful in improving vaginal blood flow to increase tissue health.[24] Other nonpharmacologic recommendations for mitigating symptoms of atrophic vaginitis can be found in **Table 14-2**.

Estrogen can also be used to alleviate symptoms of atrophic vaginitis. Topical estrogen administration via vaginal ring or cream is preferable to oral administration as a first-line pharmacologic treatment.[24] When used topically, estrogen does not need to be paired with progesterone in women with an intact uterus.[25] However, long-term data on the safety of topical estrogens are not available.[25] Some research supports the hypothesis that endometrial hyperplasia can occur with topical estrogen administration, especially with creams.[25]

If topical treatments are not sufficient to relieve severe symptoms, systemic estrogen or estrogen/progesterone can be used by menopausal or postmenopausal women, often in combination with topical estrogen.[24,25] However, oral estrogens have more side effects, including increased risk of ischemic stroke and venous thromboembolism. Breastfeeding women and women who are not cycling secondary to healthcare treatments should not be given systemic estrogens. If a woman has a history of cancer, she should consult a cancer specialist before using any estrogen-containing therapies.

Other Causes of Vaginitis

The skin of the vulva and vagina can become inflamed in response to many substances. For example, exposure to urine or stool, such as occurs with even mild incontinence, can cause chronic irritation and vaginitis. Perfumes and chemicals found in soaps, laundry detergents, and feminine hygiene products can also cause irritation. Personal lubricants can disrupt vaginal pH and normal flora, resulting in vaginitis. Spermicides may irritate the vagina as well. Douching washes away beneficial bacteria and irritates the vaginal mucosa, increasing the risk for vaginitis. A thorough exploration of all substances that contact the vulva and vagina is warranted for women who have unexplained or recurrent vaginitis. Women with incontinence should strive to keep the vulva dry and free of urine or stool, cleaning this area as needed with water and soft, nonabrasive wipes.

Anything left within the vagina can also cause vaginitis—including tampons, pessaries, or any other foreign body. Odor and irritation are often the presenting symptoms when a foreign object is in the vagina for longer than recommended use. A speculum examination can identify the cause of the odor in this situation. On speculum examination, a tampon may at first appear to be a fleshy mass, as it may blend in with surrounding tissue. Forgotten tampons have an extreme odor; thus a plastic bag or container with water should be prepared before their removal and the tampon immediately submerged as soon as it is removed. Tampons can be removed with ring forceps or digitally. Pessaries, when left in the vagina for long time periods, can become irritating, especially if the woman has atrophic vaginal changes. While an increase in vaginal discharge is normal with pessary use, it is important to assess for vaginal infections and skin erosions if the woman reports problems or discomfort.

Vaginal irritation can also occur during intercourse if the woman is not well lubricated or the contact is rough or damaging; this finding might be indicative of relationship problems and needs further exploration.

Sexually Transmitted Infections

Because sexual contact involves close bodily proximity, many microbes are shared between partners, most of which are nonpathogenic. The close contact of mucous membranes and the sharing of bodily fluids during such contact allow several pathogenic organisms the opportunity to find a new host. STIs may be caused by bacterial, viral, protozoal, or parasitic organisms, as shown in **Table 14-5**. Some clinicians, however, prefer to organize their list of differential diagnoses according to the woman's chief concern, as shown in **Table 14-6**. Many STIs are asymptomatic even while causing bodily damage. Anyone at risk for STIs should be screened according to CDC guidelines to prevent health sequelae and transmission to others. Pre-exposure prophylaxis for HIV or vaccination against hepatitis B may also be appropriate if women engage in at-risk behaviors.

Table 14-5 **Causes of Sexually Transmitted Infections**

Bacterial Disease (Organism)	Virus (Abbreviation)	Protozoal and Parasitic Disease (Organism)
Chlamydia (*Chlamydia trachomatis*)	Human papillomavirus (HPV)	Trichomoniasis (*Trichomonas vaginalis*)
Gonorrhea (*Neisseria gonorrhoeae*)	Herpes simplex virus (HSV-1 and HSV-2)	Pubic lice/crabs (*Phthirus pubis*)
Syphilis (*Treponema pallidum)*	Human immunodeficiency virus (HIV)	Scabies (*Sarcoptes scabiei*)
Chancroid (*Haemophilus ducreyi*)	Zika virus	
Lymphogranuloma venereum (*Chlamydia trachomatis* serovars L1, L2, and L3)		
Granuloma inguinale (*Klebsiella granulomatis*)		
Molluscum contagiosum		

Table 14-6 **Common Presenting Concerns for Sexually Transmitted Infections**

Increased Discharge (Organism)	Sores (Organism or Virus)	Lesions (Organism or Virus)
Chlamydia (*Chlamydia trachomatis*)	Herpes (herpes simplex virus [HSV-1 and HSV-2])	Genital warts (human papillomavirus)
Gonorrhea (*Neisseria gonorrhoeae*)	Syphilis (*Treponema pallidum)*	Condyloma acuminata (human papillomavirus)
Trichomoniasis (*Trichomonas vaginalis*)	Chancroid (*Haemophilus ducreyi*)	Condylomata lata (*Treponema pallidum*)
	Lymphogranuloma venereum (*Chlamydia trachomatis* serovars L1, L2, and L3)	
	Granuloma inguinale (*Klebsiella granulomatis*)	

General Management Considerations Specific to Sexually Transmitted Infections

When women have symptoms of an STI or possible exposure to an STI, management includes some unique approaches and special considerations.

Screening Strategies: Opt-in Versus Opt-out

Screening for any disorder can be offered in one of two ways. *"Opt-in"* (voluntary) refers to the individual being counseled about a test and then choosing or agreeing to have that screening test performed. *"Opt-out"* (universal) is a strategy in which the test is presented as part of standard care and unless the woman declines, the test is performed. Midwives using an "opt-in" approach ask women about STI tests individually and obtain consent to screen for each infection. This approach presents STI screening as routine, thereby normalizing testing, but it results in much lower rates of STI screening, as women are more likely to decline testing. By comparison, the opt-out approach, which has been studied with regard to HIV screening, greatly increases screening rates for HIV both during pregnancy and when people enter the hospital for care.[7]

The CDC advises clinicians to use an opt-out approach that presents women with a consent form that lists all of the common tests and allows them to opt out of any tests if they wish. Women should be informed of recommended tests and allowed to "opt out" of testing that is routinely recommended.[7] An opt-out approach to STI testing decreases barriers and presents these screening tests as tests that are routinely offered to all women.

Reportable Diseases

Most states in the United States require healthcare providers to notify the local health department when a person is diagnosed with certain communicable sexually transmitted diseases, as shown in **Table 14-7**.[26] Notifiable diseases are those for which obtaining information regarding individual cases is important for prevention and control of that disease. Reporting usually involves revealing the individual's name to the local health department and indicating whether the sexual partner was treated. The woman should be told that this report will take place. This information is used to notify and treat sexual partners and to monitor prevalence of STIs on local, state, and national levels.[9] U.S. law requires that the spouse (current or within the past 10 years) of a person who is positive for HIV be notified, and the health department can assist in this endeavor.[27] Midwives should be aware of the laws concerning partner notification in their state or legal jurisdiction.

Table 14-7 **Sexually Transmitted Infections Reportable for National Surveillance**

Chancroid
Chlamydia trachomatis
Gonorrhea

Hepatitis B
Hepatitis C
Human immunodeficiency virus (HIV)
Syphilis
Syphilis, congenital

Based on Adams D, Fullerton K, Jajosky R, et al. Summary of notifiable infectious diseases and conditions—United States, 2013. *MMWR.* 2015;62:1-122.[26]

Test of Cure, Test for Reinfection, and Pre-exposure or Postexposure Prophylaxis

Test of cure refers to the practice of reculturing a specimen from the site of initial infection to confirm that the infection is no longer present. A test of cure is recommended for some vaginal infections and STIs when the risk of therapy failure is well known and adverse outcomes are significant. A test of cure is performed shortly after the completion of therapy. In contrast, a *test for reinfection* is recommended when therapy is known to be highly effective but the partner may not be adequately treated. A test for reinfection is often performed a few months after completion of therapy.

Pre-exposure prophylaxis and *postexposure prophylaxis* may be recommended for women who are at risk for contracting HIV. These management strategies are reviewed in more detail in the section on HIV.

Expedited Partner Therapy

Treatment of the woman's sexual partner for STIs is within the scope of midwifery practice.[4] Partner treatment may involve examination and testing of the sexual partner or presumptive treatment without a clinic visit depending on the diagnosed STI. It is acceptable to treat male sexual partners for STIs without a physical examination—a practice known as *expedited partner therapy* (EPT).[28] EPT allows for rapid treatment of sexual partners, thereby reducing the risk of reinfection and decreasing healthcare costs. To prevent adverse outcomes from drug allergies, the clinician should provide information with the prescription about potential side effects and an allergy warning.

EPT has demonstrated positive outcomes for male sexual partners of adult women who have gonorrhea and/or chlamydia.[28] There is inadequate research to support EPT for trichomoniasis, however, and it is not well supported for use with partners of women younger than 19 years. Female partners of women with STIs should ideally be seen in the clinic to rule out pelvic inflammatory disease (PID). EPT should not be used to treat syphilis or long-term, chronic STIs such as herpes or HIV.[29]

EPT is a legally `sanctioned practice in 40 states, is potentially allowable in 8 states, and is expressly prohibited in 2 states.[28] Partner treatment is included in the American College of Nurse-Midwives' *Core Competencies for Basic Midwifery Practice*.[30] However, while a state may legally allow expedited partner treatment, some state licensure boards, such as boards of nursing, prohibit writing prescriptions for individuals who have not been evaluated

or who do not have a chart within the clinic of the prescribing provider. The CDC maintains an interactive database of state statutes concerning EPT that can provide guidance on pertinent state regulations.[31]

Indications for Sexually Transmitted Infection Screening

If a woman is concerned that she has been exposed to a STI, she should be offered tests for all STIs that are common in the geographical area, including those deemed most likely to occur based on her own or her partners' sexual behaviors.[7] Local and regional prevalence rates are used to determine which infections are prevalent in the region or in the woman's or her partner's area of origin.[6] The CDC provides a list of groups at increased risk for specific STIs.[7] The STI tests that are recommended by the CDC for women who are asymptomatic (pregnant or nonpregnant) are reviewed in **Table 14-8**.[7]

Table 14-8 CDC Screening Test Recommendations for STIs Among Asymptomatic Nonpregnant Women and Pregnant Women[a]

Infection	Nonpregnant Women	Pregnant Women
Chlamydia	Sexually active women younger than 25 years Sexually active women 25 years and older if at increased risk Rescreen for reinfection approximately 3 months after original treatment	All pregnant women younger than 25 years Pregnant women 25 years and older if at increased risk Rescreen for reinfection approximately 3 months after original treatment Test of cure 3–4 weeks after treatment Rescreen for reinfection during third trimester if younger than 25 or at risk
Gonorrhea	Sexually active women younger than 25 years Sexually active women 25 years and older if at increased risk Rescreen for reinfection approximately 3 months after original treatment	All pregnant women younger than 25 years Pregnant women 25 years and older if at increased risk Rescreen for reinfection approximately 3 months after original treatment
Hepatitis B	Women at increased risk	Test for HBsAG at first prenatal visit regardless of prior testing Rescreen for reinfection during intrapartum if at high risk
Hepatitis C	Women born between 1945 and 1965 Women at increased risk	Women born between 1945 and 1965 Women at increased risk
HIV	All women age 13–64 years with opt-out option[a] All women seeking STI evaluation	All pregnant women at the first prenatal visit with opt-out option Rescreen for reinfection during third trimester and during intrapartum if at high risk
HSV	Consider type-specific HSV serologic testing for women in high-prevalence settings (e.g., STI clinics) and those at risk (e.g., multiple sex partners)[a]	Although evidence does not support routine HSV-2 serologic screening tests for low-risk asymptomatic women, a type-specific test may be of value for women at risk of acquiring genital herpes during pregnancy[a]
Syphilis	Women at increased risk	All pregnant women at the first prenatal visit Rescreen for reinfection during third trimester and during intrapartum if at high risk
Trichomonas	Consider for women in high-prevalence settings (e.g., correctional facilities) and	Consider for women in high-prevalence settings (e.g., correctional facilities) and women at high risk (e.g., multiple

	women at high risk (e.g., multiple sex partners, drug use)[a]	sex partners, drug use)[a]

Abbreviations: CDC, Centers for Disease Control and Prevention; HBsAG, hepatitis B surface antigen; HIV, human immunodeficiency virus; HSV, herpes simplex virus; STI, sexually transmitted infection.

[a] Consideration based on evidence but not a CDC recommendation.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017.[7]

Recommended screening tests for STIs, for women of all ages, are discussed in greater detail in the *Health Promotion and Health Maintenance* and the *Prenatal Care* chapters. Women who are unable to conceive due to known infertility, menopause, or sterilization may neglect STI precautions. *Any* woman at *any* age who has a new or nonmonogamous sexual partner is at risk for STIs and should be offered testing. It is important to guard against age bias in the decision to offer STI tests; rates of STIs among people older than 50 years have been increasing.[32]

Any woman with symptoms of an STI, including urethritis, abnormal vaginal discharge, dysuria, cervicitis, or cervical motion tenderness, should be evaluated with a thorough history, targeted physical examination, and indicated laboratory tests. If a woman has one STI, she is at risk for having another, and should be offered tests for HIV and all common STIs.[7]

Half of all STIs occur in persons younger than 25 years.[6] The CDC recommends all sexually active women younger than 25 years be offered screening tests for chlamydia and gonorrhea annually.[7] In addition, they should be offered HIV screening (although the screening interval for HIV testing has not been determined) and other diagnostic tests if they are symptomatic. Pap testing with human papillomavirus (HPV) testing should begin at age 21 years, regardless of the number of sexual partners or age when the woman first became sexually active.

Adolescents can receive tests for STIs without parental notification or consent in all 50 states and the District of Columbia.[7] However, if a woman uses health insurance for payment, the services provided will be included on the insurance explanation of benefits, which could breach confidentiality.[7] Young women, especially those using parental insurance, should be informed about insurance reports. If the young woman wants to use parental insurance, it may be helpful to forewarn her parents that she was screened for STIs as a component of a recent healthcare visit. If this is not acceptable, it may be best to refer the woman to local resources for free or low-cost STI testing.

Women Who Have Sex with Women

Sex and sexual contact with female partners can pose a risk for STI transmission related to contact with skin, genitals, and oral and vaginal mucosa. While the prevalence of infections is different in women who have sex only with women, the guidelines for STI and cervical cancer testing do not change with the sex/gender of the partner.[7]

Sexually Transmitted Infections During Pregnancy

Sexually transmitted infections can adversely affect the embryo or fetus, causing a wide range of complications, including lifelong morbidity and intrauterine death. Early screening and treatment for STIs can prevent maternal and fetal/neonatal complications. For this reason, comprehensive STI testing should be performed during the first visit for pregnancy or as soon as possible. States often mandate the provision of these services within a set time frame. As a consequence, STI tests should not be delayed to coincide with other testing, such as routine second-trimester bloodwork. Women who have multiple sexual partners or women who are diagnosed with an STI during pregnancy should be rescreened in the third trimester or in labor if indicated. Women are rescreened during each pregnancy, regardless of interconceptional spacing.[7]

All pregnant women are offered testing for chlamydia and HIV using an opt-out approach in the first trimester, and women at high risk should be offered repeat screening tests in the third trimester. High-risk groups for chlamydia include women who have tested positive for chlamydia during the current pregnancy, who are younger than 25 years, or who have new, multiple, or nonmonogamous sexual partners. High-risk status for HIV is conferred by the following factors: living in a high-prevalence area, drug use, diagnosis of an STI during pregnancy, and new, multiple, nonmonogamous, or HIV-positive sexual partner(s). Even if screened earlier during pregnancy, women at risk for STIs should be rescreened in the third trimester or at the time of admission in labor. Some states mandate third-trimester testing for STIs unless the woman specifically declines such tests.[7]

Pregnant women who are considered to be at risk for gonorrhea and hepatitis C should be routinely screened for these STIs. At-risk groups for gonorrhea include individuals who have the following characteristics: living in an area with a high prevalence of gonorrhea, such as the southern United States; age younger than 25 years; a history of STIs; new or multiple sex partners; commercial sex work; drug use; and irregular condom use, which is common among pregnant women who may not consider the use of condoms for protection against STIs. Women who are at continued risk and those who acquire a risk factor over the course of pregnancy should be rescreened in the third trimester. The CDC recommends a one-time screening for hepatitis C for all adults born between 1945 and 1965. High-risk categories for hepatitis C include current or previous use of injected or intranasal drugs, obtaining an unregulated tattoo, and a history of blood transfusion or organ transplantation prior to 1992.

Regardless of their risk status, asymptomatic women do not need to be routinely screened for trichomoniasis or bacterial vaginosis during pregnancy. The degree to which identification and treatment of women with these conditions decreases adverse health outcomes and reduces the risk of preterm birth has been controversial. However, women should be tested if they have symptoms; if positive, they should be offered treatment following informed decision making based on health education about potential risks of preterm birth.[7]

Women Who Are Incarcerated

Women entering correctional facilities are a particularly vulnerable population, as the rates of STIs in this population are high.[33] Women who are incarcerated are more likely than those who are not to have been abused or exchanged sexual contact for food, housing, or money.[33] The

CDC recommends that on intake to a correctional facility and following consent, all females younger than 35 years be screened for gonorrhea, chlamydia, and HIV.[7] Syphilis screening in this population is based on local prevalence.[7]

Communicating Information About Sexually Transmitted Infections

Telling a woman that she has an STI is often difficult for both novice and experienced practitioners. The CDC advises clinicians to discuss positive HIV testing in person.[7] However, for STIs other than HIV, discussion of the diagnosis and planning for treatment may occur via the phone if an in-person visit is not feasible. Phone consultations are acceptable, but not ideal, as it is more difficult for the midwife to gauge how the woman is responding to the diagnosis. However, phone visits can facilitate prompt treatment and decrease the chance that the woman is unable to receive treatment. Prior to phoning a woman with STI results, make sure she consented to be contacted by phone. Once she answers, ask if she is in an acceptable location for the discussion before beginning the conversation.

Maintaining a normal tone of voice without judgment is helpful in being present for the woman. It may take time for the woman to adjust to the news that she has an STI. Openly discuss the fact that the infection is sexually transmitted and address whether it is possible for the infection to be transmitted in other ways, such as through common contact. While nonsexual transmission is possible with some infections, such as scabies, it is very unlikely for others, such as gonorrhea. Some infections produce symptoms soon after initial infection, whereas others, such as HPV, may not manifest for months or years. Avoid assumptions about the origin of the STI, and use open- and closed-ended questions to further explore the woman's feelings and needs. Always assess for the possibility of intimate-partner violence if the diagnosis is shared with a sexual partner. While health education to avoid future infections is needed, it can be difficult for a woman to concentrate on such health-promoting messages immediately after receiving an STI diagnosis. Written materials that the woman can take home will allow her to review the information when she is more receptive to it. A well-placed follow-up visit in person or by phone may be a better time for additional health education if the woman seems overwhelmed at the time of diagnosis.

Relationship Stressors and Violence

STIs can be associated with intimate-partner relationship difficulties. Although many STIs are acquired from a new partner who was previously infected, a new STI diagnosis may reveal infidelity of a long-term sexual partner or may be an indicator that the woman has been forced to have sex. Screening for human trafficking is warranted if the woman has more than one STI, an advanced case of the infection, or other risk factors for nonconsensual sex.[34] A diagnosis of an STI can spark or enhance relationship tension that may lead to negative emotions, cessation of the relationship, or even violence.[35] Even so, partner notification and treatment have not been shown to greatly increase the rate of violence.[35]

Psychosocial Needs

Women with STIs are more likely to be from vulnerable social groups. Being young, being poor, and using drugs are all risks for STI acquisition.[6] Sometimes sexual activity is one of

many risk-taking behaviors. In other cases, women did not have a choice about engaging in sexual contact. Women who are abused or forced into sexual encounters may not be able to advocate for condom use. Negotiating for safer sexual practices requires self-confidence and power within the relationship. Obtaining condoms requires money or access to services, which may be difficult to obtain for women of low socioeconomic status and those who do not have a support network. Women who trade sex for food, housing, drugs, or money or who are victims of trafficking are more likely to acquire STIs, as they often have multiple sexual partners who have little concern for the woman's health.[5] Women often enter care for one reason but have other needs besides the chief concern and can benefit from a holistic approach that provides them with needed information or referrals beyond their presenting diagnosis.

Contraception

Women who are sexually active with men are at risk for pregnancy. The time at which a woman is diagnosed with an STI presents an excellent opportunity to discuss her reproductive life plan. A critical first step in this discussion is to determine the woman's intentions regarding pregnancy. Straightforward questioning about whether she is planning a pregnancy in the near future can provide the midwife with answers that guide the discussion. For women who are planning a pregnancy, the midwife can use this opportunity to discuss the importance of pregnancy prevention until STI treatment is complete and returning for a test of cure if one is indicated.

When a woman is not planning a pregnancy in the near future, it is essential to review her contraceptive options and allow her to choose a method that provides prevention of unintended pregnancy as well as protection from STIs. Whereas both male and female condoms are part of safer sex practices, other birth control methods offer little protection from STI acquisition. However, condoms have a fairly high failure rate for pregnancy prevention and may be an inadequate primary contraceptive method. A woman may need to use condoms in addition to other contraceptive methods to prevent STIs and may need to be reminded of the importance of safer sexual practices even when she is not concerned about pregnancy prevention. Some women need guidance on how to negotiate for safer sexual practices even if they are protected from pregnancy.

Evaluation of the Need for Immediate Intervention, Consultation, or Collaboration

The treatment of women with STIs often involves interprofessional collaboration. Local pharmacists, physicians, and health department staff may be involved in this process. If the woman has a disseminated or advanced STI, hospitalization and consultation with physicians, pharmacists, and infectious disease specialists may be appropriate. Treatment failures may necessitate consultation with regional health departments or the CDC. Pediatric care providers need to know about any STIs in the pregnant or laboring woman that may affect her newborn or infant. Judicious use of other members of the interprofessional healthcare team can improve the quality of the care received while protecting a woman's rights and dignity. While consultation

may involve disclosing the woman's name and diagnosis, this should be done in ways that respect her rights and maintain her confidentiality. Care provider communication can facilitate quick and effective treatment, thereby preventing morbidity and mortality.[36]

Suspected sexual abuse of minors must be reported to local authorities for further investigation. In addition, reporting of STIs to local health departments is a responsibility that does not violate confidentiality. While local health departments should not reveal the name of the "index patient," there is the chance that someone could guess which partner had an STI, and this recognition could have wide-ranging implications for the woman, including violence. Therefore, the CDC advises all health departments to ask women, without coercion, to provide information about previous sexual partners so they can be notified and treated.[8]

Development of a Comprehensive Plan of Care That Is Supported by a Valid Rationale

It is important to ensure that women are treated appropriately for STIs to prevent short- and long-term morbidity and to reduce transmission. Treatment options can be presented to the woman and a plan of care developed to meet her needs. Effectiveness, cost, and convenience are chief considerations in choosing a treatment. Dosing frequency, privacy, and ability to apply or wash off the medicine may also be important in the decision-making process.

Follow-up after STI diagnosis and treatment differs by infection. While it can be difficult for women to return to the clinic, appropriate and well-timed follow-up is important to decrease infection-related morbidity, prevent transmission, and detect drug-resistant strains early.[7]

Chlamydia

Chlamydia trachomatis is a small gram-negative bacterium that is an obligate intracellular organism. Chlamydia is the most common reportable STI, with more than 1.4 million new cases occurring each year in the United States.[6] The majority of these infections occur in individuals 25 years or younger, although any sexually active person is at risk.[6] Because the majority of cases of chlamydia are asymptomatic, the CDC recommends screening all women who have a new sexual partner and annual screening for women 25 years or younger.[7]

When left untreated, chlamydia can ascend into the upper reproductive tract and cause pelvic inflammatory disease. Ascending infections increase the risk of subsequent ectopic pregnancy and infertility. Urogenital transmission is the most common form of spread of this infection, but sexual transmission to the oropharynx and rectum is possible. Vertical transmission can occur during vaginal birth, causing conjunctivitis or pneumonia in affected neonates.

Although chlamydial infection in women is usually asymptomatic, increased vaginal discharge, dysuria, or Bartholin gland infection can be presenting symptoms. Some women are initially diagnosed when they present with salpingitis (infection in the fallopian tubes) or PID.

Screening and Diagnosis of Chlamydia

All women who are younger than 25 years, have new or multiple sex partners, a sex partner with concurrent partner or a sex partner with STI, history of inconsistent condom use if not in a mutually monogamous relationship, drug use, sex work, history of a previous STI, or living in a geographic area with a high prevalence of gonorrhea (i.e., the southeastern United States) should be screened for chlamydia annually.

Physical examination may reveal cervicitis, mucopurulent discharge, or cervical motion tenderness. Although an increased number of white blood cells may be detected on a wet prep, diagnosis of chlamydia is made based on laboratory tests. Nucleic acid amplification test (NAAT) is the most common diagnostic test for chlamydia; it can be performed on urine, cervical, vaginal, or liquid cytology specimens.[7] NAAT detects small amounts of gene sequences in bacterial DNA and replicates those sequences so that a large amount is present. Most samples sent to the laboratory for STI diagnosis are tested with NAAT technology. Such tests may also be used with rectal samples, depending on the manufacturer and laboratory. Urine testing is the least invasive option and has high sensitivity rates.[7] NAAT on liquid cytology (liquid Pap test) may have a lower sensitivity for detecting infection.

Treatment and Follow-up for Chlamydia

Pharmacologic treatments for chlamydia are listed in **Table 14-9**.[7] Single-dose treatment is preferable. Ensure treatment of all sexual partner(s) from the last 60 days.[7] The woman with chlamydia should abstain from intercourse until her partner(s) are treated and for 7 days after single-dose treatment or until she completes her multidose treatment.[7]

Table 14-9 Treatments for Chlamydia

Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Breastfeeding Considerations
Azithromycin (Zithromax, Z-Pak) 1 g orally once	Erythromycin base 500 mg orally four times a day for 7 days	Azithromycin (Zithromax, Z-Pak) 1 g orally once	Azithromycin is compatible with breastfeeding.
Doxycycline (Vibramycin) 100 mg orally twice a day for 7 days	Erythromycin ethylsuccinate (E-Mycin) 800 mg orally four times a day for 7 days	Alternative treatments for pregnant women: amoxicillin 500 mg orally three times a day for 7 days	There is theoretical potential that an infant's forming teeth will become stained if doxycycline is used by a breastfeeding woman. However, its levels in breast milk are low and short courses of doxycycline are acceptable. If doxycycline is needed, consider strategies to minimize its transfer to the milk.
	Levofloxacin (Levaquin) 500 mg orally for 7 days	Erythromycin base 500 mg orally four times a day for 7 days	
	Ofloxacin (Floxin) 300 mg orally twice a day for 7 days	Erythromycin base 250 mg orally four times a day for 14 days	
		Erythromycin ethylsuccinate (E-Mycin) 800 mg orally four times a day for 7 days	
		Erythromycin ethylsuccinate (E-Mycin) 400 mg orally four times a day for 14 days	

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

Women who have a positive test for chlamydia should be tested for all common STIs, as coinfections frequently occur.[7] Nonpregnant women should be retested 3 months after treatment or at the next possible outpatient visit.

Management of Chlamydia During the Perinatal Period

Women with a history of chlamydial infection are at increased risk for ectopic pregnancy. The

CDC recommends routine screening for chlamydia for all pregnant women as early as possible during pregnancy. Pregnant women at increased risk for STIs (e.g., those younger than 25 years, those with a new or nonmonogamous sexual partner during pregnancy) are rescreened in the third trimester.[7] Treatment occurs as soon as possible after diagnosis, using a treatment regimen compatible with pregnancy. Pregnant women should be retested no sooner than 3 weeks after treatment and then rescreened again 3 months later or in the third trimester, whichever comes first.[7]

Chlamydial ophthalmia can cause ocular scarring and blindness and/or life-threatening pneumonia.[7] Because routine neonatal ocular prophylaxis is only 80% effective against chlamydia, infants born to women who are infected with chlamydia should be closely observed for symptoms but not routinely treated.[7] Additional information about eye prophylaxis for newborns to prevent chlamydial and gonorrheal ophthalmia can be found in the *Neonatal Care* chapter.

Gonorrhea

Neisseria gonorrhoeae is a gram-negative intracellular diplococcus that exclusively affects humans. This bacterium primarily infects the mucocutaneous surfaces of the genitourinary tract, pharynx, conjunctiva, and rectum. Gonorrhea is the second most common reportable communicable disease in the United States. In recent years, there has been a surge in antibiotic resistance to this organism, and the organism has the potential to become resistant to all currently available antibiotics.[6] The CDC and WHO monitor infectious disease resistance patterns and release updates as needed. Clinicians are advised to use the most current guidelines for the geographic location of acquisition and treatment.

Transmission of gonorrhea occurs through oral, anal, and vaginal sex and via contact with secretions from the source individual's urogenital tract. Vertical infection is possible during vaginal birth. Individuals infected with gonorrhea can be asymptomatic or may experience a variety of symptoms. Women may have dysuria, abnormal vaginal discharge, or bleeding if infected vaginally. Oral infection can result in a sore throat. Anal infection can cause anal itching, soreness, bleeding, discharge, or painful bowel movements.[7] Disseminated gonorrhea is a rare, life-threatening condition characterized by symptoms of joint pain and a rash. Gonorrhea can increase the risk of HIV acquisition and is associated with PID, infertility, and ectopic pregnancy.[7] Women with PID that is caused by gonorrhea can have fever, vaginal discharge, and abdominal pain. (See the section on PID.)

Screening and Diagnosis of Gonorrhea

Women at risk for gonorrhea include all sexually active women younger than 25 years and women with new sexual partners, more than one sex partner, a sex partner with concurrent partners, or a sex partner who has an STI. Women at risk should be screened for gonorrhea annually.

The physical examination may be unremarkable in persons with gonorrhea. Conversely, lymph nodes surrounding the affected area may be enlarged. Urethritis and inflammation and discharge from the periurethral (Skene's) and greater vestibular (Bartholin) glands may be present. Cervicitis is common. If the woman has mucopurulent discharge from her cervix and uterus, adnexal tenderness, or cervical motion tenderness, PID is a likely diagnosis.

Diagnosis of gonorrhea is based on laboratory tests. However, if this infection is considered a likely diagnosis based on clinical findings, treatment can begin prior to knowing the results of testing. NAAT can be performed on endocervical, vaginal, or urine samples and is acceptable for initial screening and diagnosis of gonorrhea. NAAT products on the market have differing collection methods and sample requirements, so it is important to be familiar with the manufacturer's guidelines for each product. Cultures require an endocervical sample. If rectal, oropharyngeal, or ophthalmic testing is needed, ensure that the laboratory has tests that are specific to *N. gonorrhoeae*, as common bacteria present in these areas may cause false-positive results.

Treatment and Follow-up for Gonorrhea

Table 14-10 lists the recommended treatment regimens for uncomplicated gonorrhea infection. Ideally, treatment is provided on site and observed.[7] Women with gonorrhea are also frequently coinfected with chlamydia, so all CDC-approved treatments for gonorrhea include dual therapy treatment for chlamydia; this strategy is intended both to reduce the incidence of PID and to combat increasing antibiotic resistance.[7] Thus women with gonorrhea should be treated with azithromycin (Zithromax) in addition to ceftriaxone (Rocephin), even if they have a test for chlamydia that is negative.

Table 14-10 Treatments for Uncomplicated Gonorrhea of the Cervix, Urethra, Rectum, or Pharynx in Adults

Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women Generic (Brand)
Ceftriaxone (Rocephin) 250 mg orally in one intramuscular dose AND Azithromycin (Zithromax, Z-Pak) 1 g orally once	Use alternative only if unable to give ceftriaxone (Rocephin): Cefixime (Suprax) 400 mg orally in a single dose PLUS Azithromycin (Zithromax, Z-Pak) 1 g orally in a single dose	Same as for nonpregnant women	Same as for nonpregnant women

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

Alternative regimens are recommended only if ceftriaxone (Rocephin) is not available or if the woman is severely allergic to penicillin or cephalosporins. In such a case, a physician consultation should be obtained because the choice of cephalosporin needs to be individualized. The CDC website provides further guidance for unusual or complicated cases, such as treatment failure or disseminated gonorrhea.

Individuals receiving treatment for gonorrhea should be screened for other STIs, including syphilis, chlamydia, and HIV. They should abstain from sexual contact for 7 days after treatment and until all sexual partners are screened and treated. Otherwise, healthy nonpregnant adults with uncomplicated urogenital or rectal gonorrhea do not need a test of cure if treated with the ceftriaxone (Rocephin)/azithromycin (Zithromax) regimen. If treatment with ceftriaxone (Rocephin) was not possible or if the woman had pharyngeal gonorrhea, she will need a test of cure 2 weeks after finishing treatment using either NAAT or culture for the repeat test.

The CDC recommends rescreening for gonorrhea 3 months after treatment to test for reinfection.[7] If a 3-month rescreening is not possible, the woman should be screened the next time she presents for health care.[7] Most positive tests after treatment reflect reinfection; if treatment failure is suspected, a culture should be obtained, an infectious disease specialist or the CDC should be contacted, and the individual and her partner(s) should be immediately

treated using current CDC guidelines for treatment failures.[7] Pharyngeal gonorrhea is more prone to resistance than gonorrhea in other locations due to plasmid sharing with local microflora.[37]

Management of Gonorrhea During the Perinatal Period

Pregnant women at risk for gonorrhea should be offered a diagnostic test. Women with a history of possible exposure to gonorrhea can be offered treatment before the test results are available. Pregnant women should receive a test of cure following treatment and should be tested for reinfection in 3 months or during the third trimester.[7] If the woman is allergic to recommended treatments, consultation with an infectious disease specialist is recommended.[7]

Gonorrhea can be transmitted during birth and cause ophthalmia, localized infections of mucosa, abscesses at the site of a placed fetal heart rate electrode, or disseminated infection. Because gonococcal ophthalmia can result in permanent blindness, the CDC advises ophthalmia neonatorum prophylaxis using erythromycin ophthalmic ointment for *all* newborns, regardless of maternal testing status or route of birth; this prophylaxis is reviewed in more detail in the *Neonatal Care* chapter.[7] Many state laws mandate such treatment, and a parent must sign a waiver to opt out of treatment. Infants born to women with untreated gonorrhea or suspected gonorrhea should also receive parenteral antibiotics presumptively to prevent more severe infections.[7] Pediatric care providers should be notified at the time of birth if there is a concern the newborn has been exposed to gonorrhea.

Pelvic Inflammatory Disease

Pelvic inflammatory disease (PID) is the term used to refer to a spectrum of disorders that are characterized by inflammation in the upper female genital tract; these disorders include endometritis, salpingitis, tubo-ovarian abscess, and pelvic peritonitis. PID results from a variety of causative organisms that ascend from the vagina into the upper urogenital tract, where they induce an inflammatory response.[38] *N. gonorrhoeae* and *C. trachomatis* are common causative organisms of PID. Abnormal vaginal microflora, such as BV, may increase the risk that pathogens will ascend into the uterus and fallopian tubes, causing PID. The onset of PID symptoms often occurs after menses, as the transient change in vaginal microflora during menstruation facilitates pathogen ascension into the uterus.[12] A slight increase in the risk of PID is also noted in the first few weeks following insertion of an intrauterine device (IUD), as pathogenic bacteria may be deposited in the uterus during placement.[39] While many risk factors for PID have been identified, including young age, HIV infection, and multiple or new sexual partners, any sexually active woman is at risk of developing this disease.

PID has many adverse short- and long-term sequelae for women. The inflammation from even mild cases of PID can cause scarring and permanent damage to tubal cilia, thereby impairing fertility and greatly increasing the risk of ectopic pregnancy throughout the reproductive years. Women may also develop chronic pelvic pain from PID-related adhesions.[38] Perihepatitis and tubo-ovarian abscess are immediate, severe risks of untreated PID. Both of these conditions cause severe abdominal pain. These conditions warrant further testing and hospitalization.[7] Because PID has severe, long-term outcomes, the threshold for diagnosis and treatment is low.[7]

Diagnosis of Pelvic Inflammatory Disease

To make the diagnosis of PID, the woman must have pelvic or lower abdominal pain and at least one of the following: (1) cervical motion tenderness, (2) uterine tenderness, or (3) adnexal tenderness. Additional symptoms, such as fever and mucopurulent vaginal or cervical discharge, increase the specificity of the diagnosis. Invasive tests are rarely warranted, as definitive diagnosis is not necessary before treatment is recommended. However, ultrasound and laparoscopy can be useful to rule out other diagnoses, such as ectopic pregnancy or acute appendicitis.[38] **Table 14-11** provides the full diagnostic criteria for PID.[7] A physical examination is needed to rule out other causes of abdominal pain, including gastrointestinal problems, ovarian cysts, and appendicitis or other surgical emergencies.

Table 14-11	Diagnostic Criteria for Pelvic Inflammatory Disease
Minimum Criteria (pelvic or abdominal pain AND one or more of the following criteria needed for diagnosis)	
Cervical motion tenderness OR Uterine tenderness OR Adnexal tenderness	

Additional Criteria (increase specificity of diagnosis)
Fever > 38.3°C (101°F)
Mucopurulent cervical or vaginal discharge
Numerous white blood cells on saline wet prep
Elevated C-reactive protein
Elevated erythrocyte sedimentation rate
Documented infection with *C. trachomatis* or *N. gonorrhoeae*
Definitive Criteria (not needed to begin treatment, but beneficial to rule out other diagnoses)
Transvaginal ultrasound, magnetic resonance imaging, or Doppler studies showing thickened and fluid-filled tubes
Laparoscopic visualization of pelvic inflammatory disease-related abnormalities

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017.[7]

When a woman presents with symptoms of PID, NAAT should be performed for gonorrhea and chlamydia; HIV testing is also recommended. A wide range of additional tests may be performed depending on the woman's clinical situation and the likelihood of other diagnoses. A wet mount that shows many white blood cells is suggestive of PID. Clue cells, positive whiff test, and altered pH may be present. Blood tests may reveal an elevated white blood cell count and erythrocyte sedimentation rate. A urine or blood test for human chorionic gonadotropin (hCG) might be needed to rule out ectopic pregnancy.

Diagnosis of PID is based on clinical presentation alone. The CDC advises a low threshold for diagnosis, as PID may have minimal symptoms but still cause tubal damage.[7] Laboratory tests for gonorrhea and chlamydia should be performed, but treatment should not be delayed while awaiting their results.

Treatment of Pelvic Inflammatory Disease

Women with PID are treated with antibiotics that provide a broad spectrum of coverage for both aerobic and anaerobic organisms. A woman with PID should be presumptively treated for both gonorrhea and chlamydia because a negative endocervical culture for one of these organisms does not rule out their presence in the upper genital tract (**Table 14-12**). Metronidazole (Flagyl) should be strongly considered to treat possible anaerobic organisms that may cause PID.[7] Oral treatment is acceptable unless the woman has not responded to oral treatment or is acutely ill. If a woman has an IUD in place, she should be treated as usual and the IUD does not need to be removed. That said, if the woman does not improve between 48 and 72 hours, the IUD should be removed.[7,40]

Table 14-12 **Outpatient Treatments for Pelvic Inflammatory Disease[a]**

Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women
Ceftriaxone (Rocephin) 250 mg IM once AND Doxycycline (Vibramycin) 100 mg orally twice a day for 14 days WITH or WITHOUT (literature favors with) Metronidazole (Flagyl) 500 mg orally for 14 days (for parenteral or inpatient treatment, refer to CDC guidelines)	A third-generation cephalosporin—such as Cefixime (Suprax) 400 mg orally in a single dose PLUS Azithromycin (Zithromax, Z-Pak) 1 g once a week for 2 weeks OR doxycycline (Vibramycin) 100 mg orally twice a day for 14 days WITH or WITHOUT Metronidazole (Flagyl) 500 mg twice a day for 14 days	Pregnant women with suspected PID should be hospitalized for evaluation according to CDC guidelines.	Same as for nonbreastfeeding women. Doxycycline is generally considered to be safe with breastfeeding, but longer courses of the drug have the potential to stain infants' teeth. Consider methods to decrease infant ingestion or an alternative regimen. Metronidazole administered orally to the mother is excreted in breast milk and has active metabolites in the infant. While no harm has been shown in breastfeeding infants, the potential amount ingested by the infant should be considered.
Cefoxitin (Mefoxin) 2g IM once AND Probenecid (Benemid) 1g orally administered as a single dose AND Doxycycline (Vibramycin) 100 mg orally twice a day for 14 days WITH or WITHOUT Metronidazole (Flagyl) 500 mg orally for 14 days	If woman has a severe cephalosporin allergy: Azithromycin (Zithromax, Z-Pak) 2 g orally once WITH or WITHOUT Metronidazole (Flagyl) 500 mg twice daily for 14 days		Probenecid may increase the excretion of cimetidine into breast milk.

Abbreviations: CDC, Centers for Disease Control and Prevention; IM, intramuscular; PID, pelvic inflammatory disease.

[a] For inpatient treatments, see the CDC guidelines.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

The symptoms of PID should improve dramatically within 72 hours after initiation of treatment. If no improvement is evident within this time frame, the woman should be reevaluated, and hospitalized for intravenous antibiotics and supportive therapy. Hospitalization should also be considered if the woman is pregnant; if oral treatment is not possible; when the PID appears to be severe, such as in the presence of a high fever or nausea and vomiting; and when emergencies such as appendicitis, tubo-ovarian abscess, or conditions cannot be excluded.[7,38]

Sexual partners from the 60 days preceding the onset of the woman's symptoms of PID should be treated presumptively for gonorrhea and chlamydia.[7] Health education includes recommending abstinence from sexual intercourse until therapy is completed and sexual partners have been treated, use of condoms, and other practices to decrease STI transmission, including HIV pre-exposure prophylaxis (PrEP) if indicated. If tests were positive for gonorrhea or chlamydia, the woman should be retested at 3 months or the next time she seeks health care.

Management of Pelvic Inflammatory Disease During the Perinatal Period

Women with a history of PID are at increased risk for ectopic pregnancy, and can benefit from early prenatal care. Although PID is rare during pregnancy, probably because of the protective effects of the cervical mucus, pregnant women with PID are at high risk for preterm labor and maternal morbidity. Pregnant women should be hospitalized for evaluation and treatment.[7]

Syphilis

Caused by a spiral-shaped spirochete (*Treponema pallidum*), syphilis is known as the "Great Pretender" because it may be asymptomatic or present with a variety of symptoms mimicking other conditions. The incidence of syphilis has been increasing in recent years, with a 27% increase in reported cases in women who reside in the United States between 2014 and 2015 alone.[5] Globally, 18 million people are infected with syphilis.[41] Transmission occurs through oral, anal, or vaginal sexual contact. Condoms do not fully protect against syphilis if the condom material does not cover all lesions.

Approximately 50% of individuals exposed to *T. pallidum* will develop infection if not treated. The disease progresses through three primary phases: primary syphilis (chancre and regional lymphadenopathy), secondary syphilis (disseminated skin eruptions and generalized lymphadenopathy), and tertiary syphilis (cardiovascular syphilis, neurologic symptoms and gummas [granulomatous lesions in skin and bone]).[41] A latent form of syphilis can develop between the secondary and tertiary phases. The three phases can overlap somewhat, and neurologic manifestations of syphilis can occur at any time in the disease process. In addition, a newborn may present with congenital syphilis, as the *T. pallidum* organism readily crosses the placenta.

Primary syphilis is characterized by a chancre that appears at the site of inoculation approximately 21 days after exposure. The chancre is a nontender indurated ulcerous lesion. It is usually a single lesion, filled with spirochete-laden purulent discharge, and is highly infectious. The chancre begins as a papule that erodes into a well-demarcated area, with induration of the base and circumference. Because the chancre is usually painless and heals spontaneously in 2 to 8 weeks, it may not be noticed, especially if it is inside the vagina or rectum. The chancre is an open wound, so it increases the risk of the woman developing other infections to which she might be exposed, including HIV and herpes. In addition, secondary infections may occur within the chancre, resulting in purulent discharge.

Secondary syphilis marks the change from local to systemic infection. The manifestations of secondary syphilis usually appear 4 to 10 weeks after infection, and can reappear following periods of latency. The most characteristic symptom of this stage of syphilis is a rash that appears on the palms of the hands, soles of the feet, and trunk; this rash can be macular, papular, or psoriasiform. Other symptoms include patchy alopecia, condylomata lata, lesions of the mucous membranes, and symptoms of a systemic illness such as low-grade fever, sore throat, hoarseness, malaise, headache, anorexia, and generalized lymphadenopathy. Condylomata lata can also occur; these highly contagious, flat, moist, wart-like lesions usually appear in body folds such as the vulva and perianal area.[41]

After the symptoms of secondary syphilis resolve, some affected individuals convert to early or late latent syphilis before the symptoms of tertiary syphilis appear. Persons with latent syphilis have no clinical manifestations, but their infection can be detected with blood serology tests. Individuals with latent syphilis who have been infected within the past year are classified as having early latent syphilis.[7] The WHO uses infection within the past two years to define early latent infection.[41] Individuals infected for a year or more are classified as having late latent syphilis and will need a longer course of treatment.

Tertiary syphilis can appear anywhere from one to two years after infection to 30 or more years later. Today, this form of the disease is rare in the developed world. Associated with high morbidity and mortality, tertiary syphilis takes two forms: gumma and cardiovascular syphilis.[7,41] Gumma are soft-tissue granuloma tumors, which occur in tissues throughout the body. These masses cause extensive damage within the body and are difficult to distinguish from carcinomas. Cardiovascular syphilis may result in aortic valve disease, aortic aneurysm, and coronary artery disease.[41]

Neurosyphilis can occur during any stage of syphilis. For women in the United States, clinical symptoms of central nervous system disease in the presence of positive serologic evidence of syphilis warrant examination of the cerebrospinal fluid.[7] The disease may also present as acute syphilitic meningitis, syphilis of the spinal cord, vascular neurosyphilis, or syphilitic eye disease.[41] The rate of congenital syphilis is increasing, with this rate increasing 38% between 2012 and 2014, from 8.4 per 100,000 live births to 11.6 per 100,000 live births.[42] This recent increase demonstrates the need for increased attention to screening and prevention of syphilis.

Screening and Diagnosis of Syphilis

Syphilis screening relies on one of two tests that look for the nonspecific antibodies that develop during this disease. The most common non-treponemal tests, known as VDRL (Venereal Disease Research Laboratory) and RPR (rapid plasma reagent), have a high false-positive rate. Pregnancy, autoimmune disorders, and acute bacterial or viral infections, for example, can cause a false-positive result on these tests. For this reason, all persons who have a positive non-treponemal test are retested with a confirmatory treponemal test such as fluorescent treponemal antibody absorption test (FTA-ABS), passive particle agglutination (TP-PA) assay, enzyme immunoassays (EIAs), or chemiluminescent immunoassays (CLIAs). The treponemal tests detect antibodies that are specific for *T. pallidum*.[7]

Laboratory testing for syphilis requires several steps, and different protocols can be followed. If a woman presents with a chancre, positive results on dark-field microscopy of exudate from the chancre lesion provide definitive diagnosis; however, dark-field microscopy is not available in all settings.[7]

The usual first step if no chancre is present is to screen with a non-treponemal test (VDRL or RPR); such a test is sensitive but not specific. If those results are positive, they are confirmed with a specific treponemal test. If no chancre is present, the VDRL and RPR tests may not be positive for the first few weeks of infection (false negative), or positive results on the tests may be secondary to other conditions (false positive).[7]

Although the initial diagnosis is based on a positive treponemal test, this test remains positive for life after infection; as a consequence, it cannot be used to determine the presence of active infection or response to treatment. Instead, serial quantitative non-treponemal titers of immunoglobulin antibodies are used to determine the response to treatment and to detect reinfection.[7]

The protocol for diagnosis and treatment of syphilis is more complex if the woman has a

previous history of syphilis or if she is HIV positive; the CDC guidelines specify testing strategies for current syphilis in such individuals. Symptoms of neurosyphilis are also an indication for more intensive screening in consultation with a specialist.[7]

Treatment of Syphilis

Parenteral penicillin G is the best treatment for syphilis. Other forms of penicillin are not as effective because they do not adequately penetrate into sequestered sites such as the central nervous system. **Table 14-13** identifies the recommended doses and routes of treatment based on stage of infection. While alternative treatments are provided for nonpregnant individuals, their efficacy has not been studied in depth. If a person has a penicillin allergy, consider skin testing for verification, especially for late latent syphilis or syphilis of unknown duration.[7] Individuals with tertiary syphilis, neurosyphilis, or ocular syphilis should be referred to a specialist for more intensive diagnostic testing and treatment.

Table 14-13 **Outpatient Treatments for Syphilis**

Stage of Syphilis	Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatment for Breastfeeding Women
Primary, secondary, and early latent syphilis	Benzathine penicillin G (Bicillin LA) 2.4 million units IM in a single dose	Doxycycline (Vibramycin) 100 mg orally twice a day for 14 days OR Tetracycline (Sumycin) 500 mg four times a day for 14 days Other regimens may be available in consultation with an infectious disease specialist.	Benzathine penicillin G (Bicillin LA) 2.4 million units IM in a single dose If the woman is allergic to penicillin, desensitize per CDC guidelines. Jarisch–Herxheimer reaction may cause preterm labor or fetal distress in the first 24 hours of treatment; consider additional monitoring.	Benzathine penicillin is safe during breastfeeding. Doxycycline and tetracycline have the theoretical risk of staining forming teeth. Longer courses of therapy with these drugs are not recommended for breastfeeding mothers. If alternative therapies are not available, consider strategies to minimize drug transfer to the breast milk.
Late latent syphilis or syphilis of an unknown duration	Benzathine penicillin G (Bicillin LA) 7.2 million units IM once a week for three doses	Doxycycline (Vibramycin) 100 mg orally twice a day for 28 days OR Tetracycline (Sumycin) 500 mg four times a day for 28 days For other therapies, consult an infectious disease specialist.	Benzathine penicillin G (Bicillin LA) 7.2 million units IM once a week for three doses	

Abbreviation: CDC, Centers for Disease Control and Prevention; IM, intramuscular.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

Clinical assessment and a non-treponemal test (VDRL or RPR) should be performed 6 months and 1 year following treatment to test for treatment effectiveness. With adequate treatment, titers usually decline fourfold (e.g., 1:128 down to 1:32), but in 15% of treated individuals, the titer does not decline. An increase in titers by fourfold following treatment is indicative of treatment failure or reinfection.

All women who have a positive test for syphilis should be offered HIV screening. Also, all of the woman's sexual partners from within the past 90 days should be treated presumptively for syphilis. If the person has secondary or early latent syphilis, previous sexual partners in the previous year may need to be notified and tested per CDC guidelines[7]; local or state-level health departments may be able to assist in this process.

Management of Syphilis During the Perinatal Period

Syphilis readily crosses the placenta after 9 weeks' gestation. Transmission of the pathogen to the fetus usually occurs between the 16th and 28th weeks of pregnancy.[43] As many as 40% of women with untreated syphilis during pregnancy will miscarry. Up to 80% of infants born to women with primary syphilis will have congenital syphilis, including congenital malformation, but the risk of vertical transmission decreases with the length of time the woman has been infected.[44,45] Syphilis is also associated with preterm birth, low birth weight, and congenital malformations. Thus screening for syphilis is recommended for all women at their first prenatal visit and rescreening recommended at 28 weeks for women who have a high risk for developing syphilis.

The diagnostic algorithm for detecting syphilis infection during pregnancy recommended by the CDC is the "traditional" algorithm, which screens first with a non-treponemal test (VDRL or RPR) test, which is then followed by a confirmatory treponemal-specific test. Some settings prefer the "reverse algorithm," in which a treponemal-specific test is used as the first screening, with any reactive samples then being tested quantitatively with a non-treponemal test.[45] Both algorithms have strengths and weaknesses that are influenced by the local prevalence of the disease and the cost-effectiveness of the algorithm in high- versus low-volume laboratories.

Penicillin G is the only acceptable treatment for syphilis during pregnancy. If a woman is allergic to penicillin, she should be desensitized prior to treatment.[7] Desensitization is a procedure that works to alter a person's immune reaction so that the body does not react to the antigenic agent. It is accomplished by administering sub-threshold doses of the medication that cause immunoglobulin E (IgE) to bind to basophils and mast cells, thereby making them unresponsive to higher doses of the drug.[46] An oral protocol for penicillin desensitization is recommended by the CDC.[7] The procedure should be performed only in a setting where resuscitative equipment is readily available.

Jarisch–Herxheimer Reaction

The Jarisch–Herxheimer reaction refers to the appearance of fever, chills, tachycardia, hypotension, and intensification of skin rashes within the first 24 hours of treatment for syphilis. This reaction is a response to lipoproteins from the spirochete that induce a systemic inflammatory response.[47] It can cause preterm labor and abnormal fetal heart rate changes, so additional monitoring during the first 24 hours of treatment may be indicated.[7] If the woman is treated for syphilis less than 4 weeks before giving birth, the newborn will need additional testing and treatment as outlined by the CDC.[7] Women without documented syphilis testing should be screened for syphilis during the intrapartum period.[7]

Congenital malformations are common in infants born to women with untreated syphilis during pregnancy, and include deafness, hepatomegaly, and bone abnormalities. Even when pregnant women receive treatment for syphilis, it may come too late in gestation to prevent congenital abnormalities.[43] Notify the infant's care provider of syphilis exposure to facilitate appropriate neonatal care. Syphilis can cause intrauterine fetal demise, and maternal screening

for syphilis is recommended following stillbirth at any gestation.[43] Syphilis can also cause the placenta to be large and pale. Infants born to women with positive serologic tests need in-depth evaluation, including testing of the placenta, serologic tests of the newborn's blood (cord blood is not acceptable), and dark-field microscopy testing of suspicious body lesions.[7] Abnormal results warrant treatment and more invasive testing per CDC guidelines.[7]

Chancroid

Chancroid ulcers are nonindurated painful lesions caused by a gram-negative anaerobic bacillus, *Haemophilus ducreyi*. The infection is transmitted through sexual contact with mucous membranes. These lesions are usually found on the vulva, cervix, or perineum, and approximately 50% of affected women have associated unilateral adenitis that can develop into buboes—that is, abscesses of the local lymph nodes. The buboes can rupture, spreading infection and leading to permanent scarring. While the incidence of chancroid has declined around the world, outbreaks continue to occur, often associated with the sale or exchange of sex.[41]

Diagnosis of Chancroid

Diagnosis of chancroid is based on the findings of a physical examination. The presence of a painful ulcer on the genitals combined with tender inguinal lymphadenopathy suggests chancroid. Lymph nodes may be fluctuant and drain when pressed. Because many infections can cause genital lesions (**Table 14-14**), it is important to perform several tests directly on the genital lesion, including cultures for herpes simplex virus (HSV) and secondary infections, and dark-field microscopy for syphilis. If possible, obtain a culture for *H. ducreyi*; however, the culture medium is not widely available and has a low specificity. The woman should also be evaluated for chlamydia and gonorrhea. If it has been more than 7 days since the lesion appeared, also perform a non-treponemal test (VDRL, RPR) for syphilis. Negative serologic tests for syphilis and wound tests that are negative for HSV and syphilis are needed for confirmation of chancroid diagnosis.

Table 14-14 Differentiation of Genital Lesions by Symptoms

Typically Painless	Typically Painful
Genital warts/condyloma acuminata	Herpetic vesicles and open lesions
Syphilis chancre	Chancroid
Condylomata lata	Lymphogranuloma venereum
	Lymphadenopathy and lesions

Treatment of Chancroid

Table 14-15 outlines pharmacologic treatment for chancroid. Single-dose therapy is preferred for ease of use. There have been reports of *H. ducreyi* resistance to ciprofloxacin (Cipro) and erythromycin (E-Mycin) outside the United States. All of the woman's sexual partners within the last 10 days preceding onset of symptoms should be treated presumptively for this STI, and the woman should be tested for HIV and syphilis at the time of diagnosis.

Table 14-15 Treatments for Nonsyphilitic Genital Ulcer Diseases

Genital Ulcer Disease	Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women Generic (Brand)
Chancroid	Azithromycin (Zithromax, Z-Pak) 1 g orally once OR Ceftriaxone (Rocephin) 250 mg IM once OR Ciprofloxacin (Cipro) 500 mg orally twice a day for 3 days OR Erythromycin base 500 mg orally three times a day for 7 days	None	Azithromycin (Zithromax, Z-Pak) 1 g orally once OR Ceftriaxone (Rocephin) 250 mg IM once OR Erythromycin base 500 mg orally three times a day for 7 days	Same as for pregnant women.
Lymphogranuloma venereum	Doxycycline (Vibramycin) 100 mg orally twice a day for 21 days	Erythromycin base 500 mg orally four times a day for 21 days	Erythromycin base 500 mg orally four times a day for 21 days OR Azithromycin (Zithromax, Z-Pak) 1 g orally once a week for 3 weeks may be effective but few data on its efficacy are available.	Same as for pregnant women. Doxycycline (Vibramycin) is not an ideal treatment but may be used if risks to the infant can be minimized.
Granuloma inguinale (donovanosis) All treatments require that the medication be continued for at least 3 weeks and until the lesions completely heal	Azithromycin (Zithromax, Z-Pak) 1 g orally once per week OR Azithromycin (Zithromax, Z-Pak) 500 mg daily	Doxycycline (Vibramycin) 100 mg orally twice per day OR Ciprofloxacin (Cipro) 750 mg orally twice per day OR Erythromycin base 500 mg orally four times a day OR Trimethoprim–sulfamethoxazole 106 mg/800 mg orally two times a day	Azithromycin (Zithromax, Z-Pak) 1 g orally once per week OR Azithromycin (Zithromax, Z-Pak) 500 mg daily OR Erythromycin base 500 mg orally four times a day AND if needed Gentamycin 1 mg/kg IV every 8 hours if the woman does not improve with a few days of therapy	Same as for pregnant women. Doxycycline, ciprofloxacin, and trimethoprim–sulfamethoxazole have the potential for neonatal effects, but can be used if the infant is healthy, no other drugs are available, and risks to the infant can be minimized.

Abbreviations: IM, intramuscular; IV, intravenous.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

All women with chancroid should be screened for HIV. Women should be seen for a clinical examination 3 to 7 days after treatment initiation. The pain of the lesion and the lymphadenopathy should improve by 3 days after initiation of therapy, and the lesion should be healing by day 7 of treatment. If there is no improvement in this time frame, the woman will need a more comprehensive assessment, as outlined by the CDC.[7]

Lymphogranuloma Venereum

Lymphogranuloma venereum (LGV) is caused by *C. trachomatis* that is transmitted via genital or rectal mucosal contact. LGV is primarily an infection of the lymphatic system and lymph nodes. Although it is caused by *C. trachomatis*, serovars (subtypes) of the bacterium involved in LGV are different than those that cause chlamydial infection of the vagina or upper reproductive tract. Thus, LGV is a distinct disorder that differs from the more common manifestations of chlamydial infection.

Women with LGV most commonly present with unilateral, painful inguinal lymphadenopathy or pelvic pain. Approximately one-third of infected individuals remember having a nontender genital lesion. Rectal infection is becoming more common, and this location of infection can complicate diagnosis because the symptoms mimic primary care conditions—constipation, anal pain, tenesmus, and bloody and mucoid anal discharge are common.[48] Left untreated, LGV can lead to fistulas and permanent tissue damage as lymph nodes rupture and spread infection. LGV was previously rare in developed countries, but outbreaks have been increasing in recent years.[48]

Diagnosis of Lymphogranuloma Venereum

The physical examination will be remarkable for unilateral, painful, inguinal lymphadenopathy. Lymph nodes may be fluctuant and rupture on compression. The skin over the affected area may be darkened. Rectal examination reveals symptoms of proctocolitis, including inflammation of the rectal mucosa if the rectum is infected.

Laboratory tests include NAAT for *C. trachomatis* performed vaginally, rectally, or from lymph node or lesion exudate.[7] Even though laboratory tests should be performed, clinical symptoms alone can be used for diagnosis to begin treatment.[7]

Treatment of Lymphogranuloma Venereum

Pharmacologic treatment for LGV is listed in **Table 14-15**. Very large and fluctuant lymph nodes may require needle aspiration and drainage to prevent ulcerations. Nonsteroidal anti-inflammatory drugs (NSAIDs) may help mitigate pain and inflammation. All sexual partners within the 60 days prior to onset should be evaluated, tested, and treated if needed.

Granuloma Inguinale

Granuloma inguinale is rare in the United States, but is more common in low-resource tropical areas around the world. This infection is predominately transmitted through sexual contact, although nonsexual transmission may be possible. Caused by a gram-negative bacterium (*Klebsiella granulomatis*), granuloma inguinale infection begins with painless beefy-red lesions that bleed on contact. These genital lesions are progressive, spreading across the anogenital and inguinal areas. While local lymphadenopathy does not occur, subcutaneous granulomas (pseudoboboes) may form under the skin of the inguinal area. Direct testing for *K. granulomatis* is difficult even in well-supplied laboratories. Thus, diagnosis relies heavily on clinical assessment.

Diagnosis of Granuloma Inguinale

Physical examination reveals ulcerative lesions that are red and bleed easily. Lesions may also be hypertrophic or have necrotic wound portions. Local or regional lymphadenopathy and subcutaneous granulomas may be present as well. Secondary infections may cause a purulent exudate. Laboratory tests for the differential diagnosis of granuloma inguinale include HSV culture, culture for secondary bacterial infection, and dark-field microscopy for *T. pallidum*. Consider non-treponemal testing for syphilis. Because direct culture is difficult, diagnosis relies on clinical presentation and presence of risk factors for the disease (e.g., tropical location, local prevalence).

Treatment of Granuloma Inguinale

Table 14-15 summarizes pharmacologic treatment for granuloma inguinale. Treatment also includes basic wound management to treat or prevent secondary infections. Sexual partners within the previous 60 days should be evaluated and offered treatment, but the value of treatment for asymptomatic individuals has not been established.[7]

Human Papillomavirus

HPV is estimated to be the most common sexually transmitted disease; as many as half of all sexually active adults will be infected with one or more subtypes of HPV in their lifetime.[6] More than 100 subtypes of HPV have been identified to date, of which more than 40 subtypes infect the genital and potentially oropharyngeal areas. Papilloma viruses infect the surface epithelia and mucous membranes, where they cause variable cellular changes depending on the virus subtype.

Most HPV infections are asymptomatic, and 90% are eliminated by the immune system within 2 years following infection. However, certain subtypes are associated with persistent infection, leading to symptoms such as genital warts or cancers of the genitals, cervix, rectum, and oropharynx. Symptoms can appear years after the initial infection. HPV subtypes 6 and 11 cause 90% of genital warts, although subtypes 18, 31, and 33 are also found in warts. HPV subtypes 16 and 18 cause 60% to 80% of anogenital cancers, but subtype 31 is associated with anogenital cancers as well. Subtype 16 has been strongly associated with oropharyngeal cancer.[49] Warts caused by HPV subtypes 6 and 11 can also be found on mucous membranes of the mouth, nose, and eyes. This section focuses on HPV infection manifesting as growths or warts on the vulva, perineum, or anus, or within the vagina.

The path from HPV acquisition to oncogenic cell changes is complex and may be related to the individual's overall health status. Smoking increases the risk of cellular persistence of HPV and later cancer for both genital and oropharyngeal cancers.[50,51] Prolonged alcohol use is another risk factor for oropharyngeal cancer following HPV infection.[49]

Genital warts are common in the United States, accounting for hundreds of millions of dollars in healthcare expenditures annually. However, the prevalence of HPV may decrease dramatically in the next 10 to 15 years due to administration of the HPV vaccine to females and males younger than 26 years. The approved vaccines offer protection for either two, four, or nine HPV serotypes.[51]

The virus that causes genital warts is transmitted through skin and mucous membrane contact with the genitals, vulva, perineum, rectum, and—for some subtypes—oropharynx. Condoms do not thoroughly protect from genital HPV; female condoms offer some additional protection for the vulva when compared with male condoms, but do not completely eliminate the risk of infection.

Screening and Diagnosis of Human Papillomavirus Infection and Genital Warts

Screening for cervical HPV infection and cancer is part of routine health promotion, as reviewed in the *Health Promotion and Health Maintenance* chapter. Although HPV is an STI, it is impossible to determine the time of acquisition. The onset of genital warts is not a reliable indicator of either recent infection or reinfection.

Diagnosis of HPV-related genital warts occurs via physical examination, which will reveal fleshy papules or pedunculated warty lesions on the vulva, introitus, perineum, anus, cervix, and vaginal walls. Large warts, known as condylomata acuminata, may take on a cauliflower-like appearance and may bleed when abraded. These lesions may appear similar to the

condylomata lata associated with syphilis, but condylomata lata are usually flat. Warts caused by HPV turn white when exposed to acetic acid, although this test is not recommended by the CDC as a diagnostic test due to lack of specificity. Non-treponemal blood tests (RPR, VDRL) can be useful to differentiate condylomata acuminata from condylomata lata if needed.

Treatment of Genital Warts

Many genital warts resolve spontaneously and may not need treatment. The number, size, and location of warts and the cost and availability of treatments are important considerations in shared decision making.[7] All treatments for warts have localized side effects, including irritation, pain, and burning. While treatment may diminish the size and number of genital warts, it may not affect transmission rates. The woman or provider can apply treatment; self-applied medications may facilitate prolonged treatment if it is needed. Decisions about treatment should be made in partnership with the woman.

Treatments recommended by the CDC for women are presented in **Table 14-16**.[7] Podophyllin resin is no longer recommended for any woman. The CDC advises individuals to abstain from intercourse with new partners until the warts have resolved, but the person may remain contagious even after treatment. Sexual partners should be treated only if they are symptomatic and desire treatment. While pharmacologic treatments are listed in Table 14-16, surgical excision is an option if warts are extensive in any location.

Table 14-16	Pharmacologic Treatments for Genital Warts Caused by Human Papillomavirus for Women
Treatments Generic (Brand)	**Clinical Considerations**
External	
Podofilox 0.5% solution or gel (Condylox, Podofilox)	Applied by the woman twice a day for 3 days then no treatment for 4 days Useful when warts are smaller than 10 cm × 10 cm Useful when warts are smaller than 10 cm × 10 cm Maximum dose of 0.5 mL of the solution per day Demonstrate application in the office for instruction Contraindicated during pregnancy
Imiquimod 3.75% or 5% cream (Aldara)	May worsen autoimmune or inflammatory disorders Low risk for use during pregnancy but data are limited; not recommended for use during pregnancy Applied by the woman at bedtime—for 3.75% strength, apply every night for up to 16 weeks; for 5% strength, use three times a week for up to 16 weeks Wash off cream with soap and water after 6–10 hours May cause a local inflammatory response The cream may weaken latex condoms and diaphragms
Sinecatechins 15% ointment (Veregen)	Contraindicated for individuals who have herpes or HIV, or who are immunocompromised No safety data for use during pregnancy; not recommended during pregnancy Applied by the woman in a thin film over the affected area using a finger, three times a

	day for up to 16 weeks Do not wash the film off the affected area Avoid touching the skin with applied ointment to other genital, anal, or oral tissues
External	
TCA or BCA 80–90%[a]	Applied by the provider Protect areas surrounding the wart with petroleum jelly Apply acid sparingly and allow to dry, forming a white frosting Excessive acid can be neutralized with sodium bicarbonate or liquid soap Treatment can be repeated once a week Can be used during pregnancy
Cryotherapy with liquid nitrogen	Requires specialized training and equipment Performed by the provider Can be repeated every 1–2 weeks
Vaginal	
TCA or BCA 80–90%[a]	Applied by the provider Protect areas surrounding the wart with petroleum jelly Apply acid sparingly and allow to dry, forming a white frosting Excessive acid can be neutralized with sodium bicarbonate or liquid soap Treatment can be repeated once a week
Cryotherapy with liquid nitrogen	Requires specialized training and equipment Performed by the provider Can be repeated every 1–2 weeks
Urethral Meatus	
Cryotherapy with liquid nitrogen	Requires specialized training Performed by the provider Can be repeated every 1–2 weeks.
Cervical or Anal	
TCA or BCA 80–90%[a]	Intra-anal warts require specialist consultation Applied by the provider Protect areas surrounding the wart with petroleum jelly Apply acid sparingly and allow to dry, forming a white frosting Excessive acid can be neutralized with sodium bicarbonate or liquid soap Treatment can be repeated once a week Can be used during pregnancy
Cryotherapy with liquid nitrogen	Intra-anal warts require specialist consultation Requires specialized training and equipment Performed by the provider Can be repeated every 1–2 weeks

Abbreviations: BCA, bichloroacetic acid; HIV, human immunodeficiency virus; TCA, trichloroacetic acid.

[a] Acceptable pharmacologic treatment during pregnancy.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at:

https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

The need for follow-up visits is influenced by the size of lesions and the response to treatment. Warts may return following treatment and can be (re)treated with the same agent used for treating the initial infection. A biopsy is indicated if lesions have not responded after 3 months following treatment or if they are unevenly pigmented, ulcerated, or fixed.[7] Regular Pap screening according to current guidelines is important to evaluate affected women for cervical cancer, but a diagnosis of genital warts does not change regular screening frequency. Women with genital warts should be offered screening tests for common STIs.

Management of Genital Warts During the Perinatal Period

Warts may initially appear or increase in size during pregnancy. Treatment with trichloracetic acid (TCA) or bichloroacetic acid (BCA) is safe during pregnancy, but other medications used to treat HPV, such as podophyllin, are not recommended for use in pregnant women. It may be difficult to fully eliminate warts during pregnancy, and they may regress spontaneously postpartum. Surgical treatment is indicated if the warts might complicate vaginal birth.

Genital warts are highly vascularized and may bleed excessively if torn or cut during birth. Care should be taken to avoid warts during suturing of perineal lacerations following vaginal birth. Cesarean section is indicated only if extensive warts obstruct the vaginal opening or are expected to bleed uncontrollably during birth.[7]

Infants born to women with genital warts attributable to HPV subtypes 6 and 11 may develop respiratory papillomatosis—that is, warts within their airway—during the neonatal period or beyond. The mode of transmission to the fetus or neonate is not clear, and cesarean birth may not decrease the incidence of papillomatosis.[7] Some new research suggests that prenatal vaccination of the woman may reduce intrapartum fetal transmission due to antibody transfer to the fetus.[52] The infant's healthcare provider should be alerted about the potential for respiratory papillomatosis.[7]

Herpes Simplex Virus

A variety of herpes viruses affect humans. Sexually transmitted herpes infection is caused by one or two subtypes of herpes simplex virus: HSV-1 or HSV-2. These two viruses are not completely cleared from the body after the initial infection and can cause recurrent symptoms throughout life. In a large epidemiologic study of data from the U.S. National Health and Nutrition Examination Survey in 2010, the seroprevalence of HSV-1 in individuals between the ages of 14 and 49 was 53.9%. The seroprevalence of HSV-2 in this cohort was 15.7%.[53] However, in some demographic groups, the seroprevalence of HSV-2 infection in this age group was found to be close to 50%.[54] Women are more vulnerable to the HSV infection and have higher baseline rates of infection compared to men, in part due to prolonged contact with semen during vaginal intercourse.

HSV-1 and HSV-2 can cause painful ulcerations of the anogenital region and the mucous membranes of the mouth and nose. HSV-1 is commonly acquired in childhood through oral contact but can be sexually transmitted, especially through orogenital sex. In some populations, this virus is the major cause of genital herpes.[7] HSV-1 usually has a more mild clinical presentation than HSV-2, both during the first infection and subsequent outbreaks. In addition, HSV-1 is associated with lower rates of asymptomatic viral shedding.[7] HSV-2 is more often sexually transmitted and is associated with more severe symptoms and more frequent reoccurrences compared to HSV-1 infection.[7] In the United States, HSV-2 is more commonly associated with genital herpes than is HSV-1. Genital herpes infection increases the risk of acquiring HIV by fourfold, in part due to skin breaks and open lesions

Herpes is a lifelong infection that has the potential for transmission throughout the life span. Individuals can have HSV for many years before becoming symptomatic. Although an outbreak of HSV is caused by a virus that is sexually transmitted, the timing between infection and symptoms is highly variable. Furthermore, most infections of HVS-1 and HSV-2 are asymptomatic. Because HSV can be shed from asymptomatic lesions, the disorder may be transmitted to a sexual partner unknowingly. These characteristics of the viral infection can greatly complicate health education and partner counseling.

Outbreaks of HSV are commonly categorized as primary versus recurrent. The first episode of HSV (primary outbreak) is usually more severe than subsequent outbreaks. Prodromal symptoms may occur before the appearance of lesions and include tingling and burning. Constitutional symptoms can include fever, malaise, headache, and myalgia. The primary sign/symptom of the outbreak is painful lesions at the affected site. Primary outbreaks may include lesions in a wide swath or in multiple body locations; in contrast, recurrent outbreaks are usually more localized. Women may also have dysuria when urine comes in contact with an open lesion(s). All primary outbreaks should be treated to prevent prolonged or severe illness.[7] Subsequent outbreaks may be preceded by prodromal symptoms and are milder, but lesions are still painful.

Diagnosis of Herpes Simplex Virus

A woman having an HSV outbreak usually presents with small, very painful vesicles or open

lesions on the mouth, vulva, perineum, or anus. The lesions typically appear in small groups and are disproportionately painful in comparison to their size and depth. They may be fluid-filled vesicles—that is, small skin splits that are covered, oozing, crusted, or nearly healed. Edema, diffuse inflammation, and vaginal or urethral discharge may be present as well. A speculum examination may be excessively painful and is not required for diagnosis. Tender inguinal lymphadenopathy may also be present.

Diagnosis of herpes is based on physical examination confirmed with viral and serologic testing. Cell culture or polymerase chain reaction (PCR) testing of exudate from the lesion is the standard for confirmation of viral lesions. However, viral culture has low sensitivity, especially if the area is crusted over or healing.[7] Type-specific immunoglobulin G (IgG)–based assay blood testing can diagnose herpes and reveal the subtype, which is recommended by the CDC to assist in treatment decisions. The woman who has an initial herpes diagnosis should be offered testing for all common STIs, especially if she has a new partner.

Treatment of Herpes Simplex Virus

While the clinical presentation of the two subtypes is indistinguishable, the CDC recommends typing via blood testing to guide health education and treatment decisions, as HSV-2 has more virulent reoccurrences and a higher rate of transmission.[7] Serologic and viral test results are not needed to begin treatment for HSV with antiviral therapy. Treatments for initial outbreaks, episodic outbreak, and suppressive therapy are presented in **Table 14-17**.[7,55] All initial outbreaks should be treated.[7] For episodic outbreaks, medication should be started when the first prodromal symptoms are noted for best effectiveness, but treatment is beneficial even if delayed until the outbreak of lesions.[7] Suppressive treatment should be considered if recurrences are frequent or disruptive for the woman. Suppression also decreases the risk of asymptomatic shedding and transmission.[7] The woman's need for suppressive therapy may change over time and should be reevaluated periodically.[7]

Table 14-17 **Pharmacologic Treatments for Herpes Simplex Virus**

Diagnosis	Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women Generic (Brand)
Initial outbreak of HSV in an adult	Acyclovir (Zovirax) 400 mg by mouth three times a day for 7–10 days OR Acyclovir (Zovirax) 200 mg by mouth five times a day for 7–10 days OR Famciclovir (Famvir) 250 mg by mouth three times a day for 7–10 days OR Valacyclovir (Valtrex) 1 g by mouth twice a day for 7–10 days	Acyclovir has more data supporting its benefits without risks to the fetus and is preferred by the CDC. ACOG supports the use of acyclovir or valacyclovir. Severe infections should be treated with intravenous acyclovir. Acyclovir (Zovirax) 400 mg by mouth three times a day for 7–10 days OR Acyclovir (Zovirax) 200 mg orally five times a day for 7–10 days OR Valacyclovir (Valtrex) 1 g by mouth twice a day for 7–10 days	Acyclovir (Zovirax) is considered safe in breastfeeding, with drug passage to the infant being much less than newborn therapeutic doses. Lower dosages may decrease maternal serum levels, resulting in lower breast milk concentrations. Valacyclovir (Valtrex) is acceptable for use during lactation, as it is converted to acyclovir in the mother and excreted in very low amounts into breast milk. Famciclovir (Famvir) is not recommended during lactation due to lack of data.

Recurrent outbreak treatment	Acyclovir (Zovirax) 400 mg by mouth three times a day for 5 days OR Acyclovir (Zovirax) 800 mg by mouth twice a day for 5 days OR Acyclovir (Zovirax) 800 mg by mouth three times a day for 2 days OR Famciclovir (Famvir) 125 mg by mouth two times a day for 5 days OR Famciclovir (Famvir) 1 g by mouth two times 1 day OR Famciclovir (Famvir) 500 mg by mouth once, then 250 mg twice a day for 2 days OR Valacyclovir (Valtrex) 500 mg by mouth twice a day for 3 days OR Valacyclovir (Valtrex) 1 g by mouth once a day for 5 days	The CDC states that acyclovir has a better safety profile during pregnancy. ACOG supports the use of valacyclovir if needed. Acyclovir (Zovirax) 400 mg by mouth three times a day for 5 days OR Acyclovir (Zovirax) 800 mg twice a day for 5 days OR three times a day for 2 days OR Valacyclovir (Valtrex) 500 mg by mouth twice a day for 3 days OR Valacyclovir (Valtrex) 1 g by mouth daily for 5 days	See above.
Suppression of HSV outbreaks	Acyclovir (Zovirax) 400 mg orally two times a day OR Famciclovir (Famvir) 250 mg orally two times a day OR Valacyclovir (Valtrex) 1 g orally once a day OR Valacyclovir (Valtrex) 500 mg orally once a day (may be less effective than other dosing regimens)	The CDC does not have recommendations specific to pregnant women but states that acyclovir has a better safety profile. ACOG recommends: Acyclovir (Zovirax) 400 mg by mouth three times a day beginning at 36 weeks OR Valacyclovir (Valtrex) 500 mg orally twice a day beginning at 36 weeks	See above.

Abbreviation: ACOG, American College of Obstetricians and Gynecologists; CDC, Centers for Disease Control and Prevention; HSV, herpes simplex virus.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017[20]; American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 82: management of herpes in pregnancy. *Obstet Gynecol.* 2016;109(6):1489-1498.[55]

Cost and frequency of dosing are considerations when choosing a treatment for the woman with HSV-1 or HSV-2 infection. Famciclovir (Famvir) and valacyclovir (Valtrex) have greater

oral bioavailability than acyclovir (Zovirax) but are more expensive. Topical treatments are not very effective for management of initial or subsequent outbreaks. Support includes non-opioid pain management. The woman can use a peri-bottle or urinate while sitting in water in a bowl or the bathtub to decrease symptoms of dysuria.

There is limited definitive research on complimentary suppressive therapies, although l-lysine has been found not to be effective.[56] The woman can decide if the costs and side effects of such treatments are worth the personal benefit. If the woman does not improve with treatment, she may have a strain of herpes resistant to standard antiviral treatments, and viral isolate testing is needed prior to specialist consultation for further treatment.[7]

Partner treatment is not needed when a woman has HSV infection. Ideally, the woman will have an open conversation with her sexual partners about the risk of asymptomatic transmission and the need for safer sexual practices. The woman should avoid sexual contact with uninfected partners while having prodromal or outbreak symptoms, as condoms do not provide full protection from transmission. A follow-up visit is best for health education about prevention when compared to the visit for the initial outbreak. When the woman is feeling better, she will be more prepared to understand the information.

Management of Herpes During the Perinatal Period

All women should be asked if they have a history of herpes or genital lesions as part of their initial visit for pregnancy.[7] Recurrent infections during pregnancy pose little to no risk during the prenatal period, and the woman produces immunoglobulins that cross the placenta to protect the fetus from HSV. In contrast, primary outbreaks during pregnancy have been associated with a small increase in fetal abnormalities and a larger risk for intrapartum transmission.[57]

Women can be treated for herpes infections during pregnancy, as described in **Table 14-17**. Severe outbreaks with dissemination during pregnancy warrant hospitalization and treatment with intravenous acyclovir.[7] Prophylactic administration of acyclovir beginning at 36 gestational weeks has been shown to lower the incidence of asymptomatic shedding and outbreaks at term, the rate of cesarean section for HSV lesions, and the neonatal incidence of HSV.[55] Routine administration of antiviral drugs beginning at 36 weeks' gestation for women with a history of genital HSV confirmed by serologic testing is currently recommended.[55]

Pregnant women without a history of herpes whose sexual partners have herpes should be encouraged to use safe sex practices and avoid sexual contact when the partner has prodromal symptoms and/or a visible lesion. This consideration is especially important when pregnant women are close to term. In addition, women should not receive orogenital contact near their estimated date of birth if their partner has a history of oral herpes sores.

If a woman has a history of genital herpes, a careful perianal assessment is important at the first labor examination or prior to induction to rule out active infection. The greatest risk of intrapartum transmission to the infant is during a primary outbreak. Vaginal birth during a primary outbreak can have vertical transmission rates as high as 50%.[7] Vaginal birth during recurrent infections has a lower transmission rate (less than 1%), due in part to passage of

maternal immunoglobulin to the fetus across the placenta.[7] However, because morbidity and mortality are high following neonatal infection, a cesarean section should be offered if the woman has *any* active genital lesions or prodromal symptoms at the onset of labor in an area that could come in contact with the fetus during vaginal birth.[55]

If a woman has nongenital lesions when she begins labor, vaginal birth may proceed normally. All nongenital lesions should be covered with an occlusive dressing to decrease risk of transmission, especially when handling the newborn.[55] Occasionally, a woman will have a precipitous labor and give birth vaginally during a herpetic outbreak. In these cases, viral cultures of the newborn and prophylactic treatment with acyclovir may be considered, especially if this is the mother's first outbreak.[7]

The majority of newborns in the United States who develop neonatal herpes are born to women with no known history of the disease; this is due in part to increased monitoring of women with a history of herpes.[55] Approximately 85% of newborns with HSV infection contracted the virus during birth through contact with maternal secretions, whereas 10% are infected after birth via contact with infected individuals.[57] In addition, ritual circumcision with suction of the baby's blood using the adult's mouth has been shown to transmit HSV.[58] Although rare (accounting for only 5% of all neonatal cases), HSV can be transmitted across the placenta. Manifestations of this condition are similar to intrauterine varicella infection and include skin lesions, malformations of the eyes, and central nervous system dysfunction.[57]

Regardless of the route of transmission or subtype involved, HSV infection is very risky for the newborn. Symptoms of neonatal herpes usually appear on days 5 to 12 of life and include respiratory distress, irritability, jaundice, and vesicular lesions around the site of infection. Late symptoms include seizures and shock. Although neonatal herpes can remain localized to one area, there is a large risk of disseminated infection and central nervous system involvement—a condition that can lead to mortality rates as high as 85% without treatment.[57] Early treatment with antiviral agents can reduce this risk of death to 29%.[57] The pediatric provider should be notified immediately following the birth if a newborn may have been exposed to HSV during the birth.

HSV is communicable only through contact with infected secretions from mucous membranes of the mouth or genitals. Women with HSV should be encouraged to breastfeed unless there is a herpetic lesion directly on the nipple.[55]

Hepatitis

Hepatitis is a small double-stranded DNA hepadnavirus. Eight genotypes of hepatitis viruses (A–H) have been identified, all of which have been found in the United States. Nevertheless, hepatitis A, B, and C are the most prevalent types in the United States. The hepatitis viruses primarily attack the liver and cause acute and/or chronic hepatitis. Chronic infection can lead to cirrhosis, hepatocellular carcinoma, and death.

Hepatitis viruses can be acquired in different ways. Hepatitis A virus (HAV) is primarily transmitted via fecal–oral contact and is reviewed in the *Common Conditions in Primary Care* chapter. Hepatitis B (HBV), C (HCV), and D (HDV) are transmitted via blood and bodily secretions, including semen and (rarely) saliva. Such transmission may occur during sexual contact (genital and oral), during birth, through needle-sticks or sharing of needles, and with use of nonsterilized instruments. Thus HBV, HCV, and HDV infections are considered STIs.

Hepatitis B

More than 240 million people worldwide and 2.2 million people in the United States are chronically infected with the hepatitis B virus (HBV).[59,60] HBV can remain infectious outside the body for several days, even if dry, and is able to establish infection with very few initial organisms, making it highly infectious.[59] In addition, HBV has numerous components that are antigenic, including the surface antigen (HBsAg), core antigen (HBcAg), and e antigen (HBeAg). The chance of transmission of HBV depends on the infected individual's viral load and antigen status (a marker of high infectivity).

HBV can become a chronic infection that causes cirrhosis, liver cancer, and death. The chances of chronic infection vary by age at the time of initial infection. For example, 90% of infants who acquire HBV via mother-to-child-transmission at the time of birth will develop chronic infection, compared to 5% or fewer of persons who acquire the disease during adulthood.[61] Global rates of chronic infection are higher in part because of the impaired immune response associated with coinfection with HIV and tuberculosis.[59]

HBV infection is often asymptomatic, or symptoms may be vague enough to go unnoticed. This disease typically has an incubation period of 60 to 90 days. The prodromal phase lasts 3 to 10 days, during which commonly noted symptoms include fatigue, nausea, and diffuse epigastric or right upper quadrant pain. This is followed by an icteric phase of 1 to 3 weeks that is characterized by symptoms such as jaundice, dark urine, and gray stools. Convalescence can take weeks to months.

Prevention of Hepatitis B

In the United States, the overall rate of HBV infection has declined more than 80% since 1991, when the CDC began a campaign to reduce HBV prevalence.[60] The HBV vaccine is produced from derivatives of yeast, without the use of blood products, so it cannot cause hepatitis.[7] Vaccination is recommended for several at-risk groups, as described in the *Health Promotion*

and Health Maintenance chapter.[7,59] The vaccine dose varies based on the reason for vaccination, with immunocompromised adults receiving larger doses. Individuals should receive all doses from the same vaccine manufacturer.[7]

Routine screening of pregnant women and immunoprophylaxis of all newborns of infected mothers have also been useful in decreasing the prevalence of chronic hepatitis in the United States.[60] Other preventive strategies include universal precautions, careful instrument sterilization, safer sex practices, and not sharing toothbrushes and razors with infected individuals.[7,59]

Postexposure Immunoprophylaxis.

Postexposure prophylaxis decreases rates of acute and chronic infection but is most useful if prophylaxis is administered within 24 hours of exposure.[7] If an individual who has not been previously vaccinated, or who is not fully vaccinated, is exposed to HBV, administration of the HBV vaccine at the same time (different injection sites) as hepatitis B immunoglobulin (HBIG) has been shown to reduce transmission.[7] The vaccine series should then be completed using the CDC age-appropriate dose and schedule. Information about postexposure prophylaxis resources for viral blood-borne infections can be found in the Resources section at the end of this chapter.

Screening and Diagnosis of Hepatitis B

Screening for HBV occurs via serologic testing for hepatitis B surface antigen (HBsAg). If the woman is HBsAg positive, a complete hepatitis panel can be obtained to determine acute versus chronic status. This panel includes the hepatitis B e antigen (HBeAg), which is a marker of a high degree of infectivity; IgM anticore antibody (IgM anti-HBc), which is a marker of recent infection within the last 6 months and indicates acute infection; IgG anticore antibody (IgG anti-HBc), which is a marker of past or current infection; antihepatitis B surface antibody (anti-HBs), which is a marker of an immune response to HBV; antihepatitis B e antigen (anti-HBe), which may be present in a person who is either infected or immune; and viral load, which reveals the concentration of the hepatitis B virus DNA within the blood.

Diagnosis is based on interpretation of hepatitis serology, as shown in **Table 14-18**[7,62] and **Figure 14-3**.[65] Physical examination findings during acute HBV infection include upper abdominal tenderness, hepatomegaly, and, occasionally, jaundice. Women with hepatitis may also exhibit other abnormal serum tests, including elevated aspartate aminotransferase (AST), elevated alanine aminotransferase (ALT), elevated bilirubin, and a prolonged prothrombin time.

Table 14-18 **Interpretation of Hepatitis B Serology**

Interpretation	HBsAg	HBeAg	IgM Anti-HBc	Total Anti-HBc	HepB Serum Titer	Anti-HBe	Anti-HBs
Never infected (susceptible)	–	–	–	–	–	–	–
Early acute or immediately after vaccination[a]	+	–	–	–	Variable	–	–
Acute	+	+	+	+	High	–	–
Resolving acute	+	–	+	+	Low	+ or –	–
Chronic	+	+ or –	–	+	High or low	+ or –	–
Resolved (immune)	–	–	–	+	–	+ or –	+
Vaccinated (immune)	–	–	–	–	–	–	+
False positive[b]	–	–	–	+	–	–	–

Abbreviations: anti-HBe, antihepatitis B e antibody; anti-HBs, antihepatitis B surface antibody; FDA, U.S. Food and Drug Administration; HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen; HepB, hepatitis B; IgM Anti-HBc, IgM antibody to hepatitis B core antigen; +, positive; –, negative.

[a] The HBsAg test can lead to false positive because it can react to other viral antigens. To ensure that a positive HBsAg is not a false positive, samples that are positive should be tested with an FDA-cleared test for HBsAg only.
[b] If the anti-HBc is positive and other hepatitis serologies are negative, it is unlikely the person is infected, except in case of previous blood transfusion or organ transplantation.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; Brook MG, Mutimer D. Viral hepatitis: screening and vaccination strategies. In: Zenilman JM, Shahmanesh M, eds. *Sexually Transmitted Infections.* Sudbury, MA: Jones & Bartlett Learning; 2012:241-250.[62]

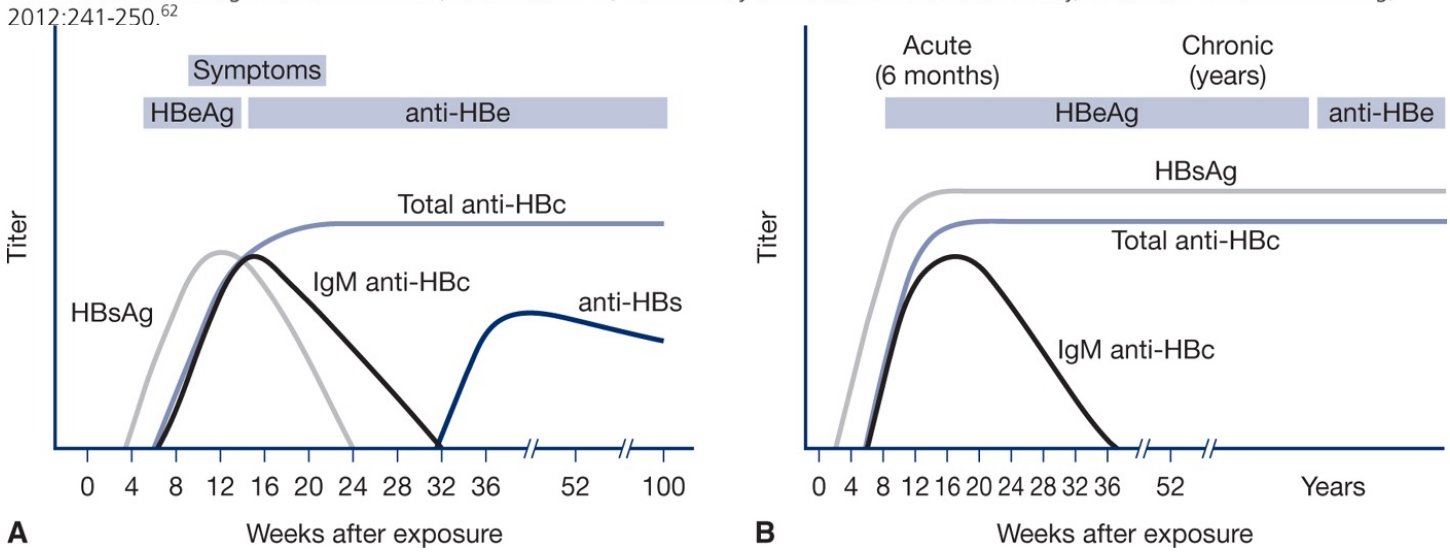


Figure 14-3 **A.** Acute hepatitis B virus infection with recovery types: serological course. **B.** Chronic hepatitis B virus infection: serological course.

Abbreviations: anti-HBc, antihepatitis B core antigen; anti-HBe, antihepatitis B e antigen; HBsAg, hepatitis B surface antigen; HBeAg, hepatitis B e antigen; IgM anti-HBc, IgM anticore antibody.

Reproduced with permission from Weinbaum CM, Williams I, Mast EE, et al. Recommendations for identification and public health management of persons with chronic hepatitis B virus infection. *MMWR Recomm Rep.* 2008;57(RR08):1-20.*65*

Treatment of Hepatitis B

All cases of newly diagnosed hepatitis B should be reported to the local health department. If a woman has hepatitis B, her household contacts should be vaccinated, and her sexual partner tested and vaccinated if not infected.[64] There is no specific treatment for adults with acute hepatitis, other than a supportive lifestyle to assist the body in healing.[7]

Individuals who are chronically infected can receive treatment to decrease the risk of liver disease,[7] and should be referred to providers knowledgeable in the management of hepatitis and chronic liver disease.[7] The woman can benefit from specialist referral for guidance about lifetime lifestyle modifications to decrease liver injury (e.g., avoidance of acetaminophen and alcohol).

Management of Hepatitis B During the Perinatal Period

Serologic screening for HBsAg is routinely included as one of the initial laboratory tests for all pregnant women during each pregnancy.[64] Hepatitis B vaccination is safe during pregnancy and appropriate for women at risk. Pregnancy is a good time for vaccination of at-risk women, as they are already in the clinic at the appropriate intervals.[64] **Figure 14-4** presents an algorithm for screening and referral for pregnant women with HBV.[64,65]

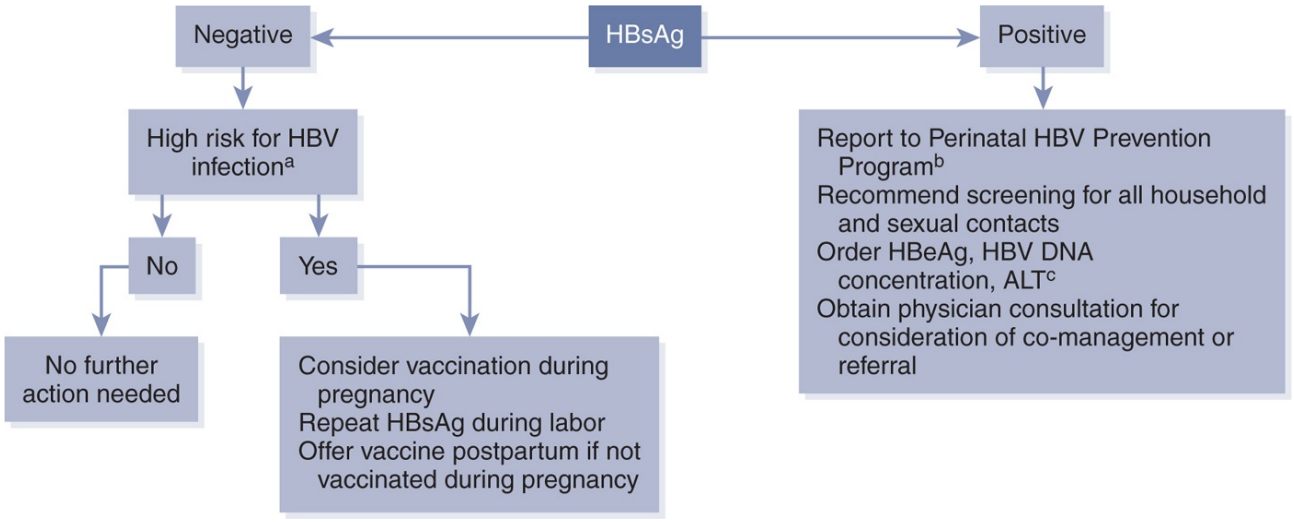


Figure 14-4 Screening and referral algorithm for hepatitis B virus infection among pregnant women.

Abbreviations: ALT, alanine transaminase; DNA, deoxyribonucleic acid; HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; HIV, human immunodeficiency virus.

[a] High risk for HBV infection includes household or sexual contacts of HBsAg-positive persons, injection drug use, more than one sex partner during the last 6 months, evaluation for or treatment of a sexually transmitted infection, HIV infection, chronic liver disease, or end-stage renal disease, and international travel to regions with HBsAg prevalence of more than 2%.

[b] The Centers for Disease Control and Prevention coordinates and funds state, local, and territorial programs that focus on preventing hepatitis B virus transmission from pregnant women to their newborns. Local reporting guidelines can be found at https://www.cdc.gov/hepatitis/partners/perihepbcoord.htm.

[c] If HBeAg positive or HBV DNA > 20,000 IU/mL or ALT ≥ 19 IU/mL, immediate referral to specialist for care during pregnancy is recommended.

Based on Centers for Disease Control and Prevention, American College of Obstetricians and Gynecologists. Screening and referral algorithm for hepatitis B virus (HBV) infection among pregnant women. 2015. Available at: https://www.cdc.gov/hepatitis. Accessed March 31, 2017.*64*

Women who are acutely infected with HBV may need additional care if they have severe symptoms, have high levels of HBV in their blood, have elevated alkaline aminotransferase levels, or show signs of coagulopathy or encephalopathy.[64,66] Viral load is an important component of management. Women with high viral loads (more than 6–8 log 10 copies/mL) can benefit from specialist involvement and antiretroviral medications during the third trimester,[67] but pregnancy does not change the course of HBV infection for most women.[66,68]

Treatment of HBV during pregnancy is primarily directed toward decreasing the risk of vertical transmission. Although current guidelines do not recommend treatment during pregnancy for all women with chronic HBV,[66] recent studies have shown that treatment of chronic HBV during pregnancy decreases the risk of vertical transmission; as this research matures, universal treatment may become recommended.[69]

The risk of vertical transmission increases with viral load and type of antigen present. Women who are HBsAg positive as well as HBeAg positive are at the greatest risk for vertically transmitting the virus, and should be referred for a specialist consultation to make a decision about treatment during the third trimester of pregnancy.[64,66,70] Invasive genetic testing can be offered to HBsAg-positive women, but vertical transmission is increased with high viral load counts at the time of the procedure.[67] The woman's chart should note her HBsAg status, and this information should be conveyed to the woman's chosen birth location to allow preparation for HBIG administration to the newborn following birth.

Intrapartum management of women infected with HBV is the same as that offered to women who are not infected with HBV. Hepatitis status should not influence the preferred route of birth (vaginal or cesarean section). However, because fetal scalp electrodes may increase the risk of neonatal infection, an electrode should be applied only if the benefits outweigh the potential risks.

The CDC advises routine HBV vaccination of all infants weighing more than 2000 g, regardless of the mother's HBV status.[71] Newborns of women infected with HBV should be given HBIG and their first hepatitis vaccine within 12 hours of birth.[71] Removal of maternal blood from the infant's skin or bathing of the infant soon after birth is also recommended to decrease neonatal contact with maternal blood.[63] The CDC is an excellent resource for comprehensive information about prevention of perinatal transmission of hepatitis B and provides health education information in a variety of languages.

Hepatitis B is *not* transmitted through breast milk, and women should be encouraged to breastfeed regardless of their hepatitis B status.[7] While transmission of HBV from women with cracked or bleeding nipples has raised some concerns, there is no conclusive evidence of transmission by this route. Breastfeeding should not be interrupted for this reason.[67]

Hepatitis C

Risk factors for HCV include nonsterile tattooing, blood transfusion before 1987, organ transplant before 1992, HIV-positive status, STIs, multiple sexual partners, evidence of liver disease, and a history of long-term hemodialysis.[72] The primary risk factors for HCV in the United States are use of blood products and illicit intravenous drug use. Compared to HBV, HCV is much less likely to be transmitted via sexual contact or via mother-to-child-transmission during birth, although transmission can occur through these routes.

The CDC recommends that all individuals with risk factors and persons born between 1945 and 1965 be tested for HCV.[7] Several forms of HCV exist, and knowing the genotype informs treatment.[72] Approximately 75% to 85% of persons with HCV will develop chronic infection, and HCV is one of the leading causes of liver cancer.[72] The U.S. Food and Drug Administration (FDA) has approved several drugs for treatment of HCV. Women with acute HCV should be referred to a specialist for care.

Management of Hepatitis C During the Perinatal Period

Women at risk for HCV should be offered screening during pregnancy, but universal screening during pregnancy is not recommended at this time.[66,73] However, as the incidence of HCV in the United States has risen in recent years in concert with the increased use of opioids, universal screening for HCV may become recommended in some geographic areas.[73,74] The risk of transmitting HCV to the fetus during birth is approximately 6%; at this time, there are no treatments proven to prevent mother-to-child-transmission.[75] Treatment of acute HCV during pregnancy may require hospitalization and physician care, but there is no standard treatment for chronic HCV at this time. Women with chronic HCV may breastfeed. Midwives, in consultation with obstetric specialists, can manage care for women with chronic HCV.

Hepatitis D

Hepatitis D is an incomplete RNA hepatitis viral particle that causes disease only in the setting of chronic HBV infection. Hepatitis D can obtain a viral envelope composed of HBsAg when in the presence of HBV, thereby becoming communicable and presenting as coinfection. HDV is primarily transmitted via blood. This hepatitis variant is associated with more severe disease and more rapid progression to cirrhosis, as compared to other hepatitis variants.

Persons with chronic HBV should be tested for HDV. If present, the individual will be treated for HBV and require specialist care. The hepatitis B vaccine also confers protection against HDV.

Human Immunodeficiency Virus

The human immunodeficiency virus is an RNA retrovirus (i.e., an RNA virus that replicates via production of DNA that is inserted into the host cell genome) that is transmitted through infected blood and bodily secretions. The CDC estimates that 50,000 individuals contract HIV in the United States every year, and 250,000 individuals have undiagnosed HIV infection. In the United States, HIV is most commonly transmitted through sexual intercourse. Exposure to infected blood through sharing of infected needles or accidental needle-sticks and other mechanisms are additional routes of transmission. Pre-exposure prophylaxis is available for those persons at high risk for HIV acquisition, and postexposure prophylaxis can be used within 72 hours of an exposure to prevent HIV acquisition.

The risk of acquisition of HIV during sex is greatly increased if a woman has other STIs, such as herpes, gonorrhea, or STIs causing genital ulcers that facilitate access to the circulation. Sexual practices that are more damaging to the vaginal and rectal mucosa also increase the risk of contracting HIV.

HIV proceeds through several stages after infecting the immune system's CD4 cells (a type of T cell). The first stage, called acute retroviral syndrome, occurs within the first few weeks after infection. It is characterized by fever, malaise, a skin rash, nausea or diarrhea, headache, sore throat, and lymphadenopathy—symptoms that are much like those associated with mononucleosis.[7] The symptoms of acute retroviral syndrome can be mild or severe. The infection then becomes asymptomatic during a period of clinical latency that can last a short time or up to 8 years or longer, depending on the individual. During this time, HIV reproduces at very low levels, although it is still active. When the CD4 cell count falls below 200 cells/mm^3, symptoms of advanced infection or acquired immunodeficiency syndrome (AIDS) appear. Symptoms of AIDS include fever, weight loss, diarrhea, cough, shortness of breath, opportunistic infections, and more intense illnesses and infections more severe than would be expected for the woman's age or health status.[7]

In late stages of untreated HIV infection, the number of functioning CD4 cells decreases as viral replication kills the host cells, rendering the woman susceptible to a wide variety of opportunistic infections. Presenting symptoms may include vulvovaginal or oral candidiasis, shingles, abnormal Pap tests, and STIs. In addition, HIV infection makes women more susceptible to cancer. Once the CD4 count falls below 200 cells/mm^3, the woman is likely to develop a variety of opportunistic infections, which collectively define the onset of AIDS.[7] However, with current treatment, HIV infection can be successfully managed, vastly improving long-term health outcomes.

Two types of HIV viruses exist: HIV-1 and HIV-2. The vast majority of HIV infections in the United States and worldwide are caused by the HIV-1 virus. HIV-2 is predominately found in West Africa, but this subtype is now becoming more common globally.[7] Both types of HIV produce similar symptoms and effects within the body, but their infectivity and treatment vary. Initial screening tests for HIV are able to detect antibodies to both HIV-1 and HIV-2, and further tests can differentiate the viruses (see **Figure 14-5** later in this section).

Prevention of HIV Infection

Strategies to prevent HIV transmission range from behavioral to pharmacologic in nature. Methods to prevent transmission can be customized, based on HIV status and sexual behaviors, for the woman and her partner(s).[75,76] The CDC has an excellent, interactive risk reduction tool that can be found in the Resources section at the end of this chapter. Women at high risk for HIV acquisition may also benefit from pre-exposure or postexposure prophylaxis[75,76]

Pre-exposure Prophylaxis

Pre-exposure prophylaxis (PrEP) has been shown to reduce the risk of HIV acquisition by as much as 92%.[77] PrEP is appropriate for any adult woman at high risk for HIV infection, including women who use intravenous drugs and have been in drug treatment, share injection equipment, or have an HIV-positive drug-injecting partner. Women who have sex with men and have had a recent bacterial STI, infrequently use condoms with one or more nonmonogamous sexual partners at risk for HIV (including a male who also has sex with males), engage in commercial sex work, or live in a high-prevalence geographic area should also be counseled about PrEP availability and effectiveness.

Prior to beginning PrEP, women should be tested for HIV, renal function, and hepatitis B and C.[76] If indicated, women should be offered vaccination for hepatitis B.[71] Women also need a method of contraception while taking PrEP.[75] All contraceptives can be used while taking antiviral medications, although fosamprenavir may have some drug interactions with contraceptives.[78]

If a woman who is HIV negative has a male sexual partner who is HIV positive and wishes to conceive, guidance for reproductive options is available on the CDC website, and specialist referral is appropriate.[79] PrEP use during pregnancy is still under study.[79] Specialist input is valuable for planning care during pregnancy, as risks and benefits of PrEP depend on several factors as well as maternal preference.

Once the woman is appropriately screened, PrEP involves a once-daily oral combination of 300 mg tenofovir disoproxil fumarate and 200 mg emtricitabine (Truvada). This regimen decreases the chance of HIV acquisition, but the woman should continue safer sex practices. While receiving PrEP, the woman needs to visit every 3 months for HIV and pregnancy testing and continued health education on risk reduction; renal function should be tested at 3 months, and then every 6 months.[77] Specialist involvement is ideal when possible, but should not delay access to PrEP when needed. The CDC website provides more information.

Postexposure Prophylaxis

Postexposure prophylaxis (PEP) differs depending on the origin of the exposure: occupational or non-occupational. When a healthcare provider is exposed to potentially infectious fluids, HIV testing should ideally be performed on the source individual (with her or his consent), and then a plan for care quickly formulated using expert consultation or the current information on the CDC website.[80,81] PEP for HIV is time sensitive and should occur within 72 hours of

exposure.[79]

Non-occupational exposure to HIV-infected fluids through sex, assault, or injection drugs also needs rapid attention (e.g., an expedited clinic appointment). Exposed individuals should receive a comprehensive assessment, including information on the exposure as well as the HIV status of the source individual. If the source individual is not known to be HIV positive, administration of PEP should be determined on an individual basis, with the woman receiving health education about the risks and benefits of each approach so she can participate in shared decision making. The risk for HIV acquisition depends on the source fluid (blood, semen, other bodily fluids) and the site of fluid contact. In addition to HIV counseling, the shared decision-making process should also consider the woman's risk for other STIs as well as hepatitis B and C acquisition.

The CDC has comprehensive information on baseline testing and PEP regimes for HIV and other infections.[82] Emergency contraception should also be offered when appropriate, as detailed in the *Family Planning* chapter.

Screening and Diagnosis of HIV Infection

Midwives are often involved in screening and diagnosis of HIV infection. Such screening should be offered in the same manner as any test. Consent for HIV testing can be included within a larger consent for all health care; a special HIV testing consent form is not needed and may act as a barrier to screening. HIV testing should be presented as the norm, and women allowed to opt out without coercion if they do not wish to be tested.[7]

Routine HIV screening should be offered to all women at risk for STIs, and annual screening provided to women at high risk, as reviewed in the *Health Promotion and Health Maintenance* chapter. An algorithm for interpretation of HIV tests is found in **Figure 14-5**.[83] As many as 16% of persons who are HIV positive do not know their HIV status.[7] Pregnant women should be screened at least once during pregnancy using an opt-out approach, and another HIV test should be considered in the third trimester.[7] Many states require third-trimester testing for all pregnant women, unless they opt out of screening.[7] State requirements can be found on state health websites and in CDC's HIV online resources.

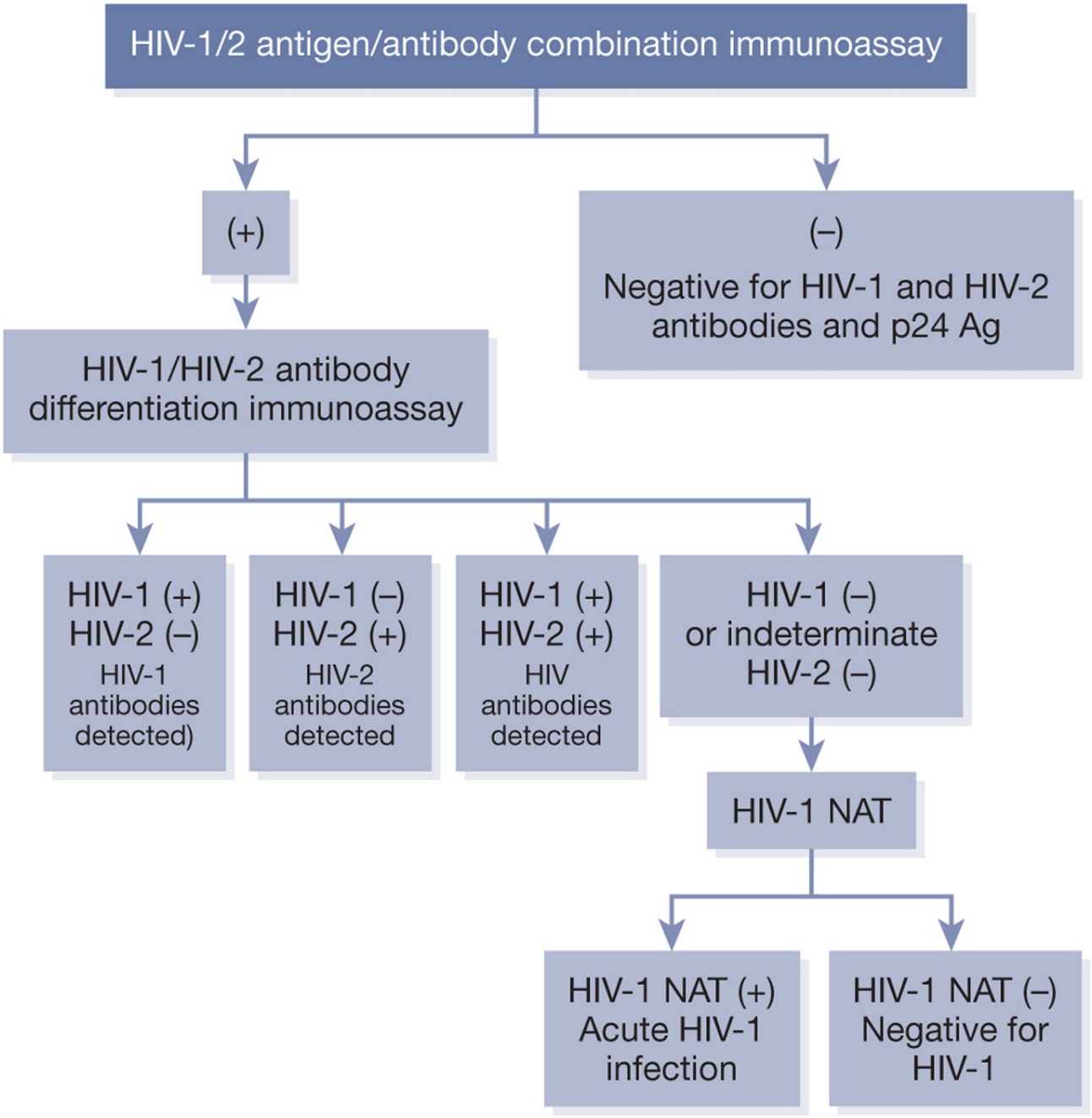


Figure 14-5 Recommended laboratory HIV testing algorithm for serum or plasma specimens.

Abbreviations: HIV, human immunodeficiency disease; NAT, nucleic acid test; +, reactive test result; –, nonreactive test result.

Reproduced with permission from Centers for Disease Control and Prevention. Laboratory testing for the diagnosis of HIV infection: updated recommendations. 2014. Available at: http://www.cdc.gov/hiv/pdf/guidelines_testing_recommendedlabtestingalgorithm.pdf. Accessed March 31, 2017.*83*

Most of the commonly used screening tools detect antigens or antibodies to HIV, rather than the presence of the virus itself, and are appropriate first tests for most individuals. Antibodies appear 2 to 12 weeks after infection occurs. Routine screening can be performed with in-office test kits using oral fluids, blood, or serum or with laboratory testing on blood specimens. In-office rapid testing allows provision of results in 20 minutes. The most commonly used

laboratory screening test is a conventional/rapid EIA. More rarely, a CLIA may be used. The provider should select the one that screens for both HIV-1 and HIV-2.[83] Note that if an individual is newly infected (as would be the case if a woman presents with acute retroviral syndrome), these antigen/antibody tests can provide a false-negative result, as they do not detect the actual virus.

All of the screening tests for HIV have a risk of false-positive results. Therefore, if a screening test is positive, it is repeated. If both tests are positive, a diagnostic test is performed to ensure the person has HIV infection. Appropriate supplemental tests for confirmation of HIV-positive status include HIV-1/HIV-2 antibody differentiation, Western blot testing, and indirect immunofluorescence assay. A positive supplementary test, such as Western blot, indirect immunofluorescence assay, or HIV RNA assay, provides the definitive diagnosis of HIV infection. If the confirmatory test is indeterminate, an HIV RNA test is performed.[83] Reporting to public health authorities is mandated when HIV infection is diagnosed, but not when a screening test is positive.[83]

If a woman has had a recent exposure to HIV and is experiencing symptoms of acute retroviral syndrome (fever, malaise, lymphadenopathy, and skin rash), she should be tested using an HIV-1 RNA assay and an antibody test. Home HIV-testing kits detect antibodies that are not present during the acute phase. Diagnosis during this initial phase is important, however, because the person is highly infectious and prompt treatment can decrease viral spread and may improve outcomes.[7]

Negative HIV test results can be conveyed by phone, but positive results should be given to the individual in person, if at all possible. At the time of diagnosis, it is best to provide contact information for a clinic, physician, or healthcare team who can offer ongoing care. In the past, an HIV diagnosis was often perceived as a death sentence, but today this infection is managed like a chronic condition needing ongoing treatment and care. Even so, HIV infection still carries significant stigma and fear. Telling a woman she has HIV infection is difficult even for experienced clinicians.

Proper preparation prior to the visit can be helpful. Often, local or regional organizations provide specific behavioral and psychological services for HIV-positive individuals and can be contacted to provide information prior to meeting with the woman. The midwife should gather needed written materials to supplement the conversation and provide the woman with reference materials in case she cannot remember the full details of the conversation. Midwifery presence and therapeutic communication skills are useful in providing an honest diagnosis while remaining compassionate and adjusting the information to the woman's needs. Similar to providing information about other STIs, the midwife should provide the woman with the diagnosis and allow her time to react and formulate questions.

The CDC recommends that a diagnosis of HIV infection be provided in conjunction with key health education points, listed in **Table 14-19**. Essential health education points should be discussed verbally and reinforced with written materials. All women with a new diagnosis of HIV should also be screened for intimate-partner violence and a discussion of how to disclose HIV status to sexual partner(s) should be explored.[84] If the woman is at risk for intimate-partner violence, resources and ways to assure safety are a priority for this visit. Information about disclosure to a sexual partner, safe sex practices, risk reduction, PrEP, and PEP can be

found on the CDC website, and the health department's partner services can be helpful as well.[85] The information provided should be compatible with the woman's educational level, culture, and desire for knowledge; health education can be prioritized if the woman appears to be overwhelmed or disinterested.

Table 14-19	Health Education for a Woman with HIV Diagnosis
Essential Teaching at Time of Diagnosis (Verbally and Written Handouts)	
Effectiveness of HIV treatments	
Potential for transmission to others, safer sex practices, risk-reduction strategies, and the availability of pre-exposure prophylaxis for partners	
Partner disclosure techniques and assistance	
Importance of early and ongoing health care	
Where to get health care for HIV infection (local clinic and provider name and phone number)	
What to expect with HIV care (daily medications even when well, preventive visits)	
Referrals to behavioral and psychosocial services designed for HIV-positive individuals	
Additional Needed Teaching When the Woman Is Ready	
Information on health insurance and healthcare provision for HIV-positive individuals	
Reproductive choices with HIV diagnosis, including need for birth control until ready to conceive	
Substance abuse screening and counseling if indicated	

Abbreviation: HIV, human immunodeficiency virus.

Based on U.S. Public Health Service. *Preexposure Prophylaxis for the Prevention of HIV Infection in the United States—2014: A Clinical Practice Guideline.* Washington, DC: US Public Health Service; 2014*78*; Centers for Disease Control and Prevention. Recommendations for partner services programs for HIV infection, syphilis, gonorrhea, and chlamydial infection. *MMWR Recomm Rep.* 2008;57(RR-9):1-83[84]; Centers for Disease Control and Prevention, Health Resources and Services Administration, National Institutes of Health, et al. Recommendations for HIV prevention with adults and adolescents with HIV in the United States, 2014: summary for clinical providers. 2014. Available at: http://stacks.cdc.gov/view/cdc/26063. Accessed March 31, 2017.[86]

Treatment of HIV Infection

Midwives should refer all women with a new HIV diagnosis to appropriate local or regional specialists and verify that these individuals have initiated care.[7] Treatment for HIV infection is dependent on the woman's current health status, including her viral load and comorbid disorders. Referral to an HIV specialist allows the woman to receive optimal care.[86]

Although the majority of midwives in the United States do not routinely care for HIV-positive individuals, some do work in multidisciplinary teams to provide holistic and comprehensive care to HIV-positive women during pregnancy and across the life span. Midwives in these settings collaborate with, consult with, or refer to a variety of healthcare providers to ensure women with HIV infection receive care at facilities that are accessible, are acceptable, and provide evidence-based care.

Women who are HIV positive, who are sexually active, and who do not desire pregnancy need contraception. While condoms are an essential part of safer sex practices, their effectiveness in prevention of pregnancy may not be adequate. The WHO and CDC eligibility

criteria list a variety of contraceptives that are safe with HIV infection, and the CDC recommendations can be accessed via a smartphone app. All contraceptive methods, other than spermicides, are acceptable for clinically well women receiving antiretroviral treatment. If the woman is not receiving treatment or is ill, the IUD may not be an ideal choice, but can be used.[78] Spermicides are implicated in STI acquisition and are not very effective in pregnancy prevention, so their use is not advised.[87] If an HIV-positive woman wants to conceive, preconception planning is essential to optimize her own health, avoid birth defects from teratogenic medications, and prevent transmission of HIV to her partner and fetus.

Women with HIV infection face a wide range of potential sequelae from this infection. Because HIV cannot be eradicated from the body, infection is lifelong. Treatment to prevent immune system depletion and opportunistic infections is ongoing and requires close collaboration between the woman and her healthcare team. Preventive care, including Pap tests and physical examinations, is more frequent for HIV-positive women due to increased susceptibility to infections and cancer.[7]

Management of HIV Infection During the Perinatal Period

All women should be offered screening for HIV early during pregnancy and, ideally, again in the third trimester. There are wide racial health disparities in vertical transmission in the United States, and ensuring women are tested and then welcomed into woman-centered, affirming, and culturally appropriate care is essential in reducing these disparities.[88]

Women who are HIV positive should receive enhanced prenatal care. They can benefit from ancillary counseling and services in addition to traditional prenatal care. While pregnancy does not affect HIV progression to AIDS, HIV infection has been associated with poor perinatal outcomes, especially in low-resource areas of the world.[89]

Early and consistent antiretroviral treatment is an essential component of HIV management during pregnancy and prevention of vertical transmission.[90] Women with HIV infection need an early ultrasound to confirm their due date because a scheduled cesarean section before labor is an option for decreasing vertical transmission. Noninvasive testing for fetal anomalies is appropriate at the same intervals as for women who are HIV negative. Invasive fetal testing, such as amniocentesis, is reserved for situations where noninvasive testing has shown abnormalities.[90]

Prenatal care for women with HIV infection should also involve practitioners skilled in HIV management and pregnancy care. Ideal care involves multidisciplinary collaboration between clinicians and behavioral and psychological health professionals. Midwives can be a component of this care, but the knowledge needed for HIV management is beyond the American College of Nurse-Midwives' Core Competencies. If advanced skills are practiced, they should be added to practice guidelines using the procedures outlined in the Standards for the Practice of Midwifery.[91] Assessment of viral load at term is indicated to determine the ideal route of birth. Cesarean section prior to the start of labor for all women with a viral load of more than 1000 copies/mL is recommended.[7] The combination of antiretroviral medications and cesarean birth for women with a high viral load has reduced vertical transmission from rates as high as

25% to 2% or less in the United States.

Women who have not been screened during pregnancy or women at risk for HIV infection without a third-trimester screening should be screened while in labor. A rapid test is appropriate, as it will yield results in time to begin intrapartum interventions to decrease transmission. While a positive rapid test warrants intrapartum treatment, a set of full screening and diagnostic tests should be performed for definitive diagnosis.[7]

If a laboring woman is HIV positive, or suspected to be HIV positive, antiretroviral medications should be initiated. If the woman's viral load is known to be more than 1000 copies/mL, a cesarean birth following several hours of antiretroviral treatment decreases the risk of vertical transmission.[90] If a vaginal birth is planned, it is appropriate to minimize risk to the infant by facilitating a physiologic birth. Fetal scalp electrodes, episiotomies, and forceps/vacuum-assisted birth are associated with increased transmission to the fetus and should be avoided.[90]

Newborns should be treated with antiretroviral therapy as soon as possible after birth, preferably within 6 to 12 hours of birth.[90] Oral zidovudine (AZT, Retrovir) is the drug of choice for term infants whose mother received prenatal prophylaxis with antiretroviral agents. Guidelines call for zidovudine treatment to continue for 6 weeks.[90] Consultation with pediatric care providers or infectious disease specialists is appropriate. Newborns of mothers treated with antiretroviral medications can develop a variety of problems, including anemia, jaundice, and birth defects, and need evaluation by clinicians experienced in HIV care.

Breastfeeding exposes the infant to HIV through breast milk and can transmit the virus. In areas where safe breast milk substitutes are consistently available, breastfeeding is not recommended. This is the case in the United States and throughout the developed world; the CDC recommends that HIV-positive women in the United States do not breastfeed their infants.[7]

Worldwide, however, the majority of cases of HIV/AIDs occur in areas where safe breast milk substitutes are not reliably available. Infants and children fed breast milk substitutes in these areas may have higher rates of morbidity and mortality when compared with their breastfed peers. In these circumstances, the risk of death from other infections, especially diarrheal illness, is higher than the risk of HIV acquisition from breastfeeding, especially if the mother is taking antiretroviral medication.[92] Local or country-level health officials should decide whether the breastfeeding benefits outweigh the risks in their particular area.[92]

If the child is already known to be HIV positive, he or she should be breastfed as long as the mother and infant desire. However, if the infant is HIV negative or has an unknown HIV status, the risk of HIV acquisition through breastfeeding accumulates as the infant ages. The mother's viral load and CD4 count play a role in maternal–child transmission of the virus, and antiretroviral therapy decreases transmission through breast milk. The mother's personal health, her environment, and the availability of safe feeding alternatives influence the decision of when to wean the infant.[92]

Zika Virus

Zika virus is the most recent virus identified as being transmitted via sexual activity. Zika is a single-stranded RNA arbovirus related to yellow fever and dengue. The virus is transmitted by the bite of *Aedes* species of mosquitos, maternal-to-child transmission, blood transfusion, or sexual contact. Most Zika virus cases are found in the southeastern portion of the United States, and in several nations in Southeast Asia and South America.[93]

Zika virus infection is generally mild and self-limited. The incubation period is approximately 3 to 12 days, and viremia lasts for several days to 1 week. However, during pregnancy, this virus can cross the placenta and infect the cells of the fetus's central nervous system. Zika infection during pregnancy is associated with microcephaly, ventriculomegaly, ophthalmologic abnormalities, intrauterine growth restriction, and stillbirth.[94] Active surveillance and research into Zika infection are being conducted, and the pathologic features of this virus continue to be the subject of investigation. The estimated risk of vertical transmission appears to be low (0.5%),[95] but the incidence of any birth defect or abnormality in the fetus is in the range of 5% to 40% if Zika infection is confirmed during pregnancy. The factors that affect transmission are largely unknown.

There is no vaccine and no antiviral prophylaxis therapy that prevents Zika infection at this time. Zika virus disease is a reportable disorder in the United States. Suspected or confirmed cases of Zika infection should be reported to the health department. Information about prevention, travel recommendations, and treatment can be found on the CDC website.

Pregnant women should avoid travel to areas in which Zika virus has been identified, use preventive measures to avoid mosquito bites, and use condoms if their sex partner lives or travels to an area where the Zika virus is present. The CDC maintains guidelines for screening of women who are not pregnant and women who are pregnant based on travel or residence in an area associated with Zika virus, reported exposure, and symptoms. These guidelines are updated frequently.[96,97] Physician consultation should be obtained for any woman who reports a risk of being exposed to this virus.

Treatment of Zika virus disease is supportive. In pregnant women, care is focused on surveillance for vertical transmission and fetal effects. Healthcare workers should follow universal precautions to protect against exposure to body fluids.[98] More information can be found on the CDC website listed in the Resources section at the end of this chapter.

Protozoal and Parasitic Infections

Several organisms classified as protozoans or parasites can cause reproductive tract infections. Trichomoniasis is the most common protozoan-related STI. Lice are parasitic organisms that may infect the head, body, or pubic area depending on the type of louse. Specifically, *Pthirus pubis* is the type of louse that infects the pubic area. Although *Pthirus pubis* is commonly spread via sexual contact, this organism can be transmitted via contact with linen or other objects. Although infections with the parasites scabies and *Molluscum contagiosum* are not traditional STIs, these organisms are often transmitted via sexual contact.

Trichomoniasis

Trichomoniasis, commonly known as “trich,” is a vaginal or urethral infection caused by the protozoa *Trichomonas vaginalis* (**Figure 14-6**). The pear-shaped protozoa have five flagella and are highly motile. They are transmitted through sexual contact, which transfers infected secretions to the urethra and vagina. Fomites are a possible, but unlikely, mechanism of infection, as trichomonads must be kept moist and warm. More than 2.3 million women in the United States have trichomoniasis at any given time; in some populations, 13% of women are infected.[7] *Trichomonas* infection is not required to be reported, however, so incidence/prevalence estimates lack precision. Trichomoniasis is associated with increased risk for preterm birth and a three-fold increase in risk of HIV acquisition.[7]

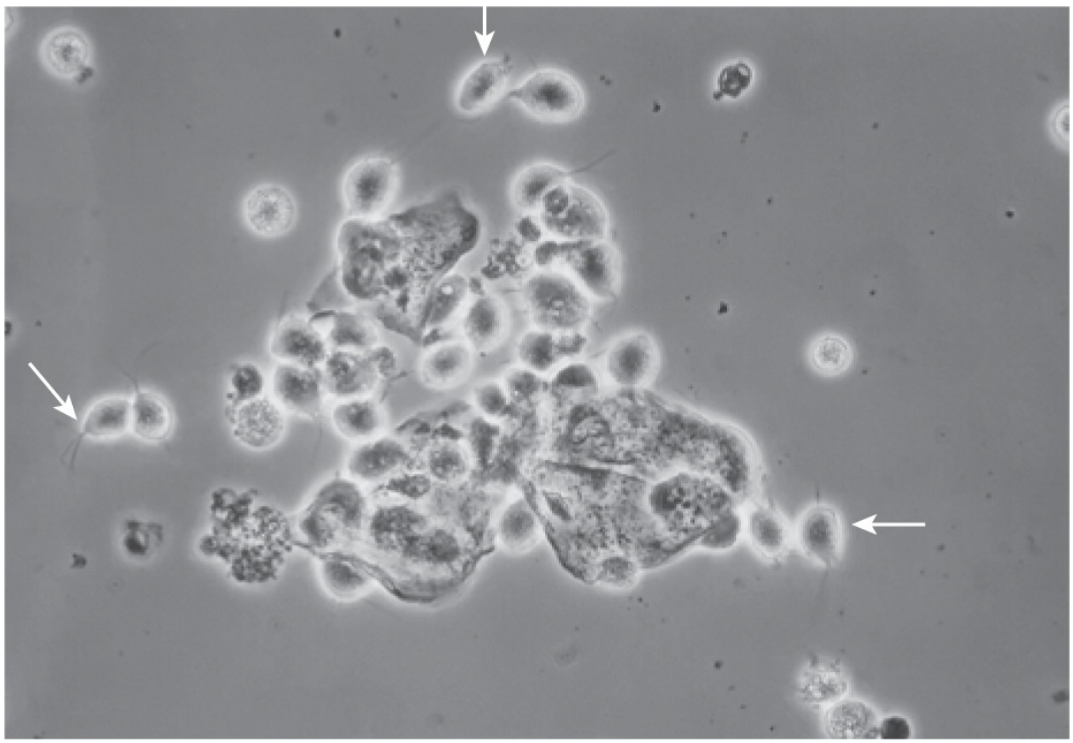


Figure 14-6 Trichomonas.

Courtesy of CDC/Joe Miller.

Trichomoniasis is frequently asymptomatic. Symptoms, when present, include vaginal itching, irritation, and malodorous vaginal discharge. Abdominal discomfort may also be present.

Screening and Diagnosis of Trichomoniasis

Routine screening for trichomoniasis is recommended only for women who are HIV positive because this coinfection is associated with an increased risk for PID. This recommendation applies to both nonpregnant and pregnant women. Routine screening and treatment have not been shown to lower risks of other adverse outcomes.

Women who have symptoms can be evaluated via physical examination and one of several

laboratory tests. Although the woman may be asymptomatic, common physical examination findings include an inflamed and irritated vulva with minor excoriations, and possibly a frothy, thin, yellow-green vaginal discharge. The vagina and cervix may be a deep red or pink, known as “strawberry cervix.”

The CDC recommends the use of tests specific for detection of *Trichomonas vaginalis*. NAAT tests are very sensitive. Other tests, such as the APTIMA assay, the OSOM *Trichomonas* Rapid Test, and Affirm VP III, are also approved for diagnosis using samples from a variety of sources, including the vagina, endocervix, and urine. These tests can be performed in clinic laboratories for on-site diagnosis. While these tests are expensive, they provide greater diagnostic certainty.

A saline wet-mount microscopic evaluation has historically been used to diagnose trichomoniasis and is still commonly employed. The criteria for diagnosis with this method are (1) vaginal pH higher than 4.5, (2) positive whiff test, and (3) motile trichomonads visualized under the microscope. It is important to remember, however, that the absence of these criteria does not rule out infection because the detection rate is only 65% with this method.[7] Trichomonads are very sensitive to salinity and temperature. If the saline has been left open and become more concentrated, the trichomonads will be lysed. If the sample becomes cold, the trichomonads will not be mobile and will be difficult to distinguish from other cells. The CDC advises clinicians to move toward more sensitive and specific tests when possible.

While the presence of *Trichomonas vaginalis* may be reported on Pap tests, this finding should not be used for diagnosis due to high rates of false-positive and false-negative findings.[7]

Treatment of Trichomoniasis

Treatment options for trichomoniasis are listed in **Table 14-20**. Oral therapies are required, as topical treatment with metronidazole is not effective in eradicating infection. Counseling includes avoidance of alcohol for 24 hours after treatment with metronidazole (Flagyl) and for 72 hours after treatment with tinidazole (Tindamax).[7] Current sexual partner(s) should be treated, even if they are asymptomatic. Both partners should abstain from sexual contact until after treatment is completed and symptoms have resolved. If the initial treatment fails to eliminate the symptoms and trichomonads, consider tinidazole or the longer course of metronidazole. For strains resistant to both these drugs, contact the CDC for susceptibility tests and additional treatments.[7] Rescreen women 3 months after treatment, if possible, to test for reinfection.

Table 14-20 **Treatments for Trichomoniasis in Adults**

Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women Generic (Brand)
Metronidazole (Flagyl) 2 g orally once	Metronidazole (Flagyl) 500 mg orally twice a day for 7 days	Metronidazole (Flagyl) 2 g orally once Treat only symptomatic women or women with HIV infection. Treatment does not seem to improve perinatal outcomes and may be associated with preterm birth.	Oral metronidazole (Flagyl) taken in high doses during breastfeeding is not recommended due to active metabolites transferred to the infant via breast milk. The woman may want to consider not breastfeeding for 12–24 hours after taking the large dose of metronidazole. Women can choose the alternative of 500 mg twice a day for 7-day treatment and time breastfeeding to coincide with low plasma levels of the drug.
Tinidazole (Tindamax) 2 g orally in a single dose (Tinidazole is more expensive but has higher cure rates.)			Women should not breastfeed for 72 hours after taking tinidazole (Tindamax). Metronidazole is preferred secondary to the higher maternal plasma levels that occur with the tinidazole 2 g single dose.

Abbreviation: HIV, human immunodeficiency virus.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

Management of Trichomoniasis During the Perinatal Period

Only women with symptoms of trichomoniasis or women who are HIV positive need to be screened for this infection during pregnancy. Infection with *Trichomonas* has been associated with an increased risk for prelabor rupture of membranes, preterm birth, and low birth weight as well as greater vertical transmission of HIV. However, treatment with metronidazole (Flagyl) does not appear to decrease preterm birth rates and may increase the risk of this outcome.[99] Until she is treated, the woman can still transmit the infection. Partner treatment can occur at any time but may need to be repeated when the woman is treated to prevent reinfection.[7]

Transfer of trichomoniasis to a fetus or newborn during vaginal birth is rare but can happen. The newborn may present with fever and either respiratory or genital infection.

Pubic Lice (Pthiriasis)

Pubic lice (*Pthirus pubis*; **Figure 14-7**), also known as "crabs," are found on coarse body hairs, including pubic, axillary, and occasionally facial hair (eyebrows, eyelashes, or beard). Women usually present with itching and report of visible lice or nits. Sexual transmission is most common, as parasites are pushed onto a new host's hair. However, pubic lice can live for as long as 44 hours off the body, so transmission via fomites such as towels, linens, and other objects is possible. Scalp infestations are not common but can occur, especially in advanced cases.

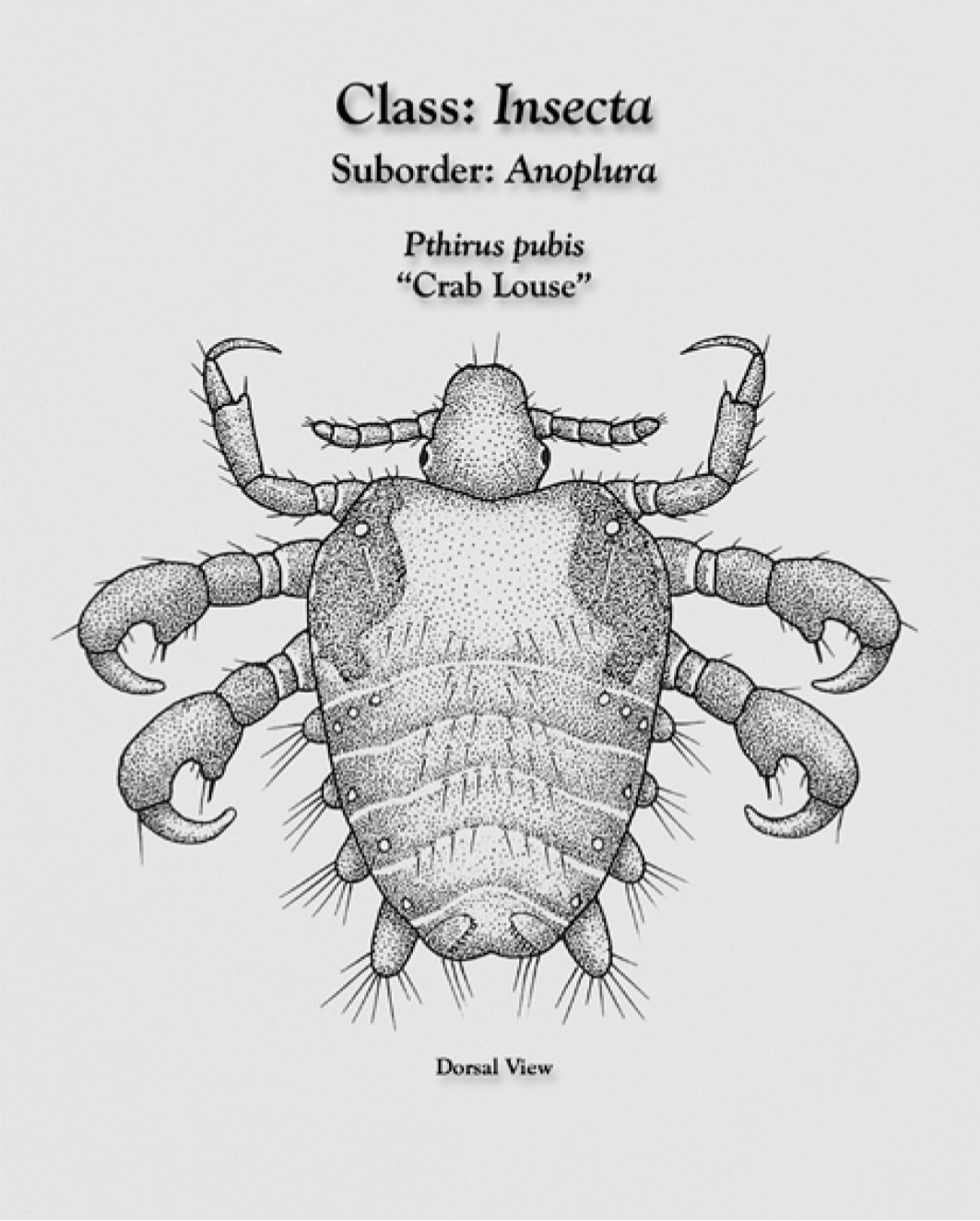


Figure 14-7 Pubic lice.

Courtesy of CDC.

While rare, head lice can occasionally be found in the genital area, especially if pubic hair is fine. Head lice (*Pediculus humanus capitis*; **Figure 14-8**) and pubic lice can be differentiated by their appearance, as pubic lice have a characteristic crab-like appearance and have a lower length-to-width ratio than head lice.

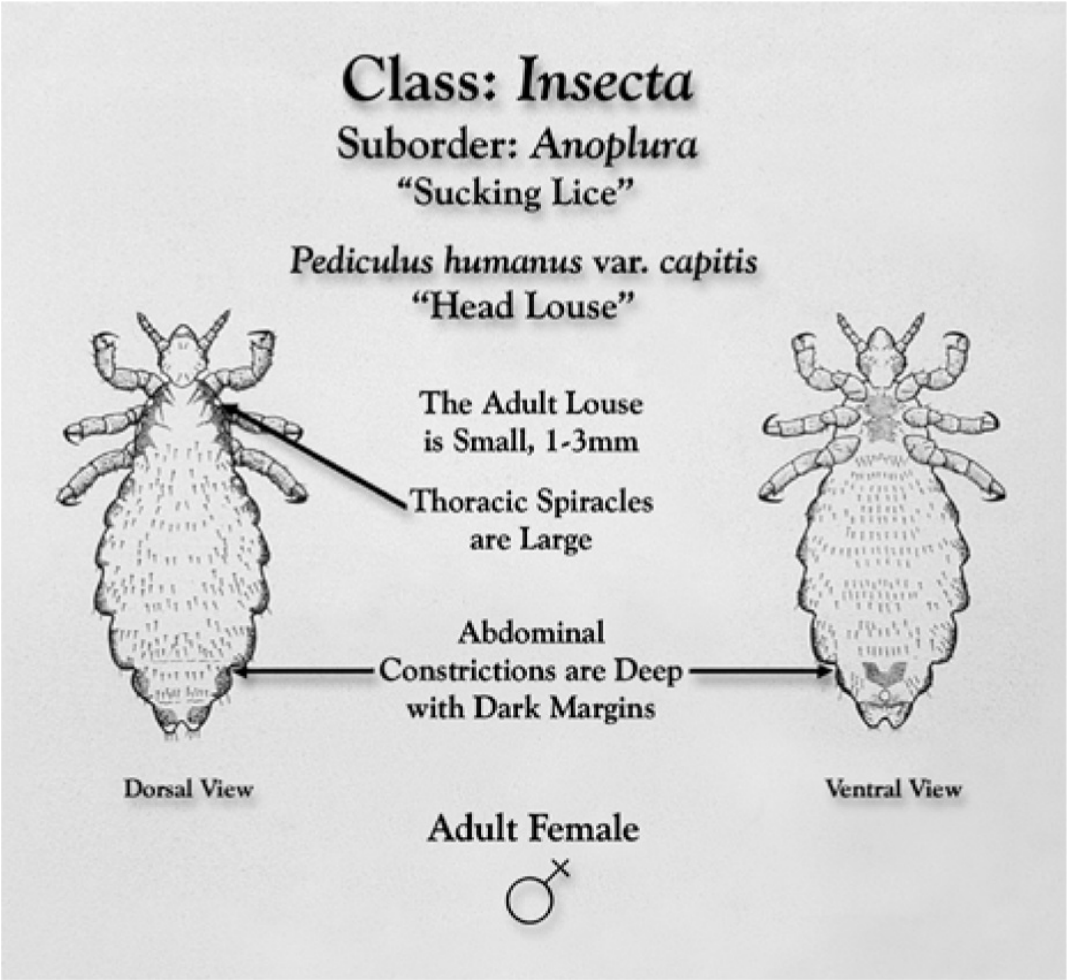


Figure 14-8 Head lice.

Courtesy of CDC.

Diagnosis of Pubic Lice

All hairy or shaved areas of the body are inspected to differentiate the type of infection. Lice and nits are visible on hairs in one or more body area(s), with coarse hairs being more frequently infested than scalp hairs. Small punctuate lesions and bluish macules may also be seen. Lice can be viewed under the microscopic for confirmation, but the physical examination is all that is needed for diagnosis.

Treatment of Pubic Lice

Table 14-21 lists the CDC-recommended treatments for all body areas except the eyes. Pubic lice infestation of the eyelashes should be treated with occlusive ophthalmic ointment twice a

day for 10 days.[7] All clothing and bedding should be washed and dried on a hot setting, dry cleaned, or kept from body contact for 72 hours. Bagging items is more affordable for women who do not have a washer or dryer.

Table 14-21 Treatments for Pthiriasis

Recommended Treatments for Nonpregnant Women and Males Generic (Brand)	Alternative Treatments for Nonpregnant Women and Males Generic (Brand)	Recommended Treatments for Pregnant Women Generic (Brand)	Recommended Treatments for Breastfeeding Women Generic (Brand)
Permethrin 1% cream (Nix) rinse applied to affected area and washed off after 10 minutes Pyrethrins with piperonyl butoxide (RID) applied to area and then washed off after 10 minutes	Malathion 0.5% (Ovide) lotion applied for 8–12 hours and washed off (has a strong odor but is useful for resistant cases) Ivermectin (Stromectol) 250 µg/kg orally with food, repeated in 2 weeks	Permethrin 1% cream (Nix) rinse applied to affected area and washed off after 10 minutes Pyrethrins with piperonyl butoxide (RID) applied to area and then washed off after 10 minutes Second-line treatment: Ivermectin (Stromectol) 250 µg/kg orally with food, repeated in 2 weeks; data suggest low risk to fetus during pregnancy Malathion (Ovide) is contraindicated during pregnancy	Permethrin 1% cream (Nix) rinse applied to affected area and washed off after 10 minutes Pyrethrins with piperonyl butoxide (RID) applied to area and then washed off after 10 minutes Second-line treatment: Ivermectin (Stromectol) 250 µg/kg orally with food, repeated in 2 weeks; avoid direct application to the breast Malathion (Ovide) is contraindicated during lactation

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017[7]; U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.[20]

The CDC advises treatment for all sexual partners from the last month. Household contacts should be examined and treated as needed.[7] Women with pubic lice should be counseled that screening for all common STIs is recommended. Retreatment is necessary if no improvement occurs in 1 week. Resistance to treatment is common, and malathion (Ovide) is recommended for retreatment of women who are not pregnant in cases where resistance is suspected.[7] Pregnant women infected with pubic lice should receive third-trimester rescreening for all STIs.

Other Infectious Agents Spread by Sexual Contact

In addition to the STIs reviewed in this chapter, a wide variety of other infections may spread through sexual contact. For example, viruses such as cytomegalovirus (CMV) and Epstein-Barr virus can be transmitted through sharing of bodily fluids. *Molluscum contagiosum* is a pox virus spread through skin-to-skin contact and, in adults, is most often seen on the genitals, upper thighs, and abdomen with sexual transmission.[48]

Parasites commonly seen in primary care can also be transmitted sexually. Scabies mites can be easily transmitted through close genital contact; such infections should be treated using CDC guidelines. Permethrin or ivermectin are first-line treatments for all adults, although permethrin is preferred for pregnant and lactating women.[7] Scabies-related itching and rash typically persist for as long as 2 weeks, but treatment failures are common due to inadequate treatment or resistant.[7] Alternative treatment regimens may be needed for resistant cases.

Intestinal infections and parasites, such as shigellosis and *Giardia*, can be transmitted sexually, especially when there is contact with the anus or mouth. The CDC provides excellent resources for treatment of women with infectious conditions and provides guidance on the need for sexual-partner and household-contact treatment for conditions spread through sexual or close physical contact. Selected conditions that can be transmitted through sexual contact are described in **Table 14-22**.

Table 14-22 Selected Parasitic Infections Spread by Sexual Contact

Parasitic Infection	Symptoms	Physical Examination	Treatment	Follow-up
Scabies (*Sarcoptes scabiei*)	Severe itching of the groin, between fingers, and in body folds. Itching is bilateral and often occurs more at night.	Excoriations surrounding papules, consisting of small linear or curved burrows, may be present. Definitive diagnosis is based on microscopic visualization of the mite obtained from skin scrapings. Secondary infections are common.	Permethrin 5% applied to the body from the neck down and washed off after 8–14 hours (safe during pregnancy and lactation) OR Ivermectin (Stromectol) 200 mcg/kg orally, repeated in 14 days (This treatment is preferred for outbreaks at residential facilities.) Household linens need to be dried using the hot setting or isolated for 72 hours.	Itching may persist for 2–4 weeks. If treatment fails, lindane is recommended as a second treatment in otherwise well, nonpregnant and nonlactating women.
Molluscum contagiosum	Lesions can be found on any part of the body. With sexual transmission, they are more common on the legs, abdomen, and genital area.	Multiple 2- to 5-mm, shiny white or flesh-colored papules with an indented (umbilicated) center. A thick white discharge can be expressed from the center of the lesion. Large and extensive lesions are more common in HIV-positive adults.	Symptoms are usually self-limiting and resolve without scarring in more than 90% of otherwise healthy adults. Treatments are not usually recommended but can decrease the spread of the infection. Treatments include cryotherapy, excision, oral cimetidine, and topical creams. Transmission can be decreased by covering lesions and not sharing towels or clothing.	None needed; lesions usually heal within 6–12 months.

Abbreviation: HIV, human immunodeficiency virus.

Based on Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep.* 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017.[7]

Infections That Mimic Sexually Transmitted Infections in Genital Areas

Many primary care conditions produce symptoms in the groin region that can mimic STIs. For example, herpes zoster (commonly known as shingles) looks identical to HSV, except that the lesions are along one or more dermatomes. *Trombica* mite bites, commonly known as chiggers, cause itchy, uncomfortable lesions in the groin that may be a presenting concern. Other conditions, such as tinea cruris and other forms of dermatitis, can manifest on and around the vulva. While targeting the examination to the woman's chief concern, the clinician should not prematurely rule out primary care diagnosis that may have genital manifestations. **Table 14-23** lists some conditions that are not STIs, but may appear as such and should be considered in a differential diagnosis.

Table 14-23 Diagnoses That May Mimic Sexually Transmitted Infections That Are Not Sexually Transmitted

Diagnosis	Presenting Concerns and History	Physical Examination	Treatment Generic (Brand)
Chiggers (*Trombicula* mite bites)	Severe itching usually 3–24 hours after outdoor activity in hot, humid weather.	Small, grouped, excoriated papules along panty-line or in fold of groin, often found along a line of clothing constriction.	Irritation is self-limiting. Topical antihistamines and corticosteroids may provide relief. Oral antihistamines can be used in severe cases.
Herpes zoster ("shingles")	Open lesions that are painful superficially and have a deeper nerve pain component. Often occurs in elderly or immunosuppressed women. 10–15% of affected individuals will have postherpetic neuralgia following an initial outbreak.	Painful papules, or vesicles that rupture and crust over. Lesions may be in various stages of forming and healing. Found on L5 and S1 dermatomes. Indistinguishable from herpes by examination. Serologic tests will be negative for HSV infection.	Treatment within 72 hours is ideal to decrease incidence of postherpetic neuralgia: Acyclovir (Zovirax) 800 mg five times a day Famciclovir (Famvir) 500 mg three times a day Valacyclovir (Valtrex) 100 mg three times a day Appropriate pain control medications Lesions are contagious via the air and physical contact, and can cause varicella in previously uninfected individuals. Covering the lesions reduces transmission risk.

Abbreviation: HSV, herpes simplex virus.

Resources

Organization	Description	Webpage
Sexually Transmitted Infections		
Centers for Disease Control and Prevention (CDC)	2015 sexually transmitted infection treatment guidelines.	https://www.cdc.gov/std/tg2015/
	Laboratory reporting of pregnancy status for hepatitis B-positive women.	https://www.cdc.gov/hepatitis/hbv/pregstatuslabreporting.htm
	HIV risk reduction tool. This tool allows individuals to explore their risk in detail and learn about risk reduction strategies.	https://wwwn.cdc.gov/hivrisk/
U.S. Library of Medicine: Medline Plus	This website includes resources for consumers and providers on diagnosis and treatment of sexually transmitted infections.	https://medlineplus.gov/sexuallytransmitteddiseases.html
Postexposure Prophylaxis and Occupational Exposure		
Clinician Consultation Center (CCC)	Post-exposure prophylaxis (PEPline). Website provides clinician consultation and expert guidance for managing healthcare-related exposure to HIV.	http://nccc.ucsf.edu/clinician-consultation/pep-post-exposure-prophylaxis/
Centers for Disease Control and Prevention (CDC)	Guidelines for occupational postexposure prophylaxis.	http://www.cdc.gov/niosh/topics/bbp/guidelines.html
Zika Virus		
Centers for Disease Control and Prevention (CDC)	Information for healthcare providers about screening and testing for Zika virus. Includes considerations for healthcare workers and occupational exposure.	https://www.cdc.gov/zika/hc-providers/index.html
	CDC website for healthcare professionals and consumers that summarizes health risks, travel advisories, and prevention strategies for Zika virus.	https://www.cdc.gov/zika/index.html
ZikaCare Connect	In collaboration with the March of Dimes, this website provides resources and referrals to healthcare providers who are actively involved in caring for women exposed to Zika virus during pregnancy.	https://www.zikacareconnect.org
Apps		
Centers for Disease Control and Prevention (CDC)	2015 Sexually transmitted infection treatment guidelines.	https://itunes.apple.com/us/app/std-tx-guide/id655206856?mt=8

Agency for Healthcare Research and Quality (AHRQ)	ePSS is a mobile app that allows the user to find U.S. Preventive Services Task Force recommendations for screening and treatment.	https://epss.ahrq.gov/PDA/index.jsp

References

1. UN General Assembly. *Transforming our world: the 2030 Agenda for Sustainable Development*. New York, NY: United Nations; 2015.
2. World Health Organization. *Global health sector strategy on sexually transmitted infections, 2016–2021: towards ending STIs*. Geneva, Switzerland: WHO Document Production Services; 2016.
3. U.S. Department of Health and Human Services. *Healthy people 2020*. Washington, DC: U.S. Department of Health and Human Services; 2012.
4. Centers for Disease Control and Prevention. *Sexually transmitted diseases surveillance 2014*. Atlanta, GA: U.S. Department of Health and Human Services; 2015:4-29.
5. World Health Organization. *WHO guidelines for the treatment of* Neisseria gonorrhoeae. Report No.: 9241549696. Geneva, Switzerland: World Health Organization; 2016.
6. Centers for Disease Control and Prevention. The NCHHSTP atlas. 2015. Available at: http://www.cdc.gov/nchhstp/atlas/about-atlas.html. Accessed March 31, 2017.
7. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep*. 2015;64(3):1-137. Available at: https://www.cdc.gov/std/tg2015/tg-2015-print.pdf. Accessed March 31, 2017.
8. Centers for Disease Control and Prevention. A guide to taking a sexual history. Available at: https://www.cdc.gov/std/treatment/sexualhistory.pdf. Accessed March 31, 2017.
9. Centers for Disease Control and Prevention. Brief sexual history tool. Available at: https://www.cdc.gov/actagainstaids/pdf/campaigns/hssc/hssc_sexualhistorytool_v4.pdf. Accessed March 31, 2017.
10. Lewis FMT, Bernstein KT, Aral SO. Vaginal microbiome and its relationship to behavior, sexual health, and sexually transmitted diseases. *Obstet Gynecol*. 2017;129(4):643-654.
11. Onderdonk AB, Delaney ML, Fichorova RN. The human microbiome during bacterial vaginosis. *Clin Microbiol Rev*. 2016;29(2):223-238.
12. Martin DH, Marrazzo JM. The vaginal microbiome: current understanding and future directions. *J Infect Dis*. 2016;214(suppl 1):S36-S41.
13. Verstraelen H, Swidsinski A. The biofilm in bacterial vaginosis: implications for epidemiology, diagnosis and treatment. *Curr Opin Infect Dis*. 2013;26(1):86-89.
14. Muzny CA, Sunesara IR, Kumar R, et al. Characterization of the vaginal microbiota among sexual risk behavior groups of women with bacterial vaginosis [erratum, *PLoS One*. 2013;8(12). doi:10.1371/annotation/f7674ab1-fbd5-4293-ad2c-d1a795962e8b]. *PLoS One*. 2013;8(11):e80254. doi:10.1371/journal.pone.0080254. eCollection 2013.
15. Manns-James L. Bacterial vaginosis and preterm birth. *J Midwifery Womens Health*. 2011;56(6):575-583.
16. Amsel R, Totten PA, Spiegel CA, Chen KC, Eschenbach D, Holmes KK. Nonspecific vaginitis: diagnostic criteria and microbial and epidemiologic associations. *Am J Med*. 1983;74(1):14-22.
17. Gutman RE, Peipert JF, Weitzen S, Blume J. Evaluation of clinical methods for diagnosing bacterial vaginosis. *Obstet Gynecol*. 2005;105(3):551-556.
18. Mohammadzadeh F, Dolatian M, Jorjani M, Alavi Majd H. Diagnostic value of Amsel's clinical criteria for diagnosis of bacterial vaginosis. *Glob J Health Sci*. 2014;7(3):8-14.
19. Kampan NC, Suffian SS, Ithnin NS, Muhammad M, Zakaria SZ, Jamil MA. Evaluation of BV® Blue Test Kit for the diagnosis of bacterial vaginosis. *Sex Reprod Healthcare*. 2011;2(1):1-5.
20. U.S. National Library of Medicine. Drugs and lactation database (LactMed). 2016. Available at: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm. Accessed June 6, 2017.
21. Senok AC. Probiotics for the treatment of bacterial vaginosis. *Cochrane Database Syst Rev*. 2009;4:CD006289. doi:10.1002/14651858.CD006289.pub2
22. Brocklehurst P, Gordon A, Heatley E, Milan SJ. Antibiotics for treating bacterial vaginosis in pregnancy. *Cochrane Database Syst Re*v. 2013;1:CD000262.
23. Sobel JD. Recurrent vulvovaginal candidiasis. *Am J Obstet Gynecol* 2016;214(1):15-21.
24. Castelo-Branco C, Cancelo MJ, Villero J, Nohales F, Juliá MD. Management of post-menopausal vaginal atrophy and atrophic vaginitis. *Maturitas*. 2005;52:46-52.
25. Lethaby A, Ayeleke RO, Roberts H. Local oestrogen for vaginal atrophy in postmenopausal women. *Cochrane Database Syst Rev*. 2016;8:CD001500. doi:10.1002/14651858.CD001500.pub3
26. Adams D, Fullerton K, Jajosky R, et al. Summary of notifiable infectious diseases and conditions—United States, 2013. *MMWR*. 2015;62:1-122.
27. U.S. Department of Health and Human Services, Office of Inspector General. The Ryan White Care Act: implementation of the spousal notification requirement. 1999. Available at: https://oig.hhs.gov/oei/reports/oei-05-98-00391.pdf. Accessed February 20, 2017.
28. American College of Obstetricians and Gynecologists. Committee Opinion No. 632: expedited partner therapy in the management of gonorrhea and chlamydial infection. *Obstet Gynecol*. 2015;125:1526-1528.

29. Handsfield HH, Hogben M, Schillinger J, Golden MR, Kissinger P, Sparling FF. *Expedited partner therapy in the management of sexually transmitted diseases*. Atlanta, GA: Centers for Disease Control and Prevention; 2006.
30. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
31. Centers for Disease Control and Prevention. Legal status of expedited partner therapy. 2016. Available at: https://www.cdc.gov/std/ept/legal/default.htm. Accessed March 31, 2017.
32. Minichiello V, Rahman S, Hawkes G, Pitts M. STI epidemiology in the global older population: emerging challenges. *Perspect Public Health*. 2012;132(4):178-181.
33. Spaulding AC, Miller J, Trigg BG, Braverman P, et al. Screening for sexually transmitted diseases in short-term correctional institutions: summary of evidence reviewed for the 2010 Centers for Disease Control and Prevention Sexually Transmitted Diseases Treatment Guidelines. *Sex Transm Dis*. 2013;40(9):679-684.
34. Borland R, Zimmerman C. *Caring for Trafficked Persons: Guidance for Health Care Providers Training Facilitator's Guide*. Geneva, Switzerland: International Organization for Migration; 2012.
35. Mathews C, Coetzee N, Zwarenstein M, Lombard C, Guttmacher S. Strategies for partner notification for sexually transmitted diseases. *Cochrane Database Syst Rev*. 2015;4:1-33.
36. American College of Obstetricians and Gynecologists. Executive summary: collaboration in practice: implementing team-based care: report of the American College of Obstetricians and Gynecologists' Task Force on Collaborative Practice. *Obstet Gynecol*. 2016;127(3):612-617.
37. Patton ME, Kidd S, Llata E, et al. Extragenital gonorrhea and chlamydia testing and infection among men who have sex with men—STD Surveillance Network, United States, 2010–2012. *Clin Infect Dis*. 2014;58(11):1564-1570.
38. Brunham RC, Gottlieb SL, Paavonen J. Pelvic inflammatory disease. *N Engl J Med*. 2015;2015(372):2039-2048.
39. Papic M, Wang N, Parisi SM, Baldauf E, Updike G, Schwarz EB. Same-day IUD placement is rarely complicated by pelvic infection. *Women's Health Issues*. 2015;25(1):22-27.
40. Tepper NK, Steenland MW, Gaffield ME, Marchbanks PA, Curtis KM. Retention of intrauterine devices in women who acquire pelvic inflammatory disease: a systematic review. *Contraception*. 2013;87(5):655-660.
41. World Health Organization. WHO guidelines for the treatment of *Treponema pallidum* (syphilis). Report No.: 9241549807. Geneva, Switzerland: World Health Organization; 2016.
42. Bowen V, Su J, Torrone E, Kidd S, Weinstock H. Increase in incidence of congenital syphilis—United States, 2012–2014. *MMWR*. 2015:64(44);1241-1245.
43. Gomez GB, Kamb ML, Newman LM, Mark J, Broutet N, Hawkes SJ. Untreated maternal syphilis and adverse outcomes of pregnancy: a systematic review and meta-analysis. *Bull WHO*. 2013;91(3):217-226.
44. Tsimis ME, Sheffield JS. Update on syphilis in pregnancy. *Birth Defects Res*. 2017;109(5):347-352.
45. Rac MW, Revell PA, Eppes CS. Syphilis during pregnancy: a preventable threat to maternal–fetal health. *Am J Obstet Gynecol*. 2017;216(4):352-363.
46. Michele N, Ho His-en, Desai M. Penicillin-desensitization: treatment of syphilis in pregnancy in penicillin-allergic patients. *Ann Allergy Asthma Immunol*. 2017;118:537-541.
47. Butler T. The Jarisch–Herxheimer reaction after antibiotic treatment of spirochetal infections: a review of recent cases and our understanding of pathogenesis. *Am J Trop Med Hyg*. 2017;96(1):46-52.
48. Basta-Juzbaši´c A, C˘eovi´c R. Chancroid, lymphogranuloma venereum, granuloma inguinale, genital herpes simplex infection, and *Molluscum contagiosum*. *Clin Dermatol*. 2014;32(2):290-298.
49. Pytynia KB, Dahlstrom KR, Sturgis EM. Epidemiology of HPV-associated oropharyngeal cancer. *Oral Oncol*. 2014;50(5):380-386.
50. Roura E, Castellsagué X, Pawlita M, et al. Smoking as a major risk factor for cervical cancer and pre-cancer: results from the EPIC cohort. *Int J Cancer*. 2014;135(2):453-466.
51. Petrosky E, Bocchini JA Jr, Hariri S, et al. Use of 9-valent human papillomavirus (HPV) vaccine: updated HPV vaccination recommendations of the Advisory Committee on Immunization Practices. *MMWR*. 2015;64(11):300-304.
52. Shah KV. A case for immunization of human papillomavirus (HPV) 6/11–infected pregnant women with the quadrivalent HPV vaccine to prevent juvenile-onset laryngeal papilloma. *J Infect Dis*. 2013:1307-1309.
53. Bradley H, Markowitz LE, Gibson T, McQuillan GM. Seroprevalence of herpes simplex virus types 1 and 2—United States, 1999–2010. *J Infect Dis*. 2013:325-333.
54. Fanfair RN, Zaidi A, Taylor LD, Xu F, Gottlieb S, Markowitz L. Trends in seroprevalence of herpes simplex virus type 2 among non-Hispanic blacks and non-Hispanic whites aged 14 to 49 years—United States, 1988 to 2010. *Sex Transm Dis*. 2013;40(11):860-864.
55. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 82: management of herpes in pregnancy. *Obstet Gynecol*. 2016;109(6):1489-1498.
56. Chi CC, Wang SH, Delamere FM, Wojnarowska F, Peters MC, Kanjirath PP. Interventions for prevention of herpes simplex labialis (cold sores on the lips). *Cochrane Database Syst Rev*. 2015;8:CD010095. doi:10.1002/14651858.CD010095.pub2

57. Pinninti SG, Kimberlin DW. Management of neonatal herpes simplex virus infection and exposure. *Arch Dis Child Fetal Neonatal*. 2014;99(3):F240-F244.
58. Centers for Disease Control and Prevention. Neonatal herpes simplex virus infection following Jewish ritual circumcisions that included direct orogenital suction—New York City, 2000–2011. *MMWR*. 2012;61(22):405-409.
59. World Health Organization. *Global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis*. Geneva, Switzerland: World Health Organization, 2016. Available at: http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf. Accessed March 31, 2017.
60. Centers for Disease Control and Prevention. Viral hepatitis surveillance. 2016. Available at: http://www.cdc.gov/hepatitis/statistics/2014surveillance/pdfs/2014hepsurveillancerpt.pdf. Accessed March 31, 2017.
61. Jaffe A, Brown RS. A review of antiviral use for treatment of chronic hepatitis B virus infection in pregnant women. *Gastroenterol Hepatol*. 2017;13(3):154-159.
62. Brook MG, Mutimer D. Viral hepatitis: screening and vaccination strategies. In: Zenilman JM, Shahmanesh M, eds. *Sexually Transmitted Infections*. Sudbury, MA: Jones & Bartlett Learning; 2012:241-250.
63. AAP Committee on Fetus and Newborn, ACOG Committee on Obstetric Practice. *Guidelines for perinatal care*. 7th ed. Elk Village, IL: American Academy of Pediatrics Books; 2012.
64. Centers for Disease Control and Prevention, American College of Obstetricians and Gynecologists. Screening and referral algorithm for hepatitis B virus (HBV) infection among pregnant women. 2015. Available at: https://www.cdc.gov/hepatitis. Accessed March 31, 2017.
65. Weinbaum CM, Williams I, Mast EE, et al. Recommendations for identification and public health management of persons with chronic hepatitis B virus infection. *MMWR Recomm Rep*. 2008;57(RR08):1-20.
66. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 86: viral hepatitis in pregnancy. *Obstet Gynecol*. 2007;110(4):941. [Reaffirmed 2016].
67. Society for Maternal–Fetal Medicine, Dionne-Odom J, Tita AT, Silverman NS. SMFM Consult Series # 38: hepatitis B in pregnancy screening, treatment, and prevention of vertical transmission. *Am J Obstet Gynecol*. 2016;214(1):6-14.
68. Brown RS, McMahon BJ, Lok AS, et al. Antiviral therapy in chronic hepatitis B viral infection during pregnancy: a systematic review and meta–analysis. *Hepatology*. 2016;63(1):319-333.
69. Sun W, Zhao S, Ma L, et al. Telbivudine treatment started in early and middle pregnancy completely blocks HBV vertical transmission. *BMC Gastroenterol*. 2017;17(1):51.
70. Chamroonkul N, Piratvisuth T. Hepatitis B during pregnancy in endemic areas: screening, treatment, and prevention of mother-to-child transmission. *Paediatr Drugs*. 2017;19(3):173-181.
71. Centers for Disease Control and Prevention. Hepatitis B vaccination recommendation for infants, children and adolescents. *MMWR Recomm Rep*. 2005;54:27-30.
72. Mohsen W, Levy MT. Hepatitis A to E: what's new? *Intern Med J*. 2017;47(4):380-389.
73. Prasad MR. Hepatitis C virus screening in pregnancy: is it time to change our practice? *Obstet Gynecol*. 2016;128(2):229-230.
74. Ly KN, Jiles RB, Teshale EH, Foster MA, Pesano RL, Holmberg SD. Hepatitis C virus infection among reproductive-aged women and children in the United States, 2006 to 2014. *Ann Intern Med*. 2017;166(11):775-782.
75. Koneru A, Nelson N, Hariri S, Canary L, et al. Increased hepatitis C virus (HCV) detection in women of childbearing age and potential risk for vertical transmission—United States and Kentucky, 2011–2014. *MMWR*. 2016;65(28):705-710.
76. Centers for Disease Control and Prevention. Revised recommendations for HIV testing of adults, adolescents, and pregnant women in health-care settings. *MMWR Recomm Rep*. 2006;55(RR14):1-17.
77. U.S. Public Health Service. *Preexposure Prophylaxis for the Prevention of HIV Infection in the United States—2014: A Clinical Practice Guideline*. Washington, DC: US Public Health Service; 2014.
78. Curtis KM. US medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(3):1-104.
79. Centers for Disease Control and Prevention. Provider information sheet: PrEP during conception, pregnancy, and breastfeeding. 2016. Available at: https://www.cdc.gov/hiv/pdf/prep_gl_clinician_factsheet_pregnancy_english.pdf. Accessed March 31, 2017.
80. Kuhar DT, Henderson DK, Struble KA, et al. Updated US Public Health Service guidelines for the management of occupational exposures to human immunodeficiency virus and recommendations for postexposure prophylaxis. *Infect Control Hosp Epidemiol*. 2013;34(9):875-892.
81. Schillie S, Murphy TV, Sawyer M, et al. CDC guidance for evaluating health-care personnel for hepatitis B virus protection and for administering postexposure management. *MMWR Recomm Rep*. 2013;62(10):1-19.
82. Centers for Disease Control and Prevention, U.S. Department of Health and Human Services. Updated guidelines for antiretroviral postexposure prophylaxis after sexual, injection drug use, or other nonoccupational exposure to HIV—United States, 2016. 2016. Available at: https://stacks.cdc.gov/view/cdc/38856. Accessed March 31, 2017.
83. Centers for Disease Control and Prevention. Laboratory testing for the diagnosis of HIV infection: updated recommendations. 2014. Available at: http://www.cdc.gov/hiv/pdf/guidelines_testing_recommendedlabtestingalgorithm.pdf. Accessed March 31, 2017.

84. Centers for Disease Control and Prevention. Recommendations for partner services programs for HIV infection, syphilis, gonorrhea, and chlamydial infection. *MMWR Recomm Rep*. 2008;57(RR-9):1-83.
85. American College of Obstetricians and Gynecologists. Committee Opinion No. 632: expedited partner therapy in the management of gonorrhea and chlamydial infection. *Obstet Gynecol*. 2015;125:1526-1528.
86. Centers for Disease Control and Prevention, Health Resources and Services Administration, National Institutes of Health, et al. Recommendations for HIV prevention with adults and adolescents with HIV in the United States, 2014: summary for clinical providers. 2014. Available at: http://stacks.cdc.gov/view/cdc/26063. Accessed March 31, 2017.
87. Wilkinson D, Ramjee G, Tholandi M, Rutherford G. Nonoxynol-9 for preventing vaginal acquisition of HIV infection by women from men (Cochrane Review). *Cochrane Database Syst Rev*. 2016;3:11.
88. Anderson LM, Adeney KL, Shinn C, Safranek S, Buckner-Brown J, Krause LK. Community coalition-driven interventions to reduce health disparities among racial and ethnic minority populations. *Cochrane Database Syst Rev*. 2015;6:CD009905. doi:10.1002/14651858.CD009905.pub2
89. Calvert C, Ronsmans C. The contribution of HIV to pregnancy-related mortality: a systematic review and meta-analysis. *AIDS*. 2013;27(10):1631-1639.
90. Panel on Treatment of HIV-Infected Pregnant Women and Prevention of Perinatal Transmission. Recommendations for use of antiretroviral drugs in pregnant HIV-1–infected women for maternal health and interventions to reduce perinatal HIV transmission in the United States. 2016. Available at: http://aidsinfo.nih.gov/guidelines. Accessed March 31, 2017.
91. American College of Nurse-Midwives, Division of Standards and Practice. *Standards for the practice of midwifery*. Silver Spring, MD: American College of Nurse Midwives; 2011.
92. World Health Organization, UNAIDS, UNICEF. *Principles and recommendations for infant feeding in the context of HIV and a summary of evidence: principles and recommendations for infant feeding in the context of HIV and a summary of evidence*. Geneva, Switzerland: World Health Organization; 2010. Available at: http://www.who.int/maternal_child_adolescent/documents/9789241599535/en/. Accessed November 10, 2016.
93. Metsky HC, Matranga CB, Wohl S, et al. Zika virus evolution and spread in the Americas. *Nature*. 2017;546(7658):411-415.
94. Rather IA, Lone JB, Bajpai VK, Park YH. Zika virus infection during pregnancy and congenital abnormalities. *Front Microbiol*. 2017;8:581.
95. Rao R, Gaw SL, Han CS, Platt LD, Silverman NS. Zika risk and pregnancy in clinical practice: ongoing experience as the outbreak evolves. *Obstet Gynecol*. 2017;129(6):1098-1103.
96. Petersen EE, Polen KN, Meaney-Delman D, et al. Update: interim guidance for health care providers caring for women of reproductive age with possible Zika virus exposure—United States, 2016. *MMWR*. 2016;65:315-322. http://dx.doi.org/10.15585/mmwr.mm6512e2.
97. Oduyebo T, Petersen EE, Rasmussen SA, et al. Update: interim guidelines for health care providers caring for pregnant women and women of reproductive age with possible Zika virus exposure—United States, 2016. *MMWR*. 2016;65:122-127. http://dx.doi.org/10.15585/mmwr.mm6505e2.
98. Zorrilla CD, Mosquera AM, Rabionet S, Rivera-Viñas J. HIV and Zika in pregnancy: parallel stories and new challenges. *Obstet Gynecol Int J*. 2016;5(6):00180. doi:10.15406/ogij.2016.05.00180
99. Gülmezoglu AM. Interventions for trichomoniasis in pregnancy. *Cochrane Database Syst Rev*. 2002;3:CD000220. doi:10.1002/14651858.CD000220

CHAPTER

15

Family Planning

MARY C. BRUCKER

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Introduction

The quest for control of reproduction has a long and rich history. Many ancient and modern methods of contraception exist and are well documented. Indeed, there is even a museum dedicated to contraceptive methods.[1] During the mid-twentieth century, the first modern hormonal contraception was developed, and by early twenty-first century, available hormonal methods included new oral hormones, implants, and vaginal rings.[2]

The term *family planning* was chosen as the title for this chapter because this term is the broadest in scope. However, several approaches to presenting the family planning content can be utilized. The focus of this section of the text is primarily on reviewing pregnancy prevention. This material could be presented in different ways, each of which has its own advantages and disadvantages. For example, this material could be presented in order of effectiveness, moving from most to least effective methods. Effectiveness is a very important concept when counseling women about family planning. Alternatively, this content could be subdivided into the methods that are nonhormonal or hormonal, which allows areas of commonality, such as mechanism of action, to be easily identified to better facilitate understanding, especially of side effects such as the unexpected bleeding associated with progestin-only methods of contraception. This is the approach taken in this text, although organizing the chapters covering these issues—*Family Planning*, *Nonhormonal Contraception*, and *Hormonal Contraception*—in this manner is not perfect. For example, some intrauterine devices are nonhormonal and others hormonal; thus, they are covered in both the *Nonhormonal Contraception* and *Hormonal Contraception* chapters, which together provide detailed discussion of the various types of contraceptive methods.

The current chapter presents an overview of family planning, contraception counseling, and factors that affect a woman's choice of a birth control method. Information about options counseling (adoption and abortion) as well as types of surgical and medical (pharmaceutical) abortion are reviewed. New suggested methods in family planning are briefly discussed at the end of the chapter.

Common Terms and Definitions

Prior to discussing details about family planning, it is wise to address the common language used in this area of health care. Although the terms "birth control," "contraception," and "family planning" are often used interchangeably, they are not analogous in meaning, nor are they always used accurately. Some terms are integral to a discussion of family planning, such as "unintended pregnancy." **Table 15-1** provides some terms commonly used in this area and their definitions.[3-5]

Table 15-1 **Definitions of Terms Used in Family Planning**

Term	Definition
Birth control	Limitation of the number of children who are conceived, or ways to control reproduction via use of methods that improve or diminish fertility.
Contraception	Use of specific methods to prevent conception (or *contra-conception*); does not include methods that act after conception, such as interference with or after implantation.
Effectiveness	The success of a method in preventing pregnancy when used "typically," including pregnancies that occur because of incorrect or inconsistent use of a given contraceptive method; poor effectiveness for a contraceptive method usually is attributed to user failure.
Efficacy	The chance that conception will occur despite consistent and correct ("perfect") use of a specific method; poor efficacy for a contraceptive method often is termed true method failure.
Family planning	A broad term generally considered to include methods to assist an individual to plan if and/or when a pregnancy is desired. Most often this involves the use of various contraceptive or birth control methods, but not exclusively.
Long-acting reversible contraceptives (LARCs)	Contraceptive methods generally including implants and intrauterine devices that have high effectiveness and usually do not require direct control of the user. Typical and perfect use statistics are very similar.
Medical Eligibility Criteria for Contraceptive Use	Publication originally developed by the World Health Organization but adapted by different countries. Currently there exists one for the United States as well as a companion piece with recommendations for selective practices.
Pregnancy	"A physiologic state of a woman that follows implantation of a blastocyst(s)."[3] This definition was published by American College of Obstetricians and Gynecologists and has been widely adopted in healthcare publications and guidelines.
Reproductive life plan (RPL)	Similar to the term *family planning*; active planning of when and how many children an individual desires to have. RPLs are a component of general health care for women and men and are an important foundation for the choice of a contraceptive method.
Short-acting reversible contraceptives (SARCs)	Contraceptive methods including daily methods that have less effectiveness than LARCs, owing primarily to their being under the direct control of the individual (e.g., oral contraceptives) and subject to user error. Perfect use generally is much higher than typical use with SARCs.
Unintended pregnancy	A pregnancy that is mistimed or unplanned at the time of conception. This term does not automatically indicate that the pregnancy will not be accepted by the woman.

Based on American College of Obstetricians and Gynecologists. Gynecology data definitions. Available at: https://www.acog.org/-/media/Departments/Patient-Safety-and-Quality-Improvement/reVITALize-Gynecology-

Definitons-V1.pdf?dmc=1&ts=20170814T2340563780. Accessed August 27, 2017[3]; Finer LB, Zolna MR. Declines in unintended pregnancy in the United States, 2008–2011. *N Engl J Med*. 2016;374(9):843-852[4]; Murphy PA, Elmore CE. Contraception. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health.* 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:864-865.[5]

Establishing Efficacy and Effectiveness

The concepts of efficacy and effectiveness are used to describe how well a form of contraception works and often are thought to be synonymous, however, they are distinctly different. *Efficacy* is the chance that conception will occur despite consistent and correct ("perfect") use of a given method. *Effectiveness* describes the success of a method in preventing pregnancy when used "typically," including pregnancies that occur because of incorrect or inconsistent use of a given contraceptive method. To clarify these concepts, efficacy can be viewed as true method failure, while effectiveness is associated with user failure.[5] Certain methods are known to be efficacious, such as oral contraceptives when used perfectly. However, because of real-life factors such as missed pills and drug–drug interactions, their effectiveness may be less than that of other methods. Effectiveness is an important point to consider when the woman's goal is to avoid unintended pregnancy.

Unintended Pregnancy

It has been estimated that approximately half (45%) of all pregnancies in the United States are unintended.[4] Unintended pregnancies occur disproportionately to low income women and those of color. In addition, they are associated with higher risk of a variety of untoward perinatal outcomes and account for a number of the 926,200 abortions reported in 2014.[4,6]

Many women conceive when using a contraceptive method, especially when the method consists of short-acting reversible contraceptives (SARCs). Decreasing the likelihood of unintended pregnancy is a shared goal among many groups and individuals. Essential in assisting women in their choice of identifying an effective method is providing information about the risks of pregnancy associated with specific methods. Although seemingly easy, calculation of the risk of unintended pregnancy associated with a particular contraceptive method is not really simple, in that problems with estimates of efficacy and effectiveness have long been recognized.[7-9]

Calculation of Effectiveness of Contraceptive Methods

The Pearl index, also called the Pearl rate, has been used for more than seven decades in clinical trials to report the effectiveness of a contraceptive method. This calculation is defined as the number of method failures per 100 woman-years of exposure to the method.[8,9] The number of pregnancies that occur is divided by the number of months of contraceptive exposure and then multiplied by 1200 or 1300. When 1300 is used, it is because a menstrual cycle is typically 28 days, which occurs 13 times in a year (365 days ÷ 28 = 13). Based on the statistical formula used, a woman-year is defined by either the number 12 (1200 for the index) to designate months or the number 13 (1300 for the index) to designate average number of

menstrual cycles based on 28 days within a year. A lower Pearl index indicates a lower risk of unintended pregnancy.

Some studies report two Pearl indices, one of which is labeled as typical or actual use for a contraceptive method (effectiveness). The data from which this effectiveness index is calculated include all pregnancies, regardless of whether they involved user error or method failure. Other reports cite the second, "perfect use" Pearl index, which includes data limited to cycles in which there is assurance that the method was correctly and consistently used (efficacy).[8]

Major problems have been found when using the Pearl index to assess a contraceptive method. For example, some assumptions on which the method is based may be invalid. Because of the way the index is calculated, if all of the women became pregnant in the first year, the pregnancy rate would not be a logical 100% or an index of 1000, but rather either 1200 or 1300 depending on the definition of woman-year used in the calculation. Another problem is the mistaken assumption that a failure rate for a method is constant over time. In a large sample of individuals, the most fertile women will get pregnant earlier, and those with lower fertility will become pregnant later, regardless of the type of contraceptive method employed. Furthermore, for some methods such as the diaphragm, the longer the method is used, the more likely women are to become experienced with using it correctly. Conversely, the effectiveness of other contraceptive methods can expire, such as implants, such that these methods are associated with more pregnancies if they are used for periods beyond the recommended length.

In addition, the Pearl index does not recognize other confounding factors. For example, it does not incorporate reasons for self-discontinuation (e.g., side effects), dissatisfaction or change in desire to attempt pregnancy, and loss of women to follow-up who may continue to use the method correctly and successfully avoid pregnancy.

To compensate for some of the problems with the Pearl index, some researchers use life tables for statistical calculations of unintended pregnancy rates.[10] These tables present separate effectiveness rates for each month of study among a group of individuals using a specific contraceptive method over a set period of time (e.g., 12 months). When the results are subdivided by months, the assumption that contraceptive failure rates remain static is corrected. Other statistical analyses using life tables may differentiate between net effectiveness, which allows for comparison of reasons for dropping out of a study, and gross effectiveness, which enables comparison of one study to another. Studies today tend to use both the Pearl index and life table analysis, although many do not clearly designate which approach they use.

In addition to this statistical quagmire, research studies reporting data often are part of clinical trials undertaken for Food and Drug Administration (FDA) approval of a method. Trials involving contraceptives differ from the more traditional drug trials, as discussed in the *Pharmacotherapeutics* chapter. Phase 2 and 3 clinical trials are typically randomized, are often double blinded, and compare an agent to a placebo. In Phase 3 trials, the participants are individuals with a health condition that the new drug is likely to treat. Obvious ethical and methodological problems prohibit the use of this approach for investigation of new contraceptives. Therefore, most clinical trials include "typical" women who are healthy and

seeking a method to use for family planning. The concept of the “typical” participant in clinical trials is also problematic because females younger than the age of 18 years are minors who are subject to pediatric research policies and, therefore, are not included in contraceptive trials. In addition to eliminating most adolescents, clinical trials of contraceptives tend to enroll participants of average weight/body mass index (BMI), even though the United States is experiencing an epidemic of obesity. Exempting women from both of these populations is a limitation for generalizing the findings to all women using contraceptives in the United States.

Another problem is apparent in the manner in which contraceptive methods are compared to one another. The effectiveness of one method can usually be compared to the effectiveness of another method only by using results from previously conducted research, not by conducting a head-to-head study. For example, assume a new oral contraceptive was found to be associated with a 20% rate of amenorrhea in users. Previously published research about an older contraceptive will most likely be used as the basis for comparison with this new contraceptive. Therefore, the assumption is made that both of the studies in question, old and new, have similar methodological approaches and populations. In reality, reasonable comparisons cannot be guaranteed.

Because of the challenges in determining an exact risk of unintended pregnancies associated with a method, quoting statistics to women about the effectiveness of contraceptive methods is an imperfect science. Many midwives use generally agreed-upon ranges of effectiveness. Others present the information by ranking which methods are more effective than others without resorting to exact, and perhaps somewhat inaccurate, numbers. In any case, although the efficacy and effectiveness of methods are critical pieces of information, a woman’s choice of a contraceptive method will include a variety of considerations, including the simple but essential factor of whether she will use a method correctly and consistently.

Fortunately, most studies provide relatively similar results regarding effectiveness and efficacy, and rates found in this chapter are generally agreed upon by several references. **Table 15-2** provides a widely accepted list of effectiveness rates for typical and perfect use of contraceptives.[7]

Table 15-2 Percentage of Women Experiencing an Unintended Pregnancy During the First Year of Typical Use and the First Year of Perfect Use of Contraceptives and the Percentage Continuing Use at the End of the First Year, United States

Method	Women Experiencing Unintended Pregnancy Within the First Year of Use (%), Typical Use[b]	Women Experiencing Unintended Pregnancy Within the First Year of Use (%), Perfect Use[c]	Women Continuing Use at 1 Year (%)[a]
No method[d]	85	85	
Spermicides	28	18	42
Fertility awareness–based methods	24		47
StandardDays method		5	
TwoDay method		4	
Ovulation method		3	
Symptothermal method		0.4	
Withdrawal	22	4	46
Sponge			
Parous women	24	20	46
Nulliparous women	12	9	36
Condom (without spermicide)			
Female	21	5	41
Male	18	2	43
Diaphragm (with spermicide)	12	6	57
Combined pill and progestin-only pill	9	0.3	67
Evra patch	9	0.3	67
NuvaRing	9	0.3	67
Depo-Provera	6	0.2	56
Intrauterine contraceptives			
ParaGard (copper T)	0.8	0.6	78
Mirena (LNG)	0.2	0.2	80
Implanon	0.05	0.05	84
Female sterilization	0.5	0.5	100
Male sterilization	0.15	0.10	100
The lactational amenorrhea method (LAM) is a highly effective, temporary method of contraception.[e]			

[a] Among couples attempting to avoid pregnancy, the percentage of who continue to use a method for 1 year.
[b] Among typical couples who initiate use of a method, the percentage who experience an accidental pregnancy during the first year if they do not stop use for any other reason.
[c] Among typical couples who initiate use of a method and who use it perfectly (both consistently and correctly), the percentage who experience an accidental pregnancy during the first year if they do not stop use for any other reason.
[d] The percentages becoming pregnant in columns 2 and 3 are based on data from populations in which contraception is not used and from women who cease using contraception so as to become pregnant. Among such populations, approximately 89% become pregnant within 1 year. This estimate was lowered slightly (to 85%) to represent the percentage who would become pregnant within 1 year among women now relying on reversible methods of contraception if they abandoned contraception altogether.
[e] To maintain effective protection against pregnancy, another method of contraception must be used as soon as menstruation resumes, the frequency or duration of breastfeeding is reduced, bottle feeding is introduced, or the baby reaches 6 months of age.

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Permanent Versus Reversible Contraceptive Methods

The first categorization of contraceptive methods is permanent versus reversible. Male or female sterilization remains the most efficacious and effective of all contraceptive methods and is the method used by approximately 20.6% of women between age 15 and 44 years.[11] Different types of sterilization are discussed in the *Nonhormonal Contraception* chapter. However, millions of individuals of childbearing age desire a reversible contraceptive method.

The search to identify the most effective contraceptives has led to categorizing reversible contraceptive methods as either long acting (LARCs) or short acting (SARCs). Hormonal contraceptives are among the most commonly used forms of birth control in the United States. In contrast to nonhormonal contraceptives, which tend to be both coital and user dependent, hormonal contraceptive use is separate from sexual activity. Nevertheless, the effectiveness of some of the hormonal methods, such as oral contraceptives, rings, and patches, depends on a woman using them consistently and correctly. These user-dependent types of methods are categorized as SARCs because most failures with these methods are associated with user error, and the perfect use rate is considerably higher than the typical use rate. Depot medroxyprogesterone acetate (DMPA), a contraceptive injection, may be considered a SARC because the user needs to regularly return to a provider to receive injections. By comparison, LARCs, such as intrauterine devices (IUDs) and implants, require a healthcare visit for placement, but are neither coital nor user dependent; they offer years of effectiveness and, therefore, their typical use rates are similar to their perfect use rates. **Figure 15-1** compares the effectiveness of the most commonly used contraceptives; as can be seen in this figure, LARCs are the most effective options.[12]

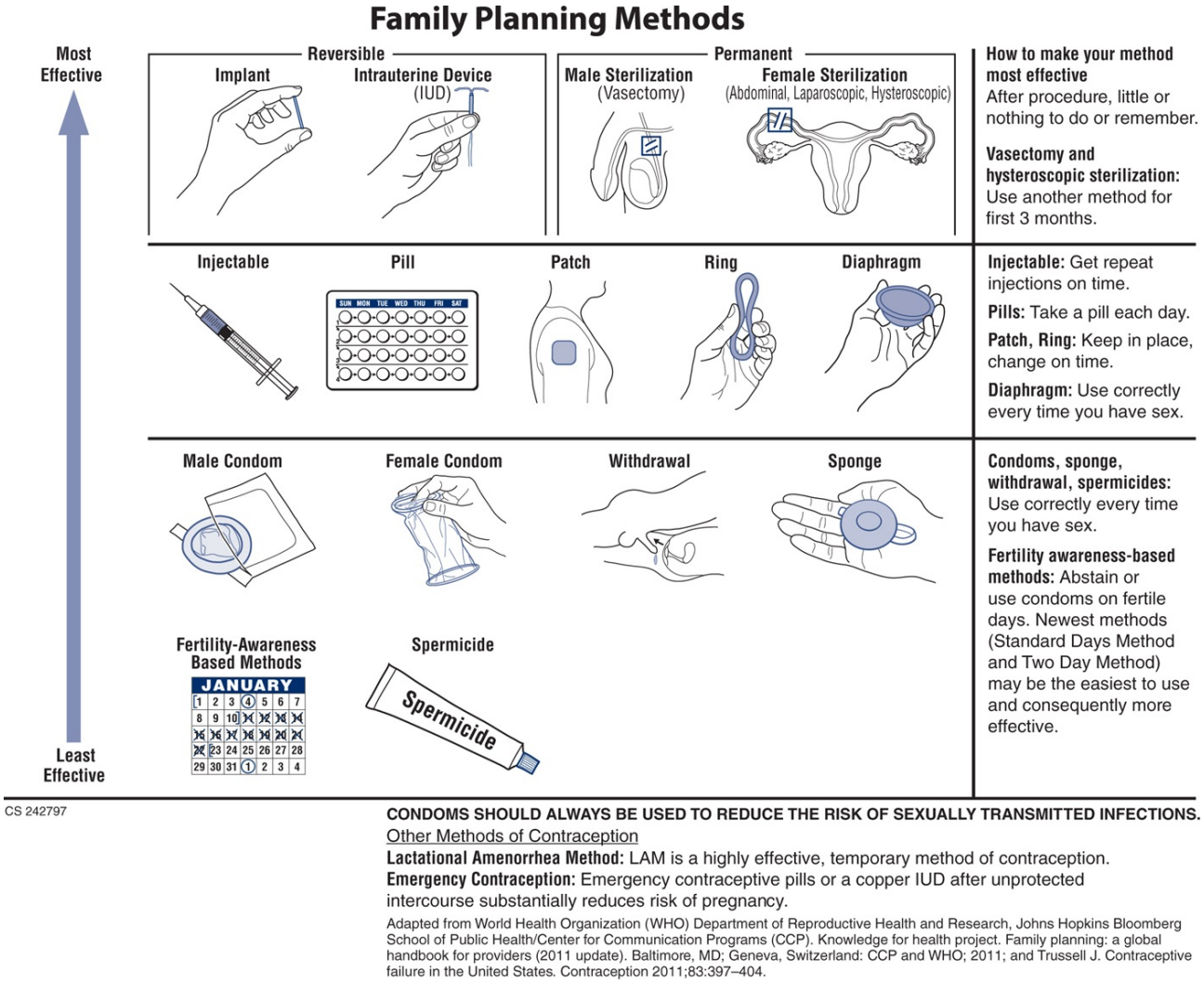


Figure 15-1 Comparing the typical effectiveness of contraceptive methods.

Modified with permission from Centers for Disease Control and Prevention. Effectiveness of family planning methods. Available at: https://www.cdc.gov/reproductivehealth/unintendedpregnancy/pdf/contraceptive_methods_508.pdf. Accessed September 15, 2017.[12]

The Centers for Disease Control and Prevention (CDC) has identified increasing access to LARCs as one of the top public health priorities for reducing adolescent pregnancy and unintended pregnancy in the United States.[13,14] LARCs have been advocated as a first-line recommended form of contraception for the majority of women, regardless of age or parity.[14] Guidelines published by the American College of Obstetricians and Gynecologists state that LARCs should be considered as a first-line option for most women, including adolescents.[15,16] In a 2014 policy statement and technical report, the American Academy of Pediatrics similarly recommended LARCs for adolescents, and subsequent studies have found that young women tend to continue to use the methods for several years.[17,18]

Initially, LARCs are the most expensive reversible methods owing to the healthcare costs incurred for both the devices and the visit for their insertion. However, once implanted or inserted, the annualized cost is the same or less than that for less effective methods. Several studies have found that state funding of free or low-cost LARCs decreases the costs associated with unplanned births and abortions and, therefore, that these methods are cost-effective.[19]

Like any contraceptive method, LARCs are not perfect, nor are they the best choice for every woman. Because they are provider dependent, lack of professionals in an area is a challenge for women seeking these types of contraception in some geographic locales.[20] The fact that major professional organizations are actively promoting use of LARCs has caused some individuals to voice concerns that a clinician might inadvertently urge a woman—especially one who is young or socioeconomically disadvantaged—to use a LARC without appropriately acknowledging her choice.[4,21,22] In addition, some relatively rare conditions exist that preclude the use of implants and intrauterine devices. Fortunately, national medical eligibility criteria for different contraceptive methods provides insight into the advisability of specific contraceptive methods.

Medical Eligibility Criteria for Contraceptive Use

In 1996, the first edition of *Medical Eligibility Criteria for Contraceptive Use* (WHO MEC) was published by the World Health Organization (WHO). This document is regularly updated based on new evidence.[23] The WHO MEC currently is in its fifth edition and is useful especially for midwives working internationally. WHO has encouraged countries to modify the MEC as needed.

In recognition that practice in the United States may differ in some respects from international care, the CDC has published nationally focused guidelines (**Figure 15-2**). Known as the *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016* (U.S. MEC for Contraceptive Use), this publication is available as a free download and is closely related to the WHO MEC. The U.S. MEC for Contraceptive Use website is listed in the Resources section at the end of this chapter. It uses the same categories for eligibility as noted in **Table 15-3**.[24]

Summary Chart of U.S. Medical Eligibility Criteria for Contraceptive Use, 2016

Condition	Sub-Condition	Cu-IUD		LNG-IUD		Implant	DMPA	POP	CHC
		I	C	I	C	I C	I C	I C	I C
Age		Menarche to < 20 yrs: **2** ≥ 20 yrs: **1**		Menarche to < 20 yrs: **2** ≥ 20 yrs: **1**		Menarche to < 18 yrs: **1** 18–45 yrs: **1** > 45 yrs: **1**	Menarche to < 18 yrs: **2** 18–45 yrs: **1** > 45 yrs: **2**	Menarche to < 18 yrs: **1** 18–45 yrs: **1** > 45 yrs: **1**	Menarche to < 40 yrs: **1** ≥ 40 yrs: **2**
Anatomical abnormalities	a) Distorted uterine cavity	**4**		**4**					
	b) Other abnormalities	**2**		**2**					
Anemias	a) Thalassemia	**2**		**1**		**1**	**1**	**1**	**1**
	b) Sickle cell disease‡	**2**		**1**		**1**	**1**	**1**	**2**
	c) Iron-deficiency anemia	**2**		**1**		**1**	**1**	**1**	**1**
Benign ovarian tumors	(*including cysts*)	**1**		**1**		**1**	**1**	**1**	**1**
Breast disease	a) Undiagnosed mass	**1**		**2**		**2***	**2***	**2***	**2***
	b) Benign breast disease	**1**		**1**		**1**	**1**	**1**	**1**
	c) Family history of cancer	**1**		**1**		**1**	**1**	**1**	**1**
	d) Breast cancer‡								
	i) Current	**1**		**4**		**4**	**4**	**4**	**4**
	ii) Past and no evidence of current disease for 5 years	**1**		**3**		**3**	**3**	**3**	**3**
Breastfeeding	a) < 21 days postpartum					**2***	**2***	**2***	**4***
	b) 21 to < 30 days postpartum								
	i) With other risk factors for VTE					**2***	**2***	**2***	**3***
	ii) Without other risk factors for VTE					**2***	**2***	**2***	**3***
	c) 30–42 days postpartum								
	i) With other risk factors for VTE					**1***	**1***	**1***	**3***
	ii) Without other risk factors for VTE					**1***	**1***	**1***	**2***
	d) > 42 days postpartum					**1***	**1***	**1***	**2***
Cervical cancer	Awaiting treatment	**4**	**2**	**4**	**2**	**2**	**2**	**1**	**2**
Cervical ectropion		**1**		**1**		**1**	**1**	**1**	**1**
Cervical intraepithelial neoplasia		**1**		**2**		**2**	**2**	**1**	**2**
Cirrhosis	a) Mild (*compensated*)	**1**		**1**		**1**	**1**	**1**	**1**
	b) Severe‡ (*decompensated*)	**1**		**3**		**3**	**3**	**3**	**4**
Cystic fibrosis‡		**1***		**1***		**1***	**2***	**1***	**1***
Deep venous thrombosis (DVT)/Pulmonary embolism (PE)	a) History of DVT/PE, not receiving anticoagulant therapy								
	i) Higher risk for recurrent DVT/PE	**1**		**2**		**2**	**2**	**2**	**4**
	ii) Lower risk for recurrent DVT/PE	**1**		**2**		**2**	**2**	**2**	**3**
	b) Acute DVT/PE	**2**		**2**		**2**	**2**	**2**	**4**
	c) DVT/PE and established anticoagulant therapy for at least 3 months								
	i) Higher risk for recurrent DVT/PE	**2**		**2**		**2**	**2**	**2**	**4***
	ii) Lower risk for recurrent DVT/PE	**2**		**2**		**2**	**2**	**2**	**3***
	d) Family history (*first-degree relatives*)	**1**		**1**		**1**	**1**	**1**	**2**
	e) Major surgery								
	i) With prolonged immobilization	**1**		**2**		**2**	**2**	**2**	**4**
	ii) Without prolonged immobilization	**1**		**1**		**1**	**1**	**1**	**2**
	f) Minor surgery without immobilization	**1**		**1**		**1**	**1**	**1**	**1**
Depressive disorders		**1***		**1***		**1***	**1***	**1***	**1***

Key:	
1 No restriction (method can be used)	**3 Theoretical or proven risks usually outweigh the advantages**
2 Advantages generally outweigh theoretical or proven risks	**4 Unacceptable health risk (method not to be used)**

Condition	Sub-Condition	Cu-IUD		LNG-IUD		Implant		DMPA		POP		CHC	
		I	C	I	C	I	C	I	C	I	C	I	C
Diabetes	a) History of gestational disease	1		1		1		1		1		1	
	b) Nonvascular disease												
	i) Non-insulin dependent	1		2		2		2		2		2	
	ii) Insulin dependent	1		2		2		2		2		2	
	c) Nephropathy/retinopathy/neuropathy‡	1		2		2		3		2		3/4*	
	d) Other vascular disease or diabetes of > 20 years' duration‡	1		2		2		3		2		3/4*	
Dysmenorrhea	Severe	2		1		1		1		1		1	
Endometrial cancer‡		4	2	4	2	1		1		1		1	
Endometrial hyperplasia		1		1		1		1		1		1	
Endometriosis		2		1		1		1		1		1	
Epilepsy‡	(*see also Drug Interactions*)	1		1		1*		1*		1*		1*	
Gallbladder disease	a) Symptomatic												
	i) Treated by cholecystectomy	1		2		2		2		2		2	
	ii) Medically treated	1		2		2		2		2		3	
	iii) Current	1		2		2		2		2		3	
	b) Asymptomatic	1		2		2		2		2		2	
Gestational trophoblastic disease‡	a) Suspected GTD (immediate postevacuation)												
	i) Uterine size first trimester	1*		1*		1*		1*		1*		1*	
	ii) Uterine size second trimester	2*		2*		1*		1*		1*		1*	
	b) Confirmed GTD												
	i) Undetectable/non-pregnant ß-hCG levels	1*	1*	1*	1*	1*		1*		1*		1*	
	ii) Decreasing ß-hCG levels	2*	1*	2*	1*	1*		1*		1*		1*	
	iii) Persistently elevated ß-hCG levels or malignant disease, with no evidence or suspicion of intrauterine disease	2*	1*	2*	1*	1*		1*		1*		1*	
	iv) Persistently elevated ß-hCG levels or malignant disease, with evidence or suspicion of intrauterine disease	4*	2*	4*	2*	1*		1*		1*		1*	
Headaches	a) Nonmigraine (mild or severe)	1		1		1		1		1		1*	
	b) Migraine												
	i) Without aura (includes menstrual migraine)	1		1		1		1		1		2*	
	ii) With aura	1		1		1		1		1		4*	
History of bariatric surgery‡	a) Restrictive procedures	1		1		1		1		1		1	
	b) Malabsorptive procedures	1		1		1		1		3		COCs: 3 P/R: 1	
History of cholestasis	a) Pregnancy related	1		1		1		1		1		2	
	b) Past COC related	1		2		2		2		2		3	
History of high blood pressure during pregnancy		1		1		1		1		1		2	
History of Pelvic surgery		1		1		1		1		1		1	
HIV	a) High risk for HIV	2	2	2	2	1		1*		1		1	
	b) HIV infection					1*		1*		1*		1*	
	i) Clinically well receiving ARV therapy	1	1	1	1	If on treatment, see Drug Interactions							
	ii) Not clinically well or not receiving ARV therapy‡	2	1	2	1	If on treatment, see Drug Interactions							

Abbreviations: C=continuation of contraceptive method; CHC=combined hormonal contraception (pill, patch, and, ring); COC=combined oral contraceptive; Cu-IUD=copper-containing intrauterine device; DMPA = depot medroxyprogesterone acetate; I=initiation of contraceptive method; LNG-IUD=levonorgestrel-releasing intrauterine device; NA=not applicable; POP=progestin-only pill; P/R=patch/ring ‡ Condition that exposes a woman to increased risk as a result of pregnancy. *Please see the complete guidance for a clarification to this classification: www.cdc.gov/reproductivehealth/unintendedpregnancy/USMEC.htm.

		I	C	I	C	I	C	I	C	I	C	I	C
Hypertension	a) Adequately controlled hypertension	1*		1*		1*		2*		1*		3*	
	b) Elevated blood pressure levels (*properly taken measurements*)												
	i) Systolic 140–159 or diastolic 90–99	1*		1*		1*		2*		1*		3*	
	ii) Systolic ≥ 160 or diastolic ≥ 100‡	1*		2*		2*		3*		2*		4*	
	c) Vascular disease	1*		2*		2*		3*		2*		4*	
Inflammatory bowel disease	(*Ulcerative colitis, Crohn's disease*)	1		1		1		2		2		2/3*	
Ischemic heart disease‡	Current and history of	1		2	3	2	3	3		2	3	4	
Known thrombogenic mutations‡		1*		2*		2*		2*		2*		4*	
Liver tumors	a) Benign												
	i) Focal nodular hyperplasia	1		2		2		2		2		2	
	ii) Hepatocellular adenoma‡	1		3		3		3		3		4	
	b) Malignant‡ (hepatoma)	1		3		3		3		3		4	
Malaria		1		1		1		1		1		1	
Multiple risk factors for atherosclerotic cardiovascular disease	(e.g., older age, smoking, diabetes, hypertension, low HDL, high LDL, or high triglyceride levels)	1		2		2*		3*		2*		3/4*	
Multiple sclerosis	a) With prolonged immobility	1		1		1		2		1		3	
	b) Without prolonged immobility	1		1		1		2		1		1	
Obesity	a) Body mass index (BMI) ≥ 30 kg/m^2	1		1		1		1		1		2	
	b) Menarche to < 18 years and BMI ≥ 30 kg/m^2	1		1		1		2		1		2	
Ovarian cancer‡		1		1		1		1		1		1	
Parity	a) Nulliparous	2		2		1		1		1		1	
	b) Parous	1		1		1		1		1		1	
Past ectopic pregnancy		1		1		1		1		2		1	
Pelvic inflammatory disease	a) Past												
	i) With subsequent pregnancy	1	1	1	1	1		1		1		1	
	ii) Without subsequent pregnancy	2	2	2	2	1		1		1		1	
	b) Current	4	2*	4	2*	1		1		1		1	
Peripartum cardiomyopathy‡	a) Normal or mildly impaired cardiac function												
	i) < 6 months	2		2		1		1		1		4	
	ii) ≥ 6 months	2		2		1		1		1		3	
	b) Moderately or severely impaired cardiac function	2		2		2		2		2		4	
Postabortion	a) First trimester	1*		1*		1*		1*		1*		1*	
	b) Second trimester	2*		2*		1*		1*		1*		1*	
	c) Immediate postseptic abortion	4		4		1*		1*		1*		1*	
Postpartum (*nonbreastfeeding women*)	a) < 21 days					1		1		1		4	
	b) 21 days to 42 days												
	i) With other risk factors for VTE					1		1		1		3*	
	ii) Without other risk factors for VTE					1		1		1		2	
	c) > 42 days					1		1		1		1	
Postpartum (*in breastfeeding or nonbreastfeeding women, including cesarean delivery*)	a) < 10 minutes after delivery of the placenta												
	i) Breastfeeding	1*		2*									
	ii) Nonbreastfeeding	1*		1*									
	b) 10 minutes after delivery of the placenta to < 4 weeks	2*		2*									
	c) ≥ 4 weeks	1*		1*									
	d) Postpartum sepsis	4		4									

		I	C	I	C	I	C	I	C	I	C	I	C
Pregnancy		4*		4*		NA*		NA*		NA*		NA*	
Rheumatoid arthritis	a) On immunosuppressive therapy	2	1	2	1	1		2/3*		1		2	
	b) Not on immunosuppressive therapy	1		1		1		2		1		2	
Schistosomiasis	a) Uncomplicated	1		1		1		1		1		1	
	b) Fibrosis of the liver‡	1		1		1		1		1		1	
Sexually transmitted diseases (STDs)	a) Current purulent cervicitis or chlamydial infection or gonococcal infection	4	2*	4	2*	1		1		1		1	
	b) Vaginitis (*including trichomonas vaginalis and bacterial vaginosis*)	2	2	2	2	1		1		1		1	
	c) Other factors relating to STDs	2*	2	2*	2	1		1		1		1	
Smoking	a) Age < 35	1		1		1		1		1		2	
	b) Age ≥ 35, < 15 cigarettes/day	1		1		1		1		1		3	
	c) Age ≥ 35, ≥ 15 cigarettes/day	1		1		1		1		1		4	
Solid organ transplantation‡	a) Complicated	3	2	3	2	2		2		2		4	
	b) Uncomplicated	2		2		2		2		2		2*	
Stroke‡	History of cerebrovascular accident	1		2		2	3	3		2	3	4	
Superficial venous disorders	a) Varicose veins	1		1		1		1		1		1	
	b) Superficial venous thrombosis (acute or history)	1		1		1		1		1		3*	
Systemic lupus erythematosus‡	a) Positive (or unknown) antiphospholipid antibodies	1*	1*	3*		3*		3*	3*	3*		4*	
	b) Severe thrombocytopenia	3*	2*	2*		2*		3*	2*	2*		2*	
	c) Immunosuppressive therapy	2*	1*	2*		2*		2*	2*	2*		2*	
	d) None of the above	1*	1*	2*		2*		2*	2*	2*		2*	
Thyroid disorders	Simple goiter/ hyperthyroid/hypothyroid	1		1		1		1		1		1	
Tuberculosis‡ *(see also Drug Interactions)*	a) Nonpelvic	1	1	1	1	1*		1*		1*		1*	
	b) Pelvic	4	3	4	3	1*		1*		1*		1*	
Unexplained vaginal bleeding	(suspicious for serious condition) before evaluation	4*	2*	4*	2*	3*		3*		2*		2*	
Uterine fibroids		2		2		1		1		1		1	
Valvular heart disease	a) Uncomplicated	1		1		1		1		1		2	
	b) Complicated‡	1		1		1		1		1		4	
Vaginal bleeding patterns	a) Irregular pattern without heavy bleeding	1		1	1	2		2		2		1	
	b) Heavy or prolonged bleeding	2*		1*	2*	2*		2*		2*		1*	
Viral hepatitis	a) Acute or flare	1		1		1		1		1		3/4*	2
	b) Carrier/Chronic	1		1		1		1		1		1	1
Drug Interactions													
Antiretroviral therapy All other ARV's are 1 or 2 for all methods.	Fosamprenavir (FPV)	1/2*	1*	1/2*	1*	2*		2*		2*		3*	
Anticonvulsant therapy	a) Certain anticonvulsants (phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine)	1		1		2*		1*		3*		3*	
	b) Lamotrigine	1		1		1		1		1		3*	
Antimicrobial therapy	a) Broad spectrum antibiotics	1		1		1		1		1		1	
	b) Antifungals	1		1		1		1		1		1	
	c) Antiparasitics	1		1		1		1		1		1	
	d) Rifampin or rifabutin therapy	1		1		2*		1*		3*		3*	
SSRIs		1		1		1		1		1		1	
St. John's wort		1		1		2		1		2		2	

Updated July 2016. This summary sheet only contains a subset of the recommendations from the U.S. MEC. For complete guidance, see: http://www.cdc.gov/reproductivehealth/unintendedpregnancy/USMEC.htm. Most contraceptive methods do not protect against sexually transmitted diseases (STDs). Consistent and correct use of the male latex condom reduces the risk of STDs and HIV.

CS266008-A

Figure 15-2 Summary chart of U.S. medical eligibility criteria for contraceptive use, 2016.

Reproduced with permission from Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR Recomm Rep.* 2016;65(3):1-103. Available at: https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf. Accessed August 27, 2017.[24]

Table 15-3 **Categories of Medical Eligibility Criteria for Contraceptive Use**

1 = A condition for which there is no restriction for the use of the contraceptive method.

2 = A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.

3 = A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.
4 = A condition that represents an unacceptable health risk if the contraceptive method is used.

Based on World Health Organization. *Medical eligibility criteria for contraceptive use*. 5th ed. Geneva, Switzerland: WHO; 2015. Available at: http://www.who.int/reproductivehealth/publications/family_planning/MEC-5/en/. Accessed August 27, 2017[23]; Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep.* 2016;65(3):1-103. Available at: https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf. Accessed August 27, 2017.[24]

Modifications of the WHO MEC for Contraceptive Use for women in the United States include exclusion of methods not available in the United States and inclusion of several additional health conditions of note in this country. In particular, modifications that address breastfeeding, intrauterine device use, valvular heart disease, ovarian cancer, uterine fibroids, and venous thromboembolism were made to the WHO MEC for Contraceptive Use to reflect the U.S. population and practice. The U.S. MEC for Contraceptive Use also added information about care of women with inflammatory bowel disease, history of bariatric surgery, rheumatoid arthritis, endometrial hyperplasia, history of peripartum cardiomyopathy, and history of solid-organ transplant that is not found in the WHO MEC for Contraceptive Use.[23,24]

A companion to the U.S. MEC for Contraceptive Use is a document focusing on selected practices that, although common, can be complicated. Titled *U.S. Selected Practice Recommendations for Contraceptive Use, 2016* (often abbreviated as SPR), this publication can be found on the CDC's website or downloaded as a free electronic book. The SPR focuses on frequently occurring conditions associated with controversial or complex interventions for women seeking contraception in the United States. The most recent changes to the SPR include guidelines on when to start a method after emergency contraception as well as medications to ease insertion of IUDs.[25]

The U.S. MEC for Contraceptive Use primarily focuses on the safety of use of various contraceptives, especially for both initiation and continuation. The midwife and the woman can quickly verify recommendations for use of a specific method by referring to multiple tables in the publication. Note that the U.S. MEC for Contraceptive Use is a set of guidelines—compliance with its recommendations is not mandatory. Thus, deviations from its recommendations may occur upon considering an individual woman's situation. Nevertheless, variations in practice are becoming rarer and the U.S. MEC for Contraceptive Use guidelines are used widely throughout the United States, augmented with information from the SPR.

Other factors that can be intertwined with eligibility for a contraceptive method include requirements for laboratory testing and physical examinations, both of which can be major barriers for women. Contrary to practices in the late twentieth century, however, such interventions are rarely necessary today. **Table 15-4** lists recommended examinations or tests based on the current SPR.[25]

Table 15-4 Examinations and Tests Recommended Before Contraceptive Initiation

	Copper IUD	LNG IUD	Implant	DMPA	CHC	POP
Examinations						
Blood pressure	Class C	Class C	Class C	Class C	Class A[a]	Class C
Body mass index	[b]	[b]	[b]	[b]	[b]	[b]
Clinical breast examination	Class C	Class C	Class C	Class C	Class C	Class C
Bimanual/cervical inspection	Class A	Class A	Class C	Class C	Class C	Class C
Laboratory Tests						
Glucose	Class C	Class C	Class C	Class C	Class C	Class C
Lipids	Class C	Class C	Class C	Class C	Class C	Class C
Liver enzymes	Class C	Class C	Class C	Class C	Class C	Class C
Hemoglobin	Class C	Class C	Class C	Class C	Class C	Class C
Thrombogenic mutations	Class C	Class C	Class C	Class C	Class C	Class C
Cervical cytology (Pap test)	Class C	Class C	Class C	Class C	Class C	Class C
Screening for sexually transmitted infections	[c]	[c]	Class C	Class C	Class C	Class C
HIV screening	Class C	Class C	Class C	Class C	Class C	Class C

Abbreviations: CHC, combined hormonal contraception; DMPA, depot medroxyprogesterone; IUD, intrauterine device; LNG, levonorgestrel; POP, progestin-only pill.

Class A: Examination or laboratory test result is essential and mandatory in all circumstances for safe and effective use of the contraceptive method.

Class B: Examination or laboratory test result contributes substantially to safe and effective use, but implementation may be considered within the public health and/or service context; the risk of not performing an examination or test should be balanced against the benefits of making the contraceptive method available.

Class C: Examination or laboratory test result does not contribute substantially to safe and effective use of the contraceptive method.

[a] In instances in which blood pressure cannot be measured by a provider, blood pressure measured in other settings can be reported by the woman to her provider.

[b] Body mass index (BMI) is not needed to determine medical eligibility for any method of contraception because all methods can be used or generally can be used by obese women. However, measuring weight and calculating BMI at baseline might be helpful for monitoring any change and counseling women who might be concerned about weight changes perceived to be associated with their contraceptive method.

[c] Most women do not require screening for sexually transmitted disease (STD) at the time of intrauterine device insertion if they have already been screened according to CDC's STD treatment guidelines (http://www.cdc.gov/std/treatment). If a woman has not been screened according to guidelines, screening can be performed at the time of the insertion of the intrauterine device and insertion should not be delayed. Women with purulent cervicitis or current chlamydial infection or gonorrhea should not undergo insertion of the intrauterine device.

Modified with permission from Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. Selected practice recommendations for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(4):1-66. Available at: https://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm. Accessed August 27, 2017.[25]

In summary, many factors are to be considered when providing care for a woman who desires to prevent pregnancy. Although the effectiveness and eligibility of specific methods are important considerations, counseling is a prerequisite to facilitating an informed choice. The best contraception for a woman is one she personally desires and can use correctly and consistently.

Family Planning Counseling and the Role of the Midwife

Hallmarks of midwifery care include concepts such as promoting family-centered care, facilitating empowerment of women as partners in health care, promoting public health, and advocating for shared decision making and the right to self-determination.[26] Responsibilities of the midwife in this area include identification of needs, health education, assisting in decision making, and provision of contraceptive methods. In some settings, midwives may also provide abortion services. To appropriately provide family planning care, midwives must be aware of their own feelings and attitudes about sexuality, gender roles, contraception, religious beliefs, and other beliefs that could affect family planning counseling. Counseling is one of the most critical aspects of family planning care, which has different components depending on the type of counseling provided.

Obtaining a comprehensive health history is the initial step in family planning counseling. This information will allow the midwife to assess for risk factors as outlined in the U.S. MEC for Contraceptive Use and tailor counseling to the individual. Some medical conditions may affect a woman's eligibility for some contraceptive methods. For example, combined hormonal contraceptives are not safe for a woman who has severe hypertension.[24]

Choice of a Specific Contraceptive Method

Not all women who are sexually active are at risk for pregnancy. For example, some have a partner of the same gender, or a heterosexual woman may be engaged in a monogamous relationship with a man who has had a vasectomy. Furthermore, among heterosexual couples who engage in sexual activity that involves vaginal penetration, one or both may be infertile. Despite these caveats, more than 60% of women between age 15 and 44 years use a contraceptive method.[11] To effectively provide any counseling, the midwife must be able to personally detach from the decision that the woman may make, acknowledging that her decision may not be the one the midwife would make or recommend. In fact, the woman's decision may even conflict with the midwife's personal beliefs.

The woman's preference is the major foundation for choice of a specific method.[27] Incorporation of a partner into the process of choosing a contraceptive method is a decision made by the woman. In some cases, midwives may provide education to couples; in other situations, the woman alone consults with the professional. Throughout this chapter, content regarding education and care may be extrapolated to include significant others when their inclusion is preferred by the woman.

Some women prefer not to share information about a chosen contraceptive method with their partners because of personal privacy concerns or fear of contraceptive sabotage. Contraceptive sabotage occurs when a partner or other exercises reproductive coercion over the woman either to conceive or to abort a pregnancy.[28] Women may seek contraceptives because they are anticipating exposure to pregnancy, voluntarily or not. For example, women in the military may be placed in positions where they are vulnerable to sexual violence, or where

a contraceptive-associated amenorrhea may be preferable because of deployment to areas with lack of hygienic conditions.[29,30] Women who have physical or intellectual disabilities also have unique needs in the area of contraception and menstruation.[31,32] These needs may be related to or separate from voluntary sexual activity.

A woman's preference for a specific contraceptive method also can be influenced by a variety of individual factors that should be acknowledged by the midwife. Desired family size and choices about contraceptive methods are strongly influenced by culture, attitudes, and religion.[33] Even though pregnancy is defined by implantation, some women may view pregnancy as starting at conception, and this belief may affect adoption of certain contraceptive methods that do not impede conception based on their mechanism of action. Some women believe exogenous hormones are unnatural and only opt for nonhormonal methods. A woman with several friends and family using one method may gravitate toward that method. Cost and insurance availability can cause women to discount certain methods. These examples are simply a few of the factors that a woman might consider.

Practice varies as to how the informed consent process is acknowledged and documented. Some practices require that women sign consent forms. In any case, informed decision making is essential, and written consent must always be obtained prior to initiation of any invasive method, such as an implant or intrauterine device.

Other Factors a Woman May Consider When Choosing a Contraceptive Method

Counseling should include sharing information about the noncontraceptive advantages of certain methods of which the woman might be unaware or does not readily consider. For example, condoms prevent transmission of sexually transmitted infections (STIs) in addition to their contraceptive action, and combined oral contraceptives (COCs) can decrease a woman's lifetime risk for ovarian cancer. The cost of the method, both for initiation and for long-term maintenance, should also be discussed with the woman. For example, some methods may be covered by health insurance, whereas other methods may result in higher out-of-pocket costs, precluding their use by some women who have lower incomes or lack insurance to defray the cost.

Some personal beliefs about the effects of a contraceptive method may be based on misinterpretation that may be clarified with scientific evidence. Others are deeply held beliefs based on culture or religion, which may in turn influence the woman's choices.

Any one single factor may be of more importance to a woman than another factor. For some women, the major concern is effectiveness of the contraceptive method, and they are willing to experience additional side effects to gain greater effectiveness. For other women, unintended pregnancies would be ill timed but not devastating; in such a case, factors such as cost, ease of use, and acceptability to their partner may contribute more to the decision about which contraceptive method to use.

The presence of or potential for transmitting genetic disorders to offspring may be an additional consideration, and for some women is a factor in choosing permanent sterilization. Of note, personal genetic risk factors may influence a woman's physiologic response to a contraceptive method as well as her personal risk factors. A woman with a strong family

history of coagulation disorders, for example, may choose an intrauterine device as opposed to a contraceptive patch because of the former's lower risk of triggering a thrombotic event. Such a decision recognizes the inheritable nature of the disorder even if the woman does not know her personal risk.

Special Populations

A woman's age can factor into her choice of contraceptive method. Adolescents may choose LARCs because they are discrete. Nonsmoking women in their 30s or 40s may choose oral contraceptives based on the noncontraceptive benefits. Other women who smoke and are older than 35 years should not take COCs based on the U.S. MEC for Contraceptive Use.[24,25] Perimenopausal women may not realize their risk for pregnancy until they receive health education.[34] Women with a mobility disability may have difficulty with insertion of a barrier method.[31,32] Women with intellectual disabilities may have a guardian who can be included in counseling, although every effort should be made to retain the woman's choice.[32]

Table 15-5 summarizes common factors that a woman may consider in the selection of a contraceptive method. Midwives are ideal providers to customize contraceptive messages and support women in their choices.

Table 15-5 Common Factors Involved in Women's Selection of Contraceptive Methods

Factor	Examples
Acceptance of critical aspects of method	View that a daily method or one that is coital dependent is inconvenient
Acceptance of unintended pregnancy	Failure of method is inconvenient but not troublesome to the woman and her family
Access to method and ability to use successfully	Inability of hands to manipulate diaphragm
	Expense of methods and no insurance to cover cost
Beliefs	Desire not to have device reside in the woman's body
	Wariness about using any exogenous hormones
Eligibility based on risk factors	Age and concomitant health conditions identified as Level 3 or 4 in the U.S. *Medical Eligibility Criteria*
Health benefits	Personal risk factors that can be ameliorated by specific methods
Need for discrete method/privacy	Desire not to let the partner or others know of sexual activity or contraception
Need for protection from sexually transmitted infections	Most methods provide contraception but not protection unless condoms are added
Opinions of partner/significant others	Family members' opinions about the benefits or complications of use of a specific method
Past history	Desire to restart or not restart a method
Pattern of sexual activity	Sexual activity infrequent
Side effects and tolerance	Interpretation/tolerance of intermenstrual bleeding; amenorrhea; other common side effects
Support and counseling from healthcare provider	Healthcare provider with a negative opinion about a specific method or about adolescents engaging in sexual intercourse

Time to return to fertility after discontinuation	Desires a pregnancy within a short period, which may preclude certain methods including LARCs

Counseling Women Who Have an Unintended Pregnancy

In most practices, a pregnancy test is performed as part of the standard protocol prior to prescribing or dispensing a contraceptive product. However, if such a laboratory test is not readily available, certain clinical criteria have a negative predictive value of 99% to 100% for the absence of current pregnancy (**Table 15-6**).[25] Even when a negative pregnancy test is obtained, the midwife may desire to document the clinical criteria listed in **Table 15-6** in the woman's health record. If a woman is unintentionally pregnant, options counseling is provided.

Table 15-6	How to Be Reasonably Certain a Woman Is Not Pregnant
A midwife can be reasonably certain that a woman is not pregnant if she has no symptoms or signs of pregnancy and she meets any one of the following criteria:	
Is ≤ 7 days after the start of normal menses	
Has not had sexual intercourse since the start of last normal menses	
Has been correctly and consistently using a reliable method of contraception	
Is ≤ 7 days after spontaneous or induced abortion	
Is within 4 weeks postpartum	
Is fully or nearly fully breastfeeding (exclusively breastfeeding or the vast majority [≥ 85%] of feeds are breastfeeds), amenorrheic, and < 6 months postpartum	

Reproduced with permission from Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2016. *MMWR Recomm Rep.* 2016;65(4):1-66. Available at: https://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm. Accessed August 27, 2017.[25]

Options Counseling

Options counseling is the process by which an individual who is undecided about available choices is provided with information about all available options.[35] When faced with an unintended pregnancy, a woman generally has the following options: She can continue the pregnancy and keep the baby; continue the pregnancy and place the newborn for adoption; or electively terminate the pregnancy. One of the Hallmarks of Midwifery identified by the American College of Nurse-Midwives (ACNM) addresses advocacy for informed choice, shared decision making, and the right to self-determination.[26] In addition, the ACNM Reproductive Health Choices position statement states: "Every woman has the right to access factual, evidence-based, unbiased information about available reproductive health choices, in order to make an informed decision."[26] These standards underscore the midwife's responsibility with regard to counseling, advising, and informing the woman of her options.

It is very important to understand the distinction between options and abortion counseling. The professional responsibility to present options does not mean the midwife must conduct abortion counseling for women who are interested in abortion. Rather, options counseling is a nonjudgmental description of the alternatives, which might be presented with values

clarification exercises, that can help the woman decide which choice is right for her. Principles of options counseling include the ability to actively listen to the woman, provide to her all necessary information from which to make her decision, and support her in evaluating her options.[35] Some midwives do not provide abortion counseling due to personal, religious, or moral conflicts. A delicate balance is involved between respecting the autonomy of the individual midwife who is morally opposed to providing termination counseling, and respecting the woman's right to be informed about all of her family planning options. A midwife whose conscience will not allow provision of abortion counseling needs to inform the woman of that fact, and facilitate the woman in finding another provider who will offer the counseling.

A midwife can therapeutically discuss options with a woman via use of open-ended questions with nonjudgmental verbal and nonverbal communication. It is imperative that the woman is guided through exploration of her own feelings and beliefs with regard to all appropriate options. Assisting her to understand the outcome of each option is important not only for her decision-making process, but also for her ability to accept the option she will eventually choose.

Pregnancy Counseling

Simply because a pregnancy is unintended, it does not mean the child is unwanted or will be unloved. Some women will opt for maintaining the pregnancy and raising the child. For those women, the midwife can provide counseling regarding the woman's health as well as the fetus. In many cases, the visit provides an opportunity to initiate some aspects of prenatal care and arrange for ongoing care. Details regarding antepartum counseling and care can be found in the *Prenatal Care* chapter.

Adoption Counseling

If a pregnant woman chooses to place an infant for adoption, the primary role of the midwife is to connect the woman with the appropriate resources to assist her in creating an adoption plan. Optimally, this activity would occur in collaboration with a social worker or other professional who has knowledge of the full range of services available in the area, though in some communities the midwife may need to personally provide these referrals. Thus, it is incumbent upon the midwife to become familiar with locally available adoption services. Adoption options include open, closed, or semi-open adoption.[36]

- *Open adoption* occurs when the birth mother and the adoptive family are known to each other and have varying levels of communication, ranging from visits to letters or phone calls, although the birth mother does not necessarily have any input about the way the child is parented. Women should understand that in most cases of open adoption, there is no legal requirement for the adoptive family to follow the terms of such an agreement and legal restrictions will vary by state.
- *Closed adoption* is an adoption in which birth records are sealed, and all identities are

concealed.
- *Semi-open adoption* is an adoption in which some identifying information is shared, and communication occurs at prearranged intervals, usually through an intermediary such as an attorney or adoption agency. The degree of communication and "openness" may change over time.

Making the decision to place a baby for adoption is a difficult one, and the woman will need emotional support, both during prenatal care and during the birth and the early postpartum period.

Abortion Counseling

One should never assume that confirmation of pregnancy will be either positive or negative news for an individual. The legalization of abortion means that every woman in the United States, upon becoming pregnant, has a choice to carry the pregnancy to its natural conclusion or abort the pregnancy. Since 1969, the CDC has gathered data regarding abortion. In 2013, the rate in the United States was 2.5 abortions per 1000 women between age 15 and 44 years, or a ratio of 200 abortions per 1000 live births. These statistics represent a decrease of approximately 5% in the number of abortions from the prior year and are the lowest level seen in the last decade. Approximately 60% of pregnancy terminations are obtained by women who have had at least one or more previous children, and 55% have never had an abortion before.[6]

A woman's decision to have an abortion is not made easily. Midwives who desire to provide abortion counseling need to be knowledgeable about local resources, the methods used, and ways to make appropriate referrals as needed. Components of abortion counseling include discussion of method-specific risks, benefits, informed consent, and gestational age limits. Gestational age limits will vary by setting, state regulations, and local availability of trained providers.

Induced Abortion

More than 90% of planned abortions occur in the first trimester. A small percentage (8.6%) are performed after 13 weeks' gestation.[6] In the first trimester, abortions are performed using pharmacologic agents or surgical interventions such as vacuum aspiration or dilation and curettage. After the first trimester, surgical dilation and evacuation is used, potentially requiring a 2-day procedure, with the insertion of osmotic dilators at least 24 hours before the termination of pregnancy. For women whose blood is Rh negative, Rho(D) immunoglobulin (anti-D serum; RhoGAM) is indicated prior to start of either medical or surgical termination.

Surgical Abortion

Manual vacuum aspiration (MVA) may be used for pregnancies that are up to approximately 12 gestational weeks. This method involves using a handheld syringe as the suction source for emptying the uterine contents.[37] MVA has been found to be equally effective as suction curettage for abortion during the first trimester.[37] The procedure takes 5 to 15 minutes, and can be performed in the office setting. It results in removal of the intact gestational sac, thereby enabling confirmation of pregnancy termination.[38] Studies have suggested that nonpharmaceutical methods can augment pharmacologic treatments to mitigate the woman's discomfort during this procedure.[39,40]

Termination using suction dilation and curettage (D & C) is accomplished by dilation of the cervix, followed by curettage to empty the uterine contents. This technique can be used up to 13 gestational weeks. The overall risk of complications is less than 1% for surgical termination. The main complications of surgical abortion are continued intrauterine pregnancy; heavy bleeding; infection, including endometritis; uterine perforation or injury to organs; and risk of retained products of conception, requiring further surgical intervention.[41,42]

Abortion Using Pharmacologic Agents

The number of medical abortions in the United States increased from 2012 to 2013 by 5%, with this technique accounting for more than 22% of legal abortions.[6] In some countries, medical abortion is considered the method of the future and already accounts for the vast majority of induced abortions.[43] Three agents currently are used for medical abortion: mifepristone (Mifeprex, RU-486), methotrexate (Rheumatrex), and misoprostol (Cytotec). A combination of mifepristone and misoprostol was FDA approved in 2000 for women seeking a medical abortion, and in 2016 the FDA released new information about use. According to the FDA approval, oral mifepristone should be administered as a dose of 200 mg. Then, 24 to 48 hours later, 800 mcg of oral misoprostol can be administered at home.[44] At 7 to 14 days post treatment, the woman should be evaluated to confirm that a complete termination has occurred.

Mifepristone inhibits progesterone, which is necessary for normal placental attachment, thereby preventing fertilization and sensitizing the uterus to the effects of prostaglandin.[45] Maximal sensitization occurs at approximately 24 to 48 hours following administration of mifepristone.[46] Misoprostol, a prostaglandin E_1 analogue, causes expulsion of uterine

contents.[23] The two-medication regimen of mifepristone followed by misoprostol has a 95% to 98% effectiveness rate when used by women whose gestations are 63 days or less after their last menstrual period.[47] When used alone, mifepristone is approximately 76% effective; hence it is rarely used as a sole agent.[47] The effectiveness of misoprostol alone varies widely, from 68% to 94%,[47] depending on whether it is used vaginally, it is inserted dry or moistened (most effective), or it is taken orally.[48] When used alone, misoprostol is associated with significantly higher rates of nausea, vomiting, fever, chills, and diarrhea. It has also been found to be a teratogen, being associated with the rare congenital neurological disorder called Mobius syndrome if the pregnancy continues.[49]

Although methotrexate has been used for pregnancy termination, it is not as effective as the other drugs. Methotrexate works by inhibiting dihydrofolate reductase, an enzyme necessary for DNA synthesis, and often is categorized as an antimetabolite or an antifolate agent.[50] Rapidly dividing cells, such as those found in the zygote, morula, or blastocyst, are not able to undergo normal mitosis when exposed to methotrexate. When used alone, this agent's effectiveness ranges from 60% to 84%.[50] This drug is also used for treatment of women with ectopic pregnancies as well as to stop the replication of other rapidly dividing cells such as those found in cancer, although many other chemotherapeutic drugs have replaced it for specific malignancies.

Women who plan a medical abortion are counseled to expect heavy bleeding and significant painful cramping. The bleeding will be heaviest in the first 6 hours after misoprostol administration. Approximately 50% of women report nausea following use of mifepristone. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be effective in mitigating pain, and they do not interfere with the effect of misoprostol. Because there is a risk of teratogenicity if pregnancy is not aborted, women should be counseled about a possible need for surgical abortion in the event of failure of the medical approach.

The contraindications to medical abortion are listed in **Table 15-7**.[50,51] Notably, women who are breastfeeding should avoid nursing their infant for 2 days after using mifepristone and for 4 hours after taking misoprostol. Complications of medical abortion methods may include minor conditions such as nausea and vomiting. Rarely, women experience incomplete expulsion of uterine contents and subsequent need for surgical procedure; uterine infection (less than 1%); and heavy bleeding that requires hemostatic curettage (0.3% to 1.3%).[46] Women should be counseled to notify their provider if they experience continuing heavy bleeding post abortion.

Table 15-7	Contraindications to Medical Abortion
General Contraindications	**Use with Caution**
Previous allergic reaction to one of the drugs involved	Long-term corticosteroid therapy (including in individuals with severe uncontrolled asthma[a])
Inherited porphyria	Hemorrhagic disorder
Chronic adrenal failure	Severe anemia
Known or suspected ectopic pregnancy (neither misoprostol	Preexisting heart disease or cardiovascular risk

nor mifepristone will treat ectopic pregnancy)	factors Intrauterine device in place; remove before beginning the regimen

[a] Severe asthma not controlled by therapy is a recommended contraindication by some organizations, but is not listed as a contraindication by others. According to the American College of Obstetricians and Gynecologists, asthma is not a contraindication because misoprostol is a weak bronchodilator. However, if the woman has severe asthma, a consultation is recommended before proceeding with a medical abortion.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 143: medical management of first-trimester abortion. *Obstet Gynecol.* 2014;123(3):676-692[50]; World Health Organization. *Clinical Practice Handbook for Safe Abortion.* Geneva, Switzerland: WHO Press; 2014.[51]

Post-Termination Care

Regardless of whether a midwife is involved in the termination procedure or even the counseling of the woman, it is common for a midwife to care for women who have recently had a pregnancy termination. Women may have an IUD inserted immediately after an uncomplicated first-trimester abortion.[52] Other contraceptive methods are classified into category 1 or 2 by the U.S. MEC.[24] Women who experience sepsis should avoid insertion of an IUD until the infection has revolved. Women who have undergone either a surgical or medical abortion should be seen for a follow-up visit within 2 weeks. The follow-up visit assessment includes assessment for complications including incomplete abortion, evaluation of emotional status, and discussion of ongoing contraceptive needs.

A pelvic ultrasound, urine pregnancy test, or qualitative serum beta–human chorionic gonadotropin (β-hCG) measurement may be used to confirm expulsion of the products of conception. A systematic review of the literature revealed controversy about routine use of ultrasound to rule out retained products of conception. Although ultrasound has high sensitivity and specificity for this indication, it is expensive and not always available. The use of alternative modalities, such as standardized questioning of a woman's symptoms, telephone consultations, and urine hCG measurement, are effective strategies for follow-up. However, if there is a strong suspicion of an abortion failure, ultrasound remains the most common intervention.[53]

Role of Midwives in Performing Abortions

In 1971, ACNM issued a practice statement that prohibited certified nurse midwives (CNMs) from performing abortions based on the rationale that it was an operative procedure best performed by a physician. This statement preceded the U.S. Supreme Court's 1973 ruling in *Roe v. Wade*, which legalized abortion across the United States. The 1971 statement was the only prohibitive statement ever issued by the ACNM and, after discussion within the membership, it was retracted in 1992.[54]

The abortion care provided by midwives and other nonphysician providers has been demonstrated to be equal to that provided by physicians.[55,56] Because medical abortion is discrete, the numbers of midwives who may be providing such intervention is unknown. The

University of California–San Francisco has provided education for CNMs, physician assistants (PAs), and nurse practitioners (NPs) in the procedure of uterine aspiration evacuation for many years. Induced abortion is not found in the ACNM core competencies, but midwives who desire to add it to their scope of practice can follow the steps outlined to incorporate new procedures as found in the *Standards for the Practice of Midwifery*.[57,58]

Emergency Contraception

Misnamed as postcoital or “morning after” contraception, this family planning method is more appropriately termed *emergency contraception*. This form of contraception is not intended for use as a regular method of birth control. Rather, it is recommended either when there has been a method failure, such as condom breakage, or after unintended unprotected intercourse. Emergency contraception may be used anytime in the first 3 to 5 days after unprotected intercourse depending on the method chosen.[59,60] Women seeking contraceptive services should be counseled about the availability and correct use of emergency contraception.

Four methods of emergency contraception currently are available in the United States: (1) the Yuzpe method; (2) levonorgestrel (LNG) formulations (Plan B One Step, Next Choice One Dose, Take Action); (3) selective progesterone receptor modulators (ulipristal acetate [UPA; ella]); and (4) the copper intrauterine device (IUD). The dose, efficacy, timing, availability, U.S. MEC rating, and clinical conditions for these methods are summarized in **Table 15-8**.[24,25,59,61,62]

Table 15-8 **Emergency Contraception in the United States**

Brand Name	Estimated Efficacy[a]	Timing after Intercourse[b]	Availability	U.S. MEC[c]	Clinical Considerations
FDA-Approved Emergency Contraception					
Plan B One Step Next Choice One Dose Take Action My Way And a number of other generics	81–90% reduced pregnancy rate	Within 72 hours	No prescription No age restriction	1 or 2	May be less effective for women with BMI ≥ 30. Avoid use of CYP3A4 inducers at same time.
ella (Ulipristal acetate or UPA) (1 pill)	2.1% failure rate	Within 120 hours	Prescription only	1 or 2	May reduce the contraceptive action of progestin-containing hormonal contraceptive methods because of its affinity for binding to the progesterone receptor; SPR recommends hormonal contraception delay for 5 days with abstinence for 7 days post UPA, although depot medroxyprogesterone acetate can be started simultaneously with UPA. Avoid use of CYP3A4 inducers at same time. Discard breast milk for 24 hours after administration due to higher concentrations in milk.
Not FDA-Approved Emergency Contraception But Available					
Combined oral contraceptives	1.3–1.5% failure rate	Within 72 hours	Prescription only	1–2	Also known as the Yuzpe method. More than 25 brands can be used.[d]
ParaGard copper IUD	99% reduced pregnancy rate	Within 120 hours after ovulation	Prescription only	1–3	U.S. MEC level varies based on risk of sexually transmitted diseases.

Abbreviations: BMI, body mass index; FDA, U.S. Food and Drug Administration; IUD, intrauterine device; SPR, *U.S. Selected Practice Recommendations for Contraceptive Use, 2016*; U.S. MEC, *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016.*

[a] Studies report different metrics regarding efficacy.
[b] Per package insert/manufacturer's guide. Some methods have been studied only up the time noted but may be effective longer.
[c] For use as emergency contraception. For use as an IUD, other rare conditions, such as a distorted uterine cavity, may also preclude use. See https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf.
[d] Currently available combined oral contraceptives and doses can be found at http://ec.princeton.edu/questions/dose.html.

Based on Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep.* 2016;65(3):1-103. Available at: https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf. Accessed August 27, 2017[24]; Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2016. *MMWR Recomm Rep.* 2016;65(4):1-66. Available at: https://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm. Accessed August 27, 2017[25]; Murphy P. Update on emergency contraception. *J Midwifery Womens Health.* 2012;57(6):593-602[59]; Kaiser Family Foundation. Major methods of emergency contraception availability and policy in the U.S. Updated 2017. Available at https://kaiserfamilyfoundation.files.wordpress.com/2010/08/major-methods-of-emergency-contraception-availability-and-policy-in-the-u-s2.png. Accessed August 26, 2017[61]; Office of Population Research and Association of Reproductive Health Professionals. The emergency contraception website. Available at: http://ec.princeton.edu/questions/dose.html. Accessed August 26, 2017.[62]

Hormonal Emergency Contraception: Mechanisms of Action

The mechanism of action of hormonal emergency contraception methods is inhibition and/or delay of ovulation secondary to suppression, delaying, or blunting of the luteinizing hormone (LH) surge, depending on when in the menstrual cycle the contraceptive pill is ingested.[50] Oral contraceptive and LNG formulations of pregnancy prevention do not effectively prevent follicular rupture if used in the late preovulatory stage; thus, these two methods are most

effective if used prior to ovulation and as soon as possible after unprotected intercourse.[63] UPA does prevent follicular rupture if taken before the onset of the LH surge and reduces endometrial thickness, thereby making the endometrium less hospitable for implantation of an embryo. Hormonal emergency contraception methods do not interfere with a conceptus that has already implanted and present no risk to that embryo.[45,63-68]

Calculating effectiveness rates for emergency contraception is complex because accurately estimating when ovulation occurs relative to the timing of sexual intercourse is difficult, and because some of the research reports use different statistics to report estimated efficacy. In general, emergency contraception is most effective when the interval between intercourse and use is short. Longer intervals result in decreased effectiveness.[65]

The Yuzpe Method

The Yuzpe method, an eponymous regimen that was first published by a Canadian scientist in the 1970s, is the oldest of the current emergency contraception methods. This method involves the ingestion of a high dose of estrogen and progestin, which is accomplished by administering several combined oral contraceptive pills in a single dose so as to inhibit ovulation. More than 25 brands of COCs can be used with this method. Because the brands of COCs can vary, when the Yuzpe method is desired, a midwife can consult a website with current information, such as the emergency contraception website supported by the Office of Population Research at Princeton University and the Association of Reproductive Health Professionals.[62]

The pregnancy rate following use of the Yuzpe method is approximately 2% to 3%. This contraceptive method frequently causes nausea, vomiting, headache, vertigo, and breast tenderness. The woman may also experience irregular bleeding or spotting in the 3 to 4 weeks following treatment.[65] The Yuzpe method is not suggested as the first choice of emergency contraception particularly because of the associated nausea and vomiting.[69] If it is used, an over-the-counter antiemetic, such as meclizine (Antivert, Bonine, Dramamine II) in a dose of 25–50 mg, can be recommended for use 1 hour prior to taking the emergency contraception dose. There is conflicting information on whether either dose should be repeated if the woman vomits within 1 hour of ingesting the medication.

The Yuzpe method may be a good choice if a woman has difficulty obtaining other dedicated emergency contraception methods because of age or lack of availability in the area. It also may present an attractive option if the woman has COCs on hand.

Levonorgestrel: Plan B One Step and Next Choice One Dose

Although still available, the Yuzpe method has been largely replaced by the progestin-only formulations made of LNG. The LNG formulations are more effective and have fewer side effects than those that contain both estrogen and progrestins.[65,70] LNG primarily inhibits or delays ovulation by preventing the LH surge if taken before the LH surge occurs.[66] If taken close to or during the LH surge, it blunts and delays the surge and makes the ovum resistant to fertilization.[59]

LNG for emergency contraception is formulated under the brand name Plan B One Step as

well as a variety of other brand and generic names. When using a one dose product, according to the package insert, a single dose of 1.5 mg LNG is taken within 72 hours following unprotected intercourse.[61,62] If vomiting occurs within 2 hours of taking a single-dose LNG, a repeat dose should be considered. Previously a two dose LNG option was available, however the convenience of taking one dose rather than two tended to improve use and effectiveness so that the two dose products are no longer on the market.

Studies that have evaluated one dose LNG agents used for emergency contraception and ingested within 72 hours following intercourse, have found the effectiveness is approximately 84% for the single-dose regimen.[71] Pregnancy rates are approximately 2% and generally lower than the pregnancy rates following use of the Yuzpe method.[59]

Ulipristal Acetate

In 2010, UPA, a selective progesterone receptor modulator marketed under the brand name "ella," received FDA approval for use as emergency contraception; thus, UPA is the first—and to date only—selective progesterone receptor modulator agent available for emergency contraception in the United States.[59] Selective progesterone receptor modulators bind to progesterone receptors and inhibit or delay ovulation, depending on when the agent is taken during the menstrual cycle. If used during the midfollicular phase, UPA prevents follicular rupture.[59] When this agent is taken in the late follicular phase, it delays the normal LH surge, thereby delaying ovulation.[67,72,73]

UPA appears to be more effective as emergency contraception than either the Yuzpe method or LNG, and can be effective up to 120 hours after unprotected intercourse—an effect attributed to its ability to prevent follicular rupture, although its effectiveness does decrease with time.[69] Side effects are the same as those reported for the Yuzpe method and LNG, but occur less frequently. Because most of these side effects are also symptoms of pregnancy, it is not clear exactly which ones occur secondary to administration of the drug.

Contraindications to Hormonal Emergency Contraception

The major contraindication to the use of any method of emergency contraception is a known pregnancy. Oral formulations of emergency contraception are contraindicated in pregnancy because they are ineffective; the contraindication is not related to concerns about teratogenicity. Because these hormones are given in low doses for a short period of time, women with healthcare conditions for which oral contraceptives are contraindicated can use emergency contraception safely.[59]

Although adverse drug–drug interactions have not been reported by women using oral emergency contraception, adverse drug interactions are possible. Anticonvulsants such as phenobarbital (Luminal), carbamazepine (Tegretol), and phenytoin (Dilantin) induce the CYP450 system and could decrease effectiveness of the contraceptive. Other drugs that interfere with the metabolism of oral contraceptive agents include rifampin (Rifadin), topiramate (Topamax), and St. John's wort.[59]

Copper Intrauterine Device

The copper IUD (ParaGard) can be used as emergency contraception when inserted within 120 hours (5 days) following intercourse and is the most effective emergency contraception method currently available.[59] The IUD is not effective for emergency contraception if inserted after the fertilized egg has implanted in the uterus, which normally occurs 6 to 12 days after ovulation.[59] The technique for insertion is described in the *Procedure for IUD Insertion* appendix to the *Nonhormonal Contraception* chapter.

Pregnancy rates following insertion of a copper IUD are approximately 0.1%.[74] The copper IUD alters tubal transport, is toxic to ovum, and incapacitates sperm, thereby preventing fertilization.[59] Unlike oral formulations of emergency contraception, the copper IUD also creates an inhospitable uterine environment for implantation.[5] If a woman is a candidate for intrauterine contraception, placement of a copper IUD for emergency contraception offers the additional advantage of establishing an ongoing contraceptive method. Note that the levonorgestrel-releasing intrauterine system IUD is not indicated for emergency contraception.

Contraindications to Use of the Copper IUD for Emergency Contraception

General contraindications to use of a copper IUD are reviewed in detail in the *Nonhormonal Contraception* chapter. Insertion of a copper IUD for emergency contraception after rape is an important topic to review. The U.S. MEC for Contraceptive Use category for insertion of a copper IUD for emergency contraception is 3 if the woman has a high risk for being exposed to an STI, but is 1 if she has a low risk for being exposed to an STI.[25] There are no agreed-upon definitions for what constitutes “high risk” versus “low risk” for STI. In addition, the U.S. MEC for Contraceptive Use category 3 means that the theoretical risks generally outweigh the advantages. Thus, clinician judgment is needed to assess the history and circumstances for a woman who has been raped and to determine whether she is a good candidate for a copper IUD.

Special Considerations for Dispensing Emergency Contraception

Some pharmacies may opt out of dispensing emergency contraception methods and a number of states protect their right to do so, as well as the rights of midwives and other professionals who do not choose to provide contraception.[75,76] In such cases, the Yuzpe regimen may be recommended. Many healthcare professionals regularly prescribe emergency contraception for women using a SARC method in case of missing COCs, breakage of a condom, or other circumstances.

Many national women’s health organizations recommend that all women who are victims of sexual assault be offered emergency contraception.[77] Some states mandate that women must be offered emergency contraception after rape. State laws can change, however, so the clinician should determine the specific state rules that apply. In any case, the midwife who may have occasion to care for a woman post assault should discuss whether she has either received appropriate information or been treated with emergency contraception.

Follow-up Care

Women who need emergency contraception also should be offered screening for STIs including human immunodeficiency virus (HIV) infection.

Women using emergency contraception may be counseled to return for evaluation 1 to 3 weeks following use of this treatment, specifically to assess the result of emergency contraception and discuss ongoing contraceptive needs, although this follow-up visit is not considered a necessary component of emergency contraception care by all professional organizations. In most situations, a suitable method of contraception should be started as soon as possible after the woman has used an emergency contraception method. Women who use hormonal emergency contraception can initiate any contraceptive method immediately, but are advised to use a barrier method or abstinence for the next 7 days. Because ulipristal acetate has a unique mechanism of action, using a hormonal contraceptive simultaneously can decrease the effectiveness of both methods. Therefore, women using UPA should use a barrier method or abstinence until the next menses, although they can begin a hormonal contraceptive method 5 days after ingestion of UPA.[25,26] Women using a copper IUD have immediate and continuing contraceptive protection.

Health Education

Women using emergency contraception should be advised that the next menses may be delayed after using oral regimens. It is important to be evaluated for pregnancy if menses does not occur within 3 weeks after emergency contraception use. Women should also be counseled regarding signs of ectopic pregnancy and advised to contact a healthcare provider as soon as possible if they develop severe abdominal pain at any point after using the emergency contraception method. In addition, women using emergency contraception should avoid unprotected intercourse until a method of contraception has been initiated.

Emerging Trends and Future Contraceptive Methods

There is no “one size fits all” method of contraception. Therefore, the greater the variety of options, the more likely that women will be able to find a method that they can consistently and correctly use in concert with their lifestyles. Prediction of new methods of contraceptives is an uncertain act, especially since development of a new method often take years and during trials complications may be identified such that the method is never introduced to the U.S. market.

Some of the proposed methods could potentially resolve common problems with contraceptives. For example, among the new methods needed are additional vaginal spermicides with microbicidal properties, agents with new delivery mechanisms, lower-cost agents, and those with dual protection for STIs.[78] Injectables and implants that offer a longer duration of effects could improve the effectiveness of current LARCs. A biodegradable implant could be more acceptable to women if it avoids the need for removal.

Among the new methods currently being investigated for women is a monthly injectable composed of medroxyprogesterone acetate and estradiol. A vaginal ring that is effective for 1 year, and that contains ethinyl estradiol and nestorone, may be available someday. A new weekly patch containing ethinyl estradiol and levonorgestrel is being researched. Several COCs are under investigation, many of which represent variations of a current formulation and others based on newer progestins.

A myriad number of fertility apps already exist, and in the future, they may potentially provide the ultimate control for women over their reproduction, enabling them to abstain during days of risk or use a barrier method then.

Although these innovations are focused on women, development of male contraception methods other than male condoms is considered desirable, although few innovations have emerged on this front. Perhaps the most promising male development involves an injection of a gel that creates a type of a chemical mesh to trap and inactivate sperm in the vas deferens without harming the surrounding tissues. The procedure, which is known as reversible inhibition of sperm under guidance (RISUG), is not permanent, as another agent can be injected to reverse the process.[79]

Prediction of future developments in health care is always difficult, and forecasting is no different in the area of family planning. However, development of an effective and acceptable method could potentially bring billions of dollars in revenues to the manufacturer; this powerful lure to would-be developers is cause for optimism in the family planning realm.

Conclusion

The midwife’s role in assessing a woman’s needs, preexisting knowledge, and desire in regard to avoiding pregnancy is paramount so that a woman can choose her best options. Such choice is individualized based on a myriad of factors, including age, health, culture, desire, beliefs, relationships, and response should a method fail. Counseling women about the options of parenting, releasing a child to adoption, and pregnancy termination are within the scope of the midwife. New methods of contraception are welcome developments, as they provide more choices for women and can ultimately decrease the rate of unintended pregnancy.

Resources

Organization	Description	Webpage
Association of Reproductive Health Professionals (ARHP) and Office of Population Research at Princeton University	Emergency contraception update including list of current formulations for the Yuzpe method	http://www.arhp.org/uploadDocs/CPECUpdate.pdf http://ec.princeton.edu/questions/eceffect.html
Centers for Disease Control and Prevention (CDC)	U.S. Medical Eligibility Criteria (U.S. MEC) for Contraceptive Use Selected Practice Recommendations (SPR) for Contraceptive Use	http://www.cdc.gov/mmwr/pdf/rr/rr59e0528.pdf https://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm Available as an e-book at https://www.cdc.gov/reproductivehealth/contraception/ebook.html
Guttmacher Institute	Research and policy organization committed to advancing sexual and reproductive health and rights in the United States and globally	http://www.guttmacher.org
World Health Organization (WHO)	Downloadable Wheel (to be printed and assembled)	http://apps.who.int/iris/bitstream/10665/173585/1/9789241549257_eng.pdf?ua=1

References

1. Dittrick Museum. The history of contraception: an online exhibit. Available at: https://dittrickmuseumblog.com/2013/07/18/the-history-of-contraception-an-online-exhibit/. Accessed August 27, 2017.
2. Tracy EE. Contraception: menarche to menopause. *Obstet Gynecol Clin North Am.* 2017;44(2):143-158.
3. American College of Obstetricians and Gynecologists. Gynecology data definitions. Available at: https://www.acog.org/-/media/Departments/Patient-Safety-and-Quality-Improvement/reVITALize-Gynecology-Definitons-V1.pdf?dmc=1&ts=20170814T2340563780. Accessed August 27, 2017.
4. Finer LB, Zolna MR. Declines in unintended pregnancy in the United States, 2008–2011. *N Engl J Med*. 2016;374(9):843-852.
5. Murphy PA, Elmore CE. Contraception. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. *2nd ed*. Burlington, MA: Jones & Bartlett Learning; 2017:864-865.
6. Jones RK, Jerman J. Abortion incidence and service availability in the United States, 2014. *Perspect Sex Reprod Health*. 2017;49(1):17-27.
7. Trussell J. Contraceptive failure in the United States. *Contraception*. 2011;83(5):397-404.
8. Trussell J. Understanding contraceptive failure. *Best Pract Res Clin Obstet Gynaecol*. 2009;23(2):199-209.
9. Pearl R. Factors in human fertility and their statistical evaluation. *Lancet.*1993;222:607-611.
10. Kuo TM. The multistate life table method: an application to contraceptive switching behavior. *Demography*. 2008;45(1):157-171.
11. Daniels K, Daugherty J, Jones J, Mosher W. Current contraceptive use and variation by selected characteristics among women aged 15–44: United States, 2011–2013. *Natl Health Stat Report*. 2015;(86):1-14.
12. Centers for Disease Control and Prevention. Effectiveness of family planning methods. Available at: https://www.cdc.gov/reproductivehealth/unintendedpregnancy/pdf/contraceptive_methods_508.pdf. Accessed September 15, 2017.
13. Kelly PJ, Cheng AL, Witt J. Advanced practice registered nurses and long-acting reversible contraception. *Midwifery Womens Health*. 2017;62(2):190-195.
14. Centers for Disease Control and Prevention. Strategic plan: improving women's reproductive health, pregnancy health, and infant health. Available at: https://www.cdc.gov/reproductivehealth/drh/strategicplan.htm. Accessed August 27, 2017.
15. Committee on Gynecologic Practice, Long-Acting Reversible Contraception Working Group of the American College of Obstetricians and Gynecologists. Committee Opinion No. 642: increasing access to contraceptive implants and intrauterine devices to reduce unintended pregnancy. *Obstet Gynecol*. 2015;126(4):e44-e48.
16. Committee on Adolescent Health Care, Long-Acting Reversible Contraception Working Group of the American College of Obstetricians and Gynecologists. Committee Opinion No. 539: adolescents and long-acting reversible contraception: implants and intrauterine devices. *Obstet Gynecol*. 2012;120(4):983-988.
17. Ott MA, Sucato GS. From the American Academy of Pediatrics technical report: contraception for adolescents. *Pediatrics*. 2014;134(4):e1257-e1281.
18. Diedrich JT, Klein DA, Peiper JF. Long-acting reversible contraception in adolescents: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2017;216(4):364.e1-364.e12.
19. Cleland K, Peipert, JF, Spear S, Trussel J. Family planning as a cost-saving preventive health service. *N Engl J Med*. 2011;364(37):e37.
20. Thaxton L, Espey E. Family planning American style redux: unintended pregnancy improves, barriers remain. *Obstet Gynecol Clin North Am.* 2017;44(1):41-56.
21. Gomez AM, Fuentes L, Allina A. Women or LARC first? Reproductive autonomy and the promotion of long-acting reversible contraceptive methods. *Perspect Sex Reprod Health*. 2014;46(3):171-175.
22. Committee on Adolescent Health Care of the American College of Obstetricians and Gynecologist. Committee Opinion No 699: adolescent pregnancy, contraception, and sexual activity. *Obstet Gynecol. 2*017;129(5):e142-e149.
23. World Health Organization. *Medical eligibility criteria for contraceptive use*. 5th ed. Geneva, Switzerland: WHO; 2015. Available at: http://www.who.int/reproductivehealth/publications/family_planning/MEC-5/en/. Accessed August 27, 2017.
24. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(3):1-103. Available at: https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf. Accessed August 27, 2017.
25. Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. selected practice recommendations for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(4):1-66. Available at: https://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm. Accessed August 27, 2017.
26. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD:

American College of Nurse-Midwives; 2012.
27. Espey E. Feminism and the moral imperative for contraception. *Obstet Gynecol*. 2015;126(2):396-400.
28. Grace KT. Caring for women experiencing reproductive coercion. *J Midwifery Womens Health*. 2016:61(1);112-115.
29. Doherty ME, Scannell-Desch E. Women's health and hygiene experiences during deployment to the Iraq and Afghanistan wars, 2003 through 2010. *J Midwifery Womens Health*. 2012;57(2):172-177.
30. Borrero S, Callegari LS, Zhao X, et al. Unintended pregnancy and contraceptive use among women veterans: the ECUUN study. *J Gen Intern Med*. 2017;32(8):900-908.
31. Grover SR. Gynaecological issues in adolescents with disability. *J Paediatr Child Health*. 2011;47(9):610-613.
32. FIGO Committee for Ethical Aspects of Human Reproduction and Women's Health. Ethical issues in the management of severely disabled women with gynecologic problems. *Int J Gynaecol Obstet*. 2011;115(1):86-87.
33. Baker V, Kelly PJ, Cheng A, Witt J. The role of previous contraception education and moral judgment in contraceptive use. *J Midwifery Womens Health*. 2014;59(4);447-451.
34. Bond S. Safe and effective contraception during perimenopause may also alleviate perimenopause symptoms. *J Midwifery Womens Health*.2015:60(3);334.
35. Singer J. Options counseling: techniques for caring for women with unintended pregnancies. *J Midwifery Womens Health*. 2004;49(3):235-242.
36. Grotevant HD, Lo AY. Adoptive parenting *Curr Opin Psychol*. 2017;15:71-75.
37. Stubblefield PG, Carr-Ellis S, Borgatta L. Methods for induced abortion. *Obstet Gynecol*. 2004;101(1):174-178.
38. Edelman A, Nichols MD, Jensen J. Comparison of pain and time of procedures with two first-trimester abortion techniques performed by residents and faculty. *Am J Obstet Gynecol*. 2001;184(7):1564-1567.
39. Bond S. Women seeking first trimester abortion overwhelmingly recommend support from doulas during procedure. *J Midwifery Womens Health*. 2015. 2015;60(3):334.
40. Tschann M, Salcedo J, Kaneshiro B. Nonpharmaceutical pain control adjuncts during first-trimester aspiration abortion: a review. *J Midwifery Womens Health*. 2016;61(3):331-338.
41. Cleland K, Creinin MD, Nucatola D, Nshom M, Trussell J. Significant adverse events and outcomes after medical abortion. *Obstet Gynecol*. 2013;121(1):166-171.
42. Niinimäki M, Pouta A, Bloigu A, et al. Immediate complications after medical compared with surgical termination of pregnancy. *Obstet Gynecol*. 2009; 114(4):795-804.
43. Oppegaard KS, Sparrow M, Hyland P, et al. What if medical abortion becomes the main or only method of first trimester abortion? A roundtable of views. *Contraception*. August 3, 2017. [Epub ahead of print]. doi:10.1016/j.contraception.2017.04.004.
44. Simmonds KE, Beal MW, Eagen-Torkko MK. Updates to the US Food and Drug Administration regulations for mifepristone: implications for clinical practice and access to abortion. *J Midwifery Womens Health*. 2017:62;348-352.
45. Hatcher R, Trussell J, Nelson A, Cates W Jr, Kowal D, Policar M, eds. *Contraceptive Technology*. 20th ed. New York, NY: Ardent Media; 2011:705.
46. Bartz D, Goldberg A. Medication abortion. *Clin Obstet Gynecol*. 2009;52(2):140-150.
47. Nguyen T, Blum J, Raghavan S, et al. Comparing two early medical abortion regimens: mifepristone + misoprostol vs. misoprostol alone. *Contraception*. 2011;83:410-417.
48. Ngai S, Tang O, Chan Y, Ho P. Vaginal misoprostol alone for medical abortion up to 9 weeks of gestation: efficacy and acceptability. *Hum Reprod*. 2000;15(5):1159-1162.
49. Bos-Thompson MA, Hillaire-Buys D, Roux C, Faillie JL, Amram D. Möbius syndrome in a neonate after mifepristone and misoprostol elective abortion failure. *Ann Pharmacother*. 2008;42(6):888-892.
50. American College of Obstetricians and Gynecologists. Practice Bulletin No. 143: medical management of first-trimester abortion. *Obstet Gynecol*. 2014;123(3):676-692.
51. World Health Organization. *Clinical Practice Handbook for Safe Abortion*. Geneva, Switzerland: WHO Press; 2014.
52. Patil E, Darney B, Orme-Evans K, et al. Aspiration abortion with immediate intrauterine device insertion: comparing outcomes of advanced practice clinicians and physicians. *J Midwifery Womens Health*. 2016:61(3);325-330.
53. Grossman D, Grindlay K. Alternatives to ultrasound for follow-up after medication abortion: a systematic review. *Contraception*. 2011;83(6):504-510.
54. Summers L. The genesis of the ACNM 1971 statement of abortion. *J Nurs Midwifery*. 1992;37(3):168-174.
55. Levi A, James E, Taylor D. Midwives and abortion care: a model for achieving competency. *J Midwifery Womens Health*. 2012;57:285-289.
56. Renner RM, Brahmi D, Kapp N. Who can provide effective and safe termination of pregnancy care? A systematic review. *BJOG*. 2013;120(1):23-31.
57. American College of Nurse-Midwives. Standards for the practice of midwifery. 2011. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000051/Standards_for_Practice_ Accessed August 27, 2017.
58. Phillippi JC, Avery MD. The 2012 American College of Nurse-Midwives core competencies for basic midwifery

practice: history and revision. *J Midwifery Womens Health*. 2014;59(1):82-90.
59. Murphy P. Update on emergency contraception. *J Midwifery Womens Health*. 2012;57(6):593-602.
60. Lee JK, Schwarz EB. The safety of available and emerging options for emergency contraception. *Expert Opin Drug Saf*. July 21, 2017. [Epub ahead of print]. doi:10.1080/14740338.2017.1354985.
61. Kaiser Family Foundation. Major methods of emergency contraception availability and policy in the U.S. Updated 2017. Available at: https://kaiserfamilyfoundation.files.wordpress.com/2010/08/major-methods-of-emergency-contraception-availability-and-policy-in-the-u-s2.png. Accessed August 26, 2017.
62. Office of Population Research and Association of Reproductive Health Professionals. The emergency contraception website. Available at: http://ec.princeton.edu/questions/dose.html. Accessed August 26, 2017.
63. Gemzell-Danielsson K, Berger C, Lalitkumar PGL. Emergency contraception: mechanisms of action. *Contraception*. 2013;87(3):300-308.
64. Trussell J, Ellertson C, von Hertzen H, et al. Estimating the effectiveness of emergency contraceptive pills. *Contraception*. 2003;67(4):259-265.
65. Trussell J, Ellertson C, Dorflinger L. Effectiveness of the Yuzpe regimen of emergency contraception by cycle day of intercourse: implications for mechanism of action. *Contraception*. 2003;67(3):167-171.
66. Novikova N, Wesiberh E, Stanczyk F, Croxatto H, Fraser I. Effectiveness of levonorgestrel emergency contraception given before or after ovulation: a pilot study. *Contraception*. 2007;75(112):2007.
67. Brache V, Cochon L, Jesam C, et al. Immediate pre-ovulatory administration of 30 mg ulipristal acetate significantly delays follicular rupture. *Hum Reprod*. 2010;25(9):2256-2263.
68. Marions L, Hultenby K, Lindell I, Sun X, Ståbi B, Gemzell Danielsson K. Emergency contraception with mifepristone and levonorgestrel: mechanism of action. *Obstet Gynecol*. 2002;100(65):2002.
69. Shen J, Che Y, Showell E, Chen K, Cheng L. Interventions for emergency contraception. *Cochrane Database Syst Rev*. 2017;8:CD001324. doi:10.1002/14651858.CD001324.pub5.
70. American College of Obstetricians and Gynecologists. Practice Bulletin No. 152: emergency contraception. *Obstet Gynecol*. 2015;126(3):e1-e11.
71. Sambol N, Harper C, Kim L, Liu CY, Darney P, Raine TR. Pharmacokinetics of single-dose levonorgestrel in adolescents. *Contraception*. 2006;74(104):104-109.
72. Glasier A, Cameron S, Fine P, et al. Ulipristal acetate versus levonorgestrel for emergency contraception: a randomised non-inferiority trial and meta-analysis. *Lancet*. 2010;375(9714):555-562.
73. Fine P, Mathe H, Ginde S, Cullins V, Morfesis J, Gainer E. Ulipristal acetate taken 48–120 hours after intercourse for emergency contraception. *Obstet Gynecol*. 2010;115(2 pt 1):257-263.
74. Cleland K, Zhu H, Goldstuck N, Cheng L, Trussell J. The efficacy of intrauterine devices for emergency contraception: a systematic review of 35 years of experience. *Hum Reprod*. 2012;27(7):1994-2000.
75. Wernow JR, Grant DG. Dispensing with conscience: a legal and ethical assessment. *Med Law Ethics*. 2008;42(11):1669-1678.
76. Guttmacher Institute. Refusing to provide health services. August 2017. Available at: https://www.guttmacher.org/state-policy/explore/refusing-provide-health-services. Accessed August 27, 2017.
77. Committee on Health Care for Underserved Women of the American College of Obstetricians and Gynecologists. Committee Opinion No. 542: access to emergency contraception. *Obstet Gynecol*. 2012;120(5):1250-1253.
78. Jensen J. The future of contraception: innovations in contraceptive agents: tomorrow's hormonal contraceptive agents and their clinical implications. *Am J Obstet Gynecol*. 2011;205(4):S21-S25.
79. Kanakis GA, Goulis DG. Male contraception: a clinically-oriented review. *Hormones*. 2015;14(4):598-614.

CHAPTER

16

Nonhormonal Contraception

MARY C. BRUCKER

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Introduction

Today, women who want to use a form of contraception have an array of both hormonal and nonhormonal methods available. Many choose a nonhormonal option, both in the United States and worldwide. Some choose a nonhormonal method because they have concerns about the use of exogenous hormones or want to avoid exposure to additional hormones. Others have preexisting health conditions, such as thromboembolic disorders that preclude the use of some of the hormonal types of contraception. Still other women use nonhormonal methods as secondary contraceptive methods while initiating or transferring between hormonal methods or as one of multiple contraceptive methods used simultaneously.

This chapter reviews the contraceptive methods that do not contain hormones for their mechanism of action. Nonhormonal reversible methods can be categorized as behavioral, spermicidal, barrier methods or copper intrauterine devices (IUDs). In addition, male and female sterilization are permanent nonhormonal methods of contraception.

Intrauterine contraceptive devices include a copper device that is similar to the IUDs containing levonorgestrel (LNG). Both copper and LNG-containing IUDs are highly effective long-acting reversible contraceptives (LARC) with multiple commonalities. General information that applies to both types of IUDs is found in this chapter. Because the copper type of IUD does not contain any hormone, it is discussed in depth in this chapter, whereas those IUDs containing levonorgestrel are reviewed in the *Hormonal Contraception* chapter.[1]

The contraceptives that are within the behavioral, spermicidal, and barrier categories are controlled by the user, are relatively inexpensive, and have few or minor side effects. Many nonhormonal methods do not require a prescription (e.g., foam, condoms), and most are used only when coitus is anticipated or during coitus. Behavioral methods use no device at all, relying instead on biologic processes such as breastfeeding or observing changes during the menstrual cycle for contraceptive effectiveness.

Most of the nonhormonal contraceptive methods are safe, and with a few exceptions, these forms of contraception have no absolute contraindications. Thus, they are listed as Category 4 in the Centers for Disease Control and Prevention (CDC) guidelines entitled U.S. Medical Eligibility Criteria (U.S. MEC) for Contraceptive Use.[2] Health risks associated with these products are detailed in the section specific to each method in this chapter.

The Effectiveness of Nonhormonal Methods in Preventing Pregnancy

Each individual has personal reasons to choose one contraceptive method instead of another. For the majority, effectiveness is a major consideration in the choice of a contraceptive method, albeit not necessarily the *most* important. For example, some women may consider effectiveness to be less important than other factors and view a pregnancy that ensues a bit earlier than planned not to be a major problem. For other women, effectiveness is a primary consideration. The best contraceptive method is the one the user desires and will use consistently and correctly.

If a woman or a couple uses no contraceptive method, approximately 85 of every 100 will have an unintended pregnancy in the first year of no contraceptive use.[1,3] Effectiveness rates for typical use of the nonhormonal reversible methods vary widely, ranging from fewer than 1 unintended pregnancy per 100 women who use the copper IUD to 28 unintended pregnancies per 100 women who use spermicide only. The lactational amenorrhea method is highly effective, but the duration of its effectiveness is only approximately 6 months; this method also depends on certain specific requirements that can be socially difficult to fulfill for some women in the United States. Fertility awareness methods have quite variable rates of effectiveness and are highly dependent upon health education and clear understanding and commitment by users. Ultimately, the decision about type of contraception (if any) belongs to the woman, and providers should be aware of their own beliefs to avoid injecting personal bias when counseling women about contraceptive methods.[4]

Behavioral Methods

Behavioral contraceptive methods focus on identifying the likely fertile time for an individual woman.[5] These methods are highly dependent upon the woman/couple's motivation, understanding of fertility, and presence of normal predictable menstrual cycles. Behavioral contraceptive methods may be particularly attractive to individuals who do not want artificial interventions. Behavioral methods can be used for pregnancy prevention, attainment of pregnancy, or pregnancy spacing. Globally, behavioral methods are among the oldest and popular methods, with more than 5% of couples who are contracepting worldwide using coitus interruptus or fertility awareness.[6]

Abstinence

When undertaken for the purpose of avoiding pregnancy, abstinence is defined as refraining from penile–vaginal intercourse. However, if abstinence is defined to include prevention of sexually transmitted infections (STIs), then abstinence includes not engaging in sexual contact that has a risk of STI transmission. For example, anal intercourse with an infected partner confers a higher risk of transmission of human immunodeficiency virus (HIV) than vaginal penetration. By comparison, oral intercourse confers a higher risk of transmission of human papillomavirus (HPV) and herpes simplex virus (HSV).

Abstinence is free of cost, available without a healthcare provider visit, and free of side effects. Complete abstinence is 100% effective and prevents exposure to STIs as well as pregnancy. Planned abstinence can be unrealistic for some couples, however, and may not be possible to achieve in coercive situations.

Coitus Interruptus/Withdrawal Method

With the coitus interruptus (withdrawal) method, the penis is completely removed from the vagina and away from the female external genitalia before ejaculation. In a survey of contraceptive usage among women from 2006 to 2008, approximately 5% of women reported current use of this method, but more than half reported they had used this method at a prior time.[6,7] In addition, some couples use this method interchangeably or in combination with other methods, such as fertility awareness or condoms.[7] Coitus interruptus is free, requires no contact with healthcare services, and has no side effects. With typical use, 12 of 100 women will experience an unintended pregnancy within the first year of use.[3]

Using coitus interruptus relies to a great extent on both the male partner's awareness of the sensation of imminent ejaculation and his ability to withdraw consistently and completely from the vagina in time to avoid semen coming in contact with the vagina during each act of intercourse. Of note, pre-ejaculate fluid may contain sperm, and could possibly result in pregnancy.[8] Interrupting the sexual response cycle also may diminish pleasure for either or both partners. Women also may not be comfortable with the idea of relinquishing control of contraception to their male partners.

Coitus interruptus, including correct use and estimated effectiveness, should be included in a

discussion of contraceptive methods, especially as rates of its use have been reported to be as high as 25% to 60% among adolescents in the United States.[9] However, coitus interruptus does not protect against STIs, nor does it have any noncontraceptive benefits.

Lactational Amenorrhea Method

The lactational amenorrhea method (LAM) relies on physiologic changes associated with breastfeeding for contraception. Breastfeeding confers a natural method of contraception in the initial postpartum period because the high levels of prolactin that occur during breastfeeding inhibit secretion of gonadotropin-releasing hormone from the hypothalamus, thereby preventing ovulation. Pumping or manual expression of breast milk may reduce the effectiveness of LAM.[10,11] To be successful as a means of contraception, LAM requires that three essential conditions be present, as outlined in **Table 16-1**.[10-12]

Table 16-1 **Criteria for the Lactational Amenorrhea Method**

1. The nursing infant is age 6 months or less.
2. The nursing infant receives all nutrition from suckling, with no more than 5% from food or formula supplementation, and is nursing on demand, with no more than 4 hours between feeds in the daytime and no more than 6 hours between feeds in the night.
3. Maternal menses has not resumed since the infant's birth.

Based on King J. Contraception and lactation. *J Midwifery Womens Health.* 2007;52(6):614-620[10]; Labbok MH. Postpartum sexuality and the lactational amenorrhea method for contraception. *Clin Obstet Gynecol*. 2015;58(4):915-927[11]; Kramer MS, Kakuma R. Optimal duration of exclusive breastfeeding. *Cochrane Database Syst Rev.* 2012;8:CD003517. doi:10.1002/14651858.CD003517.pub2.[12]

Globally, LAM is widely used as a birth spacing method. The major advantages of LAM include that it is immediately available, requires no healthcare visit, and is free, with effectiveness rates as high as 98% to 99.5% during the first 6 months postpartum when the three essential conditions are operational.[1,6] Unfortunately, this method has a limited lifespan because of the short period for which it is effective. In the United States, it also can be difficult to meet the three essential conditions, especially for women who return to work and are separated from their child for several hours each day.

Fertility Awareness Methods

Fertility awareness methods of contraception are occasionally referred to as "natural family planning."[13] These approaches are based on identifying the fertile days in the menstrual cycle when an ovum can be fertilized. However, the term "natural family planning" generally is reserved for a fertility awareness method in which couples use only abstinence during fertile periods; thus, this method is morally acceptable to the Catholic Church.

Fertility awareness methods have been characterized as an inferior method or "old" method

when compared to “modern” methods, perhaps causing individuals and providers to discount using them.[14] The most common concern about fertility awareness methods is poor effectiveness for pregnancy prevention. Reliable data for many of the fertility awareness methods are generally lacking because of the rarity of randomized controlled trials comparing these methods to each other or to other methods. The studies that have been published have been plagued by multiple protocol violations and high dropout rates.[15] For example, several reports have estimated that 24 in 100 women who use these contraceptive methods will have an unintended pregnancy in the first year of use, although the rate of perfect use has been estimated to be 5% or less.[16]

Fertility awareness methods are low-cost, user-controlled contraceptive methods that are available at any time. A multicountry survey of more than 2000 couples using fertility awareness methods reported that the majority were not only satisfied with the method, but also believed that the methods improved their sex lives.[17] Like many contraceptive methods, fertility awareness methods do not protect against transmission of STIs. **Appendix 16A** provides an overview of the various fertility awareness method options.

All of the fertility awareness–based methods are highly dependent on the couple’s motivation and desires; consequently, it is difficult to recommend one method over the others. Emerging technology may be used in combination with these methods, thereby potentially increasing their effectiveness. More than 100 apps are now available, but not all are equally accurate. In 2016, an analysis of the most popular apps listed those considered most appropriate, although the authors also cautioned that use of only an app without education about the contraceptive method may be insufficient to prevent pregnancy.[18]

Spermicidal Agents

Spermicides are chemical agents that kill sperm. Spermicidal agents are formulated as gels, creams, aerosol foam, suppositories, vaginal film, and sponges. The active agent in all spermicidal preparations available in the United States is nonoxynol-9, a surfactant that destroys the sperm cell membrane. This agent is combined with an inert base that creates the specific formulation, such as a cream or gel. The inert base material serves as a mechanical barrier to the cervical os and facilitates vaginal distribution and formation of a surface film that withstands coital activity. Effectiveness of any contraceptive method using the spermicide is directly related to the nonoxynol-9 dose: Products with at least 100 mg of nonoxynol-9 are more effective than preparations with lower doses.[19]

Table 16-2 provides detailed information on spermicidal products. Spermicidal preparations are relatively easy to use, are woman controlled, and are immediately effective. These agents are available widely in stores and online without prescription. **Figure 16-1** depicts the Today sponge, which is impregnated with nonxynol-9.

Table 16-2 Spermicidal Preparations

Preparation	Nonoxynol-9 (mg/dose)	Timing of Application[a]	Duration of Action	Comments
Creams	52–100	Up to 1 hour before coitus	1 hour[b]	Use alone or with a diaphragm
Film	280	Insert 15 minutes before coitus	3 hours[b]	Becomes a gel as it dissolves under body temperature Use dry fingers to place the film deep into the vagina
Foam	100–125	Insert immediately before coitus	1 hour[b]	Aerosol, under pressure in a container; apply with an applicator that is inserted into the vagina in a similar manner to a tampon applicator
Gels	3–4	Insert immediately before coitus	1 hour[b]	Use alone or with a diaphragm
Suppository or insert	100–125	Insert 15 minutes before coitus	1 hour[b]	Activated when melted; spermicide is embedded in cocoa butter or glycerin Remove the covering and insert the suppository deep into the vagina
Sponge	1000	Any time up to 24 hours before coitus	Leave in place for 6 hours after coitus, but no longer than 30 hours total to avoid the rare risk of toxic shock syndrome	Polyurethane "pillow"; moistening with water until suds appear prior to use activates nonoxynol-9 Fold the sponge by using the dimple; place it deeply within the vagina, with the string loop on the bottom for easy removal

[a] Insert another application of nonoxynol 9 for additional acts of intercourse.

[b] Do not douche for this method for a minimum of 6 hours post coitus.



Figure 16-1 Today sponge.

Courtesy of Tekoa L. King, CNM, MPH.

For this contraceptive method to be effective, the woman or couple must use the spermicide consistently with each act of vaginal intercourse. Also, for some preparations, as noted in **Table 16-2**, timing of initiating use is critical for effectiveness. For example, the contraceptive film and suppositories require 15 minutes in the vagina to become effective prior to being exposed to sperm. Women who are not comfortable inserting a finger into the vagina may find film and suppositories problematic, as both need to be placed deep into the vagina so as to cover the cervix. Some postcoital leakage is common with all of the spermicidal preparations except film, which some women consider esthetically unpleasing. Film melts at body temperature and does not result in any additional vaginal discharge.

Spermicidal preparations have a low effectiveness rate for typical use. Among typical users during the first year of use, approximately 28% will have an unintended pregnancy—a rate similar to that for the withdrawal method.[3] The one exception is the sponge, for which effectiveness rates differ according to women's parity: The rates of unintended pregnancy in the first year of use are 12% and 24% for nulliparous and multiparous women, respectively.[3] Even with this limited effectiveness, using any form of this contraceptive method is more effective than using chance or no method.

Side effects and adverse effects of nonoxynol-9 include a contact dermatitis local irritation of the vulva, vagina, or penis, which is often mischaracterized as an allergic reaction. Nonoxynol-9 is a surfactant with the potential to damage the vaginal epithelium, increasing risks of infections, including STIs. Spermicides are absolutely contraindicated (Category 4) for women at high risk for exposure to HIV per the U.S. MEC for Contraceptive Use.[2] The risk of microabrasions to vaginal epithelium increases with use more than twice a day, which in

turn increases the risk of HIV infection.[20] In addition, women who are HIV positive or who have acquired immunodeficiency syndrome (AIDS) likely should not use spermicides because the microabrasions caused by the spermicide also increase viral shedding, thereby increasing the risk of HIV transmission to an uninfected partner.[21]

A rare but serious side effect of sponge use is toxic shock syndrome—that is, an immunologic-mediated, potentially fatal, septic reaction to bacterial toxins of the species *Staphylococcus aureus* and/or *Streptococcus pyogenes*.[22] This condition was discovered in the 1980s to be associated with use of the contraceptive sponge as well as super-absorbent tampons that were composed of carboxymethylcellulose and polyester instead of cotton. Such factors as recent childbirth, use for more than 24 hours, difficult removal, or fragmentation of the aforementioned items appear to increase the risk of toxic shock syndrome.[23] However, the absolute risk of toxic shock syndrome is extremely rare.[24] This infection usually presents as 2 to 3 days of mild symptoms, such as low-grade fever, muscle aches, chills, and malaise. The symptoms worsen rapidly, and include fever higher than 38°C (101.4°F), with diffuse macular erythema and hypotension. Women using diaphragms or the sponge should be educated about the symptoms of toxic shock syndrome, and can decrease their risk of this rare disorder by removing the device within 24 hours of its insertion. Super-absorbent tampons are no longer available on the market.

Barrier Methods

Diaphragms, cervical caps, and condoms are among the oldest contraceptive methods. These methods usually are described as barrier methods because of their mechanism of action. To achieve the maximum effectiveness, spermicides must be used with these barriers so that sperm can be killed before conception occurs. The “barrier” refers to the combination of the physical product that traps ejaculate in the vagina and the chemical barrier supplied by the spermicide.[25] Effectiveness of barrier methods ranges from approximately 88% for diaphragms to an estimated 80% for condoms for women or men.[3]

Diaphragm

The diaphragm is a dome-shaped silicone cup with a flexible rim that permits compression of the device to facilitate insertion into the vagina, but regains its shape and fit so that it fits snugly against the vaginal walls. When properly positioned, the diaphragm rim rests against the anterior (posterior aspect of the symphysis pubis) and lateral vaginal walls, and extends posteriorly to completely cover the cervix. A diaphragm is used with a spermicidal gel or cream that is spread inside the dome of the diaphragm and lightly around the rim which is how the diaphragm provides both physical and chemicals barrier to sperm.[25]

Diaphragms come in three types, based on the presence or absence of a spring in the rim. Each type is available in several sizes, although all require a prescription. Advantages of diaphragm use include that this method is controlled by the woman herself. This method is not necessarily coital dependent, and insertion and removal can be done privately. Previously some diaphragms were composed of latex, but all of today’s diaphragms are silicon based.

Approximately 12% of typical diaphragm users will have an unintended pregnancy in the first year of use, making the diaphragm more effective than male condoms and all other female barrier methods.[3,26] Effectiveness appears to be inversely related to frequency of intercourse. Studies have shown that among individuals who are consistent diaphragm users, those who have intercourse at least three times weekly are almost three times as likely to have an unintended pregnancy.[26] Male condoms can be used simultaneously with the woman using a diaphragm, enhancing contraceptive effectiveness and adding protection against STI transmission.

The size of the diaphragm must be determined individually for each woman, because the distance from the posterior fornix to the posterior aspect of the symphysis pubis varies among women. Sizing is not specifically associated with parity. **Appendix 16B** describes the procedure for determining the right size of diaphragm for a woman.

Little evidence regarding timing of insertion and removal of diaphragms is available, but the usual instructions state that the diaphragm should be coated with approximately 1 teaspoon of a spermicide inside the dome and rim, inserted less than 3 hours before coitus, and left in place for at least 6 hours after intercourse (so that the spermicide remains effective), but no more than 24 hours.[27] If another act of intercourse occurs, the diaphragm should not be removed, but another application of spermicide is then applied intravaginally.

Side effects of diaphragm use include an increased risk for urinary tract infections as

compared to other contraceptive methods, perhaps due to pressure on the urethra exerted by the diaphragm's rim, causing incomplete bladder emptying, or from the spermicide altering the vaginal flora, which may increase the risk of *Escherichia coli* bacteriuria.[28] If the woman experiences recurrent urinary tract infections, the diaphragm may be too large or the rim too rigid, and refitting is indicated. If refitting does not resolve the problem, the woman can be counseled to select another contraceptive method.

Local irritation from the nonoxynol-9 spermicidal agent used may occur. If the diaphragm has been improperly fitted or is left in place longer than 24 hours after having intercourse, vaginal wall abrasions can occur. When recurrent vaginal or vulvar irritation occurs without evidence of an infection, these symptoms may indicate allergy to the diaphragm itself, especially if it is latex. Although latex diaphragms are no longer being produced, some women may have an older one that is made of latex. In such a case, the woman should be advised to discontinue use or switch to a nonlatex device. In addition, the risk of toxic shock syndrome may increase—though it remains low—if the diaphragm is left in place for more than 24 hours.

Contraindications to use of a diaphragm are the same as those to use of spermicide and are related to the spermicide component of this contraceptive method. Diaphragms are classified into the U.S. MEC for Contraceptive Use Category 3 for women with a history of toxic shock syndrome, because toxic shock syndrome has been associated with use of the device.[2] Occasionally a diaphragm cannot be fitted for a woman who has an anatomic abnormality such as pelvic organ prolapse or vaginal septum. The diaphragm does not protect against STI transmission.

A general consensus exists that a woman should be examined for a refit of a diaphragm if she experiences weight change of more than 15 pounds, has had a second-trimester abortion, or has had vaginal birth within 6 weeks, although these guidelines are empiric and not based on strong evidence.[25] The diaphragm should not be used after birth until uterine involution is complete and the vagina has regained its tone. All women using a diaphragm should be instructed about the symptoms of toxic shock syndrome.

Cervical Cap

The cervical cap is a dome-shaped silicone cap that resembles a sailor's hat (**Figure 16-2**).[30] The concave dome fits snuggly over the cervix and is held in place by the muscular walls of the vagina. The brim is slightly wider on the side that fits into the posterior fornix. The cap has a strap that stretches over the diameter of the dome, which facilitates removal. Spermicide is applied inside the dome, around the brim, and in the groove between the dome and the brim prior to insertion of the device.[29]

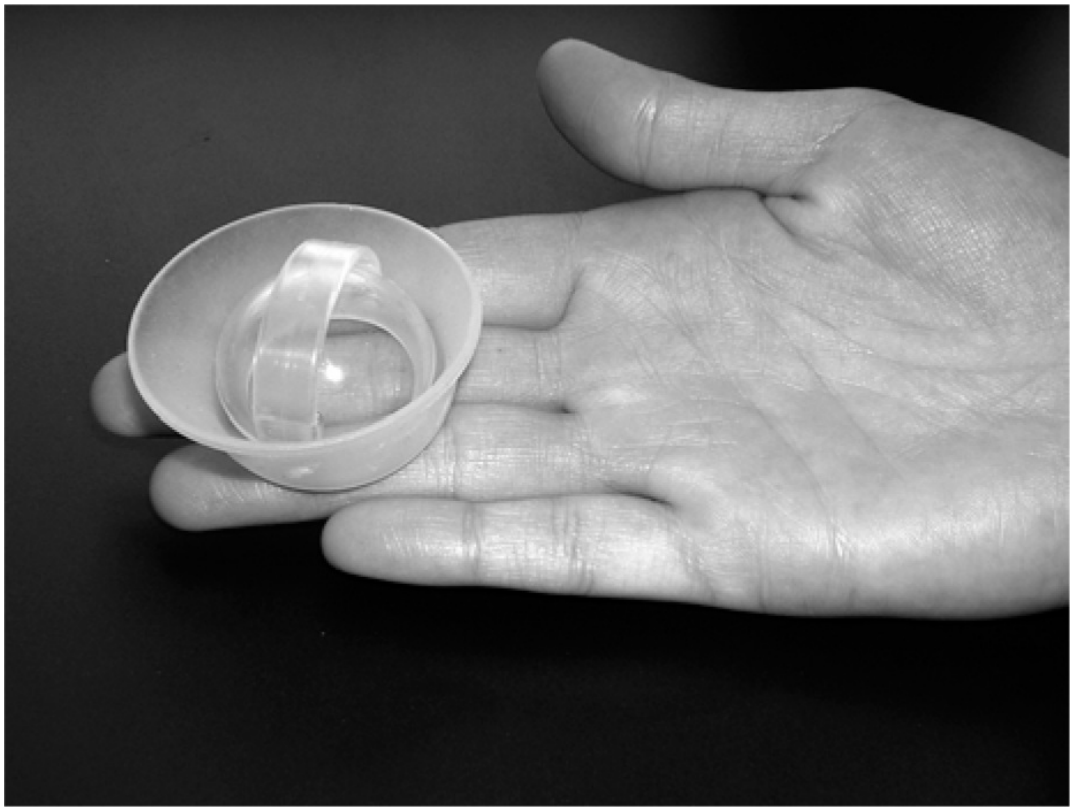


Figure 16-2 Cervical cap (FemCap).

Reproduced with permission from Cervical Barrier Advancement Society and Ibis Reproductive Health.

The only cervical cap currently available in the United States is marketed with the brand name FemCap.[30] Originally approved by the U.S. Food and Drug Administration (FDA) in 2003, it is available in three sizes, with size determined by a woman's pregnancy history: 22 mm if nulligravida, 26 mm if ever pregnant (nullipara), and 30 mm if the woman has had a full-term vaginal birth. A prescription is needed, and the device is available at selected sites or online. The advantage of the cervical cap is that it can be inserted as long as 42 hours before intercourse, thereby avoiding interruption of foreplay; it should be left in situ for 6 hours after intercourse.[29,31] When the cap is inserted more than 1 hour prior to coitus, an application of spermicide should be added without removing the cap.

In the original clinical trial reported to the FDA prior to approval of the originally designed cap, the 6-month effectiveness rate was 77% overall, but 86% for nulliparous women.[29,31] The current FDA-approved FemCap is a different design; only limited data on its effectiveness are available, and it is unclear whether it is as effective or even more effective than the diaphragm.[31]

Contraindications to use of a cervical cap are the same as the contraindications to use of spermicides and diaphragms, with a few additions. Women who have cervical cancer, cervical intraepithelial neoplasia, or a marked abnormally shaped cervix should be advised to use another form of contraception.[29] Using the cervical cap during menstruation may increase the risk of toxic shock syndrome and, therefore, should be avoided.[31] The cap does not protect against STI transmission, so using a male condom in combination with the cap adds this

protection.

While a pelvic examination is not needed to select the correct size, a clinician should insert and remove the device to illustrate proper technique for the woman, and then have the woman perform a return demonstration. **Figure 16-3** illustrates how a woman can insert a cervical cap in a manner similar to inserting a diaphragm (Appendix 16B). The mild suction exerted by the device allows it to fit snugly next to the cervix; it is properly placed when it does not move easily away from the cervix. The cap is most easily inserted before sexual arousal because sexual arousal can cause the cervix to become slightly soft. Removal is accomplished in a manner similar to removing a diaphragm: The woman inserts a clean finger into her vagina, firmly moves the cap off the cervix, and removes it by slipping it down the vagina and forward.

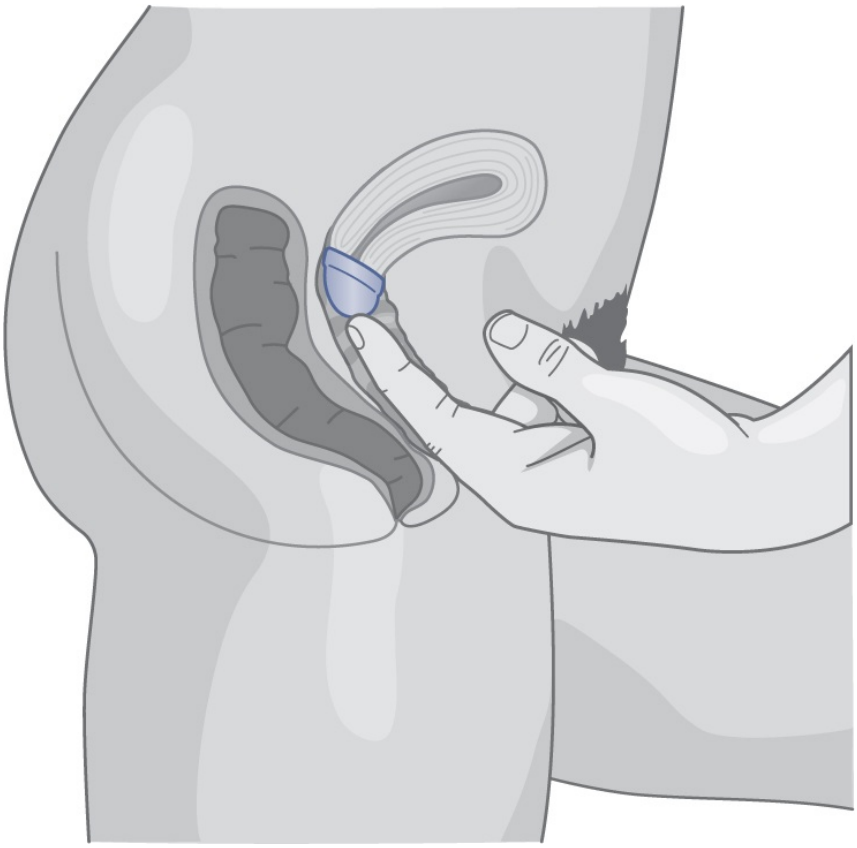


Figure 16-3 Proper insertion of a cervical cap by a woman.

Urinary tract infections occur less frequently in women using the cap compared to the diaphragm, and vaginitis at similar rates to those experienced by diaphragm users.[29] The cap should be replaced after each year of use.[29] The size should not need to be changed unless the woman's history changes—for example, she was nulligravid when the size was selected, but since last using it she has experienced a miscarriage, abortion, or given birth.

Male Condom

Contraceptives for men have long been a subject of research.[32],[33] Even so, only two methods are currently available: a barrier method, the male condom, and coitus interruptus. Male

condoms usually are sold over the counter and packaged inside a foil or plastic wrapper.

Many male condoms available in the United States are made of natural rubber latex. Condoms composed of polyurethane, a synthetic material, provide an option for individuals who are allergic to latex. Compared to latex condoms, the polyurethane condoms are odorless and colorless, fit more loosely, have a longer shelf life, and can be used with any lubricant.[34] Latex condoms are effective in preventing transmission of HIV and STIs; however, the effectiveness of nonlatex condoms in protection against HIV and STIs has not been established.[34]

Condoms come in a variety of colors, textures, transparencies, sizes, and shapes, although polyurethane condoms are more expensive than latex ones. Both types can be used either dry or with lubricants. Oil-based lubricants such as petroleum jelly or massage oils should not be used as they can cause breakage of the latex. Condoms used in conjunction with spermicides have enhanced effectiveness compared to those used either dry or with nonspermicidal lubricants. However, the spermicide should be used judiciously, as applying too much of the agent can cause slippage of the condom.

Male condoms remain the most widely available and commonly used barrier method in the United States. Approximately 16% of women using a contraceptive have reported that their male partners use condoms, making it a popular method for preventing pregnancy.[1,6] When used correctly and consistently with each coitus, during the first year of use among typical users, approximately 18% of couples would be expected to have an unintended pregnancy.[3] Most condom failures result from breakage or slippage during intercourse or while removing the condom. In a meta-analysis of 11 randomized trials that compared latex and synthetic condoms, failure rates were comparable for all latex and synthetic condoms, although most synthetic condoms had higher reported rates of breakage and slippage.[34]

Condoms can cause vaginal irritation or discomfort, with synthetic condoms reported to cause less vaginal irritation and discomfort than the latex versions.[35] Disadvantages include perceived reduced sensitivity and lack of spontaneity during sexual activity, especially during foreplay, when the condom should be worn, as well as latex allergy of either partner. Some men have difficulty maintaining erections while the condom is on, and some individuals may be embarrassed to use a condom or ask that one be used. Lastly, male condoms are male controlled. Women in relationships where they cannot negotiate condom use by their partner may be exposed to risks of unwanted pregnancy and/or STIs.

Condoms can be used as an adjunct to other contraceptives. For example, if a woman using combined oral contraceptives misses one or more pills, using a condom for the duration of the pill cycle provides protection from pregnancy. Dual use of condoms with another contraceptive should also be advocated for women with multiple or new partners to reduce risk of STIs, and for women seeking added protection against unintended pregnancy.[36]

Reviewing correct use is best done by demonstration, using a penile model or a substitute such as a banana. Basic instructions include the description that the male condom is a strong, thin, elastic sheath that is applied to the tip of an erect penis and unrolled to catch the seminal fluid during ejaculation and prevent it from being deposited in the vagina. Some space should be left in the tip of the condom to have a repository for semen; otherwise, the fluid can be

forced outside the base. In the event of condom breakage or slippage, emergency contraception is available as an option, as described in the *Family Planning* chapter.

Female Condom

The female condom is a soft, loosely fitting, thin sheath (**Figure 16-4**).[37] It has two flexible rings—one on the closed end of the sheath that is inserted into the vagina, and a larger ring on the open end that remains outside the vagina and covers the introitus. The female condom is available in only one size and does not need to be fitted by a healthcare professional.[38] Although the product may be prelubricated with a silicone-based, nonspermicidal agent, additional lubricants or spermicidal preparations may be used with it. Each condom is intended for one-time use. Current female condoms are made of nitrile, latex, or silicon to diminish the issue of crinkling sound with the previous generation of polyurethane-based female condoms.



Figure 16-4 Female condom.

Reproduced with permission from Cervical Barrier Advancement Society and Ibis Reproductive Health.

The effectiveness of the female condom is generally considered slightly less than that of male condoms, with approximately 79% of users in the first year of typical use avoiding unintended pregnancy.[3] The discrepancy in failure rates between perfect use and typical use may reflect problems mastering insertion as well as inconsistent use.[39] The female condom protects as effectively as male condoms against STIs and HIV transmission when used correctly, in part because the external portion provides a barrier between the labia and the

base of the penis during intercourse.[3,28]

The female condom can be inserted as long as 8 hours before sexual intercourse, but must be in place before the penis enters the vagina. Some couples prefer the freedom of movement and looseness of the female condom compared to the male condom. Some women have reported that the edge of the outer ring provides clitoral stimulation. Both partners may experience uncomfortable sensations, including feeling the inner ring, having the condom adhere to the penis, and feeling the outer ring press against the vulva during intercourse. Checking for correct placement and adding more lubricant can alleviate these problems. Learning the correct steps for insertion may be challenging, but can be achieved with close instruction. **Table 16-3** reviews the steps involved in correct use of the female condom. Most women need to practice two to three times to successfully place the condom.[39]

Table 16-3	General Instructions for Use of Female Condom
Insertion	
Lubricate the outside of the closed end with spermicide.	
Relax in a comfortable position (e.g., lie down, squat, stand with one foot on a chair).	
Squeeze the sides of the inner ring at the closed end together.	
Insert the condom deep in the vagina, in similar fashion as inserting a tampon.	
Slip the inner ring into the vagina as far as possible (until it reaches the cervix).	
Allow the outer ring to hang approximately 1 inch (2.5 cm) outside the vagina.	
Removal	
Squeeze and twist the outer ring to keep semen inside the pouch.	
Gently remove the condom directly out of the vagina.	
Discard the condom, but do not flush it down the toilet.	

The female condom usually moves from side to side in the vagina during vaginal intercourse. However, if the penis slips between the condom and the walls of the vagina, or if the outer ring is pushed into the vagina, the woman is at risk for contraception failure. As long as the male has not ejaculated, the condom can be removed, repositioned, and additional spermicide added. The female condom also has been used by some individuals for anal intercourse, but this use is not FDA approved. In the event of condom breakage or slippage, emergency contraception is available as an option, as described in the *Family Planning* chapter. The female condom does protect against STIs.

The Intrauterine Device

Several synonyms are used to describe IUDs, including intrauterine contraceptives (IUCs), intrauterine contraceptive devices (IUCDs), and intrauterine system (IUS). For consistency, the term IUD is used throughout this chapter. IUDs and contraceptive implants are the two types of long-acting reversible contraceptive (LARC) devices available in the United States. IUDs are the most commonly used reversible contraceptive globally, although in the United States among women using a birth control method, more continue to choose oral contraceptives.[40]

Rates of IUD use were increasing in the United States during the 1970s, but decreased significantly following highly publicized pelvic inflammatory disease (PID) episodes linked to the Dalkon Shield before this device's eventual removal from the market.[41] Thus, the current generation of reproductive-age women has been underexposed to IUDs as a method of contraception. Multiple factors have contributed to the lower prevalence of IUD use in the United States, including a lack of widespread marketing, especially compared to oral contraceptives; common misconceptions about IUDs' mechanism of action; misinformation about associated risks; and a history of negative publicity. Recent surveys of contraceptive preference, however, indicate that IUDs are increasing in favor among women of various age groups, perhaps indicating a resurgence of the method's acceptability in the United States.[3,6]

IUDs have been found to be among the most effective reversible forms of contraception. In addition to their effectiveness, an emerging body of research suggests that these methods may provide protection against cervical cancer.[42] Additional study needs to be conducted in the area to confirm this benefit.

Types of Intrauterine Devices

IUDs are classified as either hormonal or nonhormonal. Two types of T-shaped IUDs are available in the United States: one impregnated with copper (Copper T 380A [ParaGard]), and the other impregnated with levonorgestrel (levonorgestrel-releasing intrauterine system [LNG-IUS or LNG-IUD]). More commonalities exist for the two types than differences. Therefore, this chapter presents an overview of both types of IUDs and specific information about the nonhormonal copper IUD. Use of the copper IUD for emergency contraception is described in the *Family Planning* chapter. Information specific to the LNG-IUD is addressed in the *Hormonal Contraception* chapter.

Mechanism of Action of Intrauterine Devices

The primary mechanism of action of both types of IUDs is prevention of fertilization via inhibition of sperm mobilization and sperm viability as well as changes in transport speed of the ovum in the fallopian tube transport.[21] The two types of IUD have additional effects based on their major components.

Contraindications to Use of Intrauterine Devices

Contraindications to any IUD use and those specific for the copper containing IUD are listed in **Table 16-4**.[2] Contraindications for use of the LNG-IUD are listed in the *Hormonal Contraception* chapter. Any abnormal uterine bleeding should be evaluated for pathologic causes prior to IUD insertion. An IUD should not be used if the woman has a known hypersensitivity to any device component. The copper IUD is contraindicated for women with Wilson's disease—a condition that causes impairment of copper metabolism.

Table 16-4 **Contraindications for Use of Any Intrauterine Device: U.S. Medical Eligibility Criteria for Contraceptive Use Categories 3 or 4[a]**

Contraindications	Medical Eligibility Criteria for Contraceptive Use Category
Any IUD	
Cervical cancer awaiting treatment	4 for initiation; 2 for continuation
Distorted uterine cavity	4
Endometrial cancer	4 for initiation; 2 for continuation
Gestational trophoblastic disease with decreasing or undetectable beta-hCG levels	3
Gestational trophoblastic disease with persistently elevated beta-hCG levels or malignant disease	4
Immediately post septic abortion	4
Pelvic inflammatory disease	4
Pelvic tuberculosis	4 for initiation; 3 for continuation
Pregnancy	4
Puerperal sepsis	4
Sexually transmitted infections	
Chlamydia	4 for initiation; 2 for continuation
Gonorrhea	4 for initiation; 2 for continuation
Purulent cervicitis	4 for initiation; 2 for continuation
Increased risk for STI	3 if a woman has a very high risk of chlamydia or gonorrhea; otherwise 2
Solid organ transplant	3 for initiation; 2 for continuation
Unexplained vaginal bleeding that is suspicious for a serious condition	4
Copper IUD	
Allergy to copper	Listed in product label
Use for emergency contraception in case of rape	3 if high risk for STI; 1 if low risk for STI
Systemic lupus erythematosus with severe thrombocytopenia	3 for initiation; 2 for continuation
Wilson's disease	Listed in product label

Abbreviations: hCG, human chorionic gonadotropin; IUD, intrauterine device; STI, sexually transmitted infection.

[a] U.S. Medical Eligibility Criteria for Contraceptive Use Categories: Category 1: a condition for which there is no restriction; Category 2: a condition for which the advantages of using the method generally outweigh the theoretical or proven risks; Category 3: a condition for which the theoretical or proven risks usually outweigh the advantages of using the method; Category 4: a condition that represents an unacceptable health risk if the contraceptive method is used.

Modified with permission from Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR*. 2016;65(3):1-106.[2]

The procedure for inserting and removing an IUD is described in **Appendix 16C**. This appendix includes discussion of timing and pre-insertion medication and side effects associated with insertion for both types of IUDs.

Adverse Effects Associated with Intrauterine Devices

Women should be counseled that use of IUDs may be associated with changes in menstrual bleeding patterns. The copper IUD often increases the amount of menstrual bleeding, whereas the LNG-IUD often decreases the amount of menstrual bleeding. Some inherent risks are related to the minimally invasive procedures of IUD insertion and removal. Bleeding, infection, perforation of the uterus, and pain can occur, although serious complications are rare. **Table 16-5** lists effects that should be considered for follow-up visits by using the mnemonic "PAINS."

Table 16-5 PAINS Mnemonic for Adverse Effects of Intrauterine Devices

Period	Amenorrhea, especially sudden onset; with the copper IUD, may be associated with pregnancy. Light bleeding and amenorrhea are side effects of LNG-IUDs.
Abdominal pain	General abdominal pain can suggest an ectopic pregnancy, sexually transmitted infection, or intolerance of copper.
Infection	Vaginal discharge, pelvic pain, history of exposure, or any suspicion of sexually transmitted infections.
Not feeling well	Malaise, fever, nausea, and vomiting suggesting infection or sepsis.
Strings	Strings or threads of the IUD changing in length or missing entirely.

Abbreviations: IUD, intrauterine device; LNG-IUD, levonorgestrel-releasing intrauterine device.

Intrauterine Device Expulsion

Expulsion rates are generally low (2–10%) following insertion of an IUD in a nonpregnant woman. Expulsion is most likely to occur during the first-year post insertion. Expulsion rates among devices are relatively similar, and adolescents were more likely to experience expulsion regardless of parity.[43] The rate of expulsion in the immediate postpartum period is typically higher than at other times, but women who receive immediate insertion of an IUD following childbirth are more likely to demonstrate continued use as compared to women who defer IUD insertion until a postpartum office visit.[44] Some researchers suggest that insertion after the 14th postpartum day is associated with an expulsion rate equal to that of insertion at 6 weeks or later.[45]

Encouraging regular string or thread checks can help detect expulsion. Education should include signs and symptoms of expulsion, including detection of the device at the os, pain and cramping, abnormal uterine bleeding, discharge, or fever.

Missing Intrauterine Device Strings

A clinician's inability to find IUD strings during an examination can be attributed to four possible causes: uterine perforation that leaves the IUD in the abdomen, spontaneous expulsion, strings cut too short, or, rarely, pregnancy in the second or third trimester when the uterus has enlarged and the device risen in the uterus.[46] Pregnancy should be considered whenever strings are not visible. Often strings have simply receded into the cervix, out of view, or the threads have curled behind the cervix and become difficult to palpate. Gentle cervical exploration with a cytobrush or cotton swab may be able to guide and straighten the strings—a technique colloquially termed "fishing" for the strings. Care should be taken not to pull on the threads and dislodge the device. If the strings are not apparent to the clinician during a clinical examination, it is possible that the device was spontaneously expelled and the woman is unaware of its loss. An ultrasound can identify whether the IUD is in situ. **Figure 16-5** provides an algorithm for managing the care of a woman with missing IUD strings.

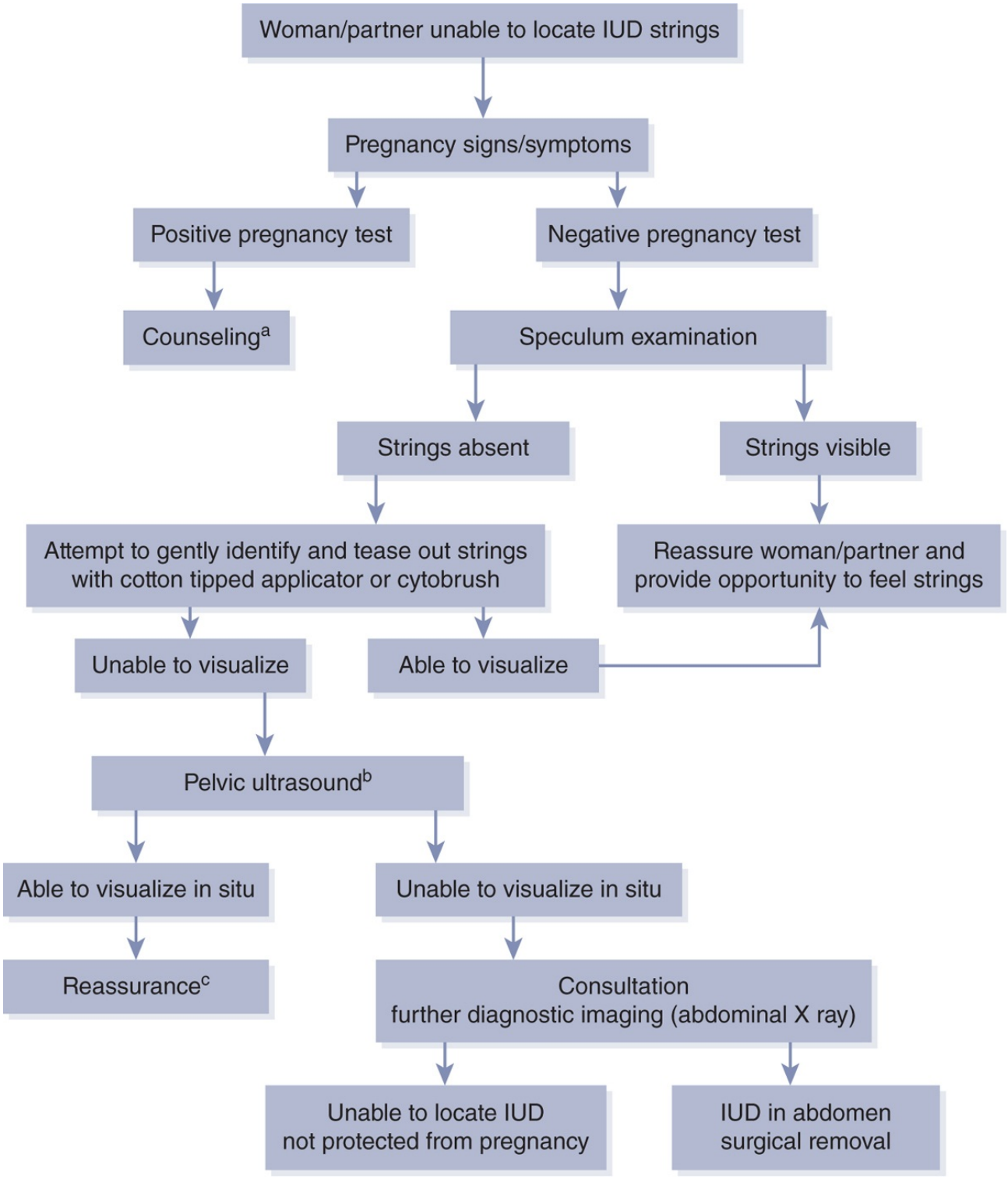


Figure 16-5 Algorithm for management of a woman with missing intrauterine device strings.

Abbreviation: IUD, intrauterine device.

[a] Counseling is required for shared decision making with risks and benefits outlined.

[b] Pelvic ultrasound may be ordered, or care may be transferred to a specialist based on practice policies at this point.

[c] Reassurance can be given that the IUD is providing protection from pregnancy, but some women may request a removal and replacement for regular reassurance by feeling strings.

In the unusual event of the IUD being appropriately positioned in the fundus but the threads are not palpable, the woman needs to make an informed decision regarding whether she wishes to have the IUD removed and replaced or to continue as is. Contraceptive effectiveness is not dependent upon the threads extending from the uterus into the vagina. If the ultrasound reveals that the IUD either is dislodged and no longer in the expected placement (e.g., in a fallopian tube) or has perforated the uterus and entered the abdomen, immediate referral is indicated.

Pregnancy with an Intrauterine Device in Situ

A woman who becomes pregnant with an IUD in place should be informed of the risks involved if the pregnancy is continued—namely, chorioamnionitis, spontaneous abortion, septic abortion, and preterm birth.[47] Although the risk of pregnancy is small with an IUD in place, women who become pregnant with an IUD in place should also be evaluated for ectopic pregnancy, as the risk of any pregnancy being ectopic is higher with an IUD in situ.

If pregnancy is confirmed, an ultrasound reveals the placement of the IUD, and the IUD strings are visible, the IUD should be removed to decrease the risk of spontaneous abortion. The incidence of spontaneous abortion is slightly higher if the IUD remains than if the device is removed.[47,48] This benefit is most apparent when the IUD is removed during the first 12 weeks' gestation. Removing the IUD for a woman who wishes to terminate the pregnancy reduces her risk of septic abortion.[48] The IUD is removed in the usual fashion as described in Appendix 16C. Often a woman who has an IUD in situ during pregnancy is referred to a physician, although midwives can accomplish the procedure as long as the woman understands the risks and benefits, she is in the first trimester of pregnancy, and the threads are visible.

When pregnancy is confirmed and the strings of the IUD are not visible, ultrasound may reveal that the device has been covertly expelled, such as with a heavy menses; the woman may not have been aware of its loss. If the ultrasound reveals the IUD placed abnormally (e.g., within a fallopian tube) or in the uterus but without strings visible the woman should be referred to a specialist.

Copper T 380A (ParaGard)

The only nonhormonal IUD currently available in the United States, known as ParaGard, is composed of polyethylene and an inert plastic material that is flexible, is non-inflammatory, and resumes its original shape easily after being flexed for insertion (**Figure 16-6**). The vertical stem of the polyethylene body is wrapped with 176 mg of copper wire, while each of its horizontal arms has a 68.7-mg copper collar attached.[49] The copper components of ParaGard release ions into the endometrial cavity that affect tubal and endometrial fluids, and subsequently incapacitate sperm. The ParaGard IUD has a length of plastic thread attached to the lower segment that extends through the cervix into the vagina when the IUD is in place. The string facilitates removal and enables the midwife and the woman to confirm the presence of the device. Embedded within this IUD's polyethylene body is a small amount of barium sulfate, which allows the device to be localized with standard X-ray imaging. ParaGard provides contraception for as long as 10 years after insertion.

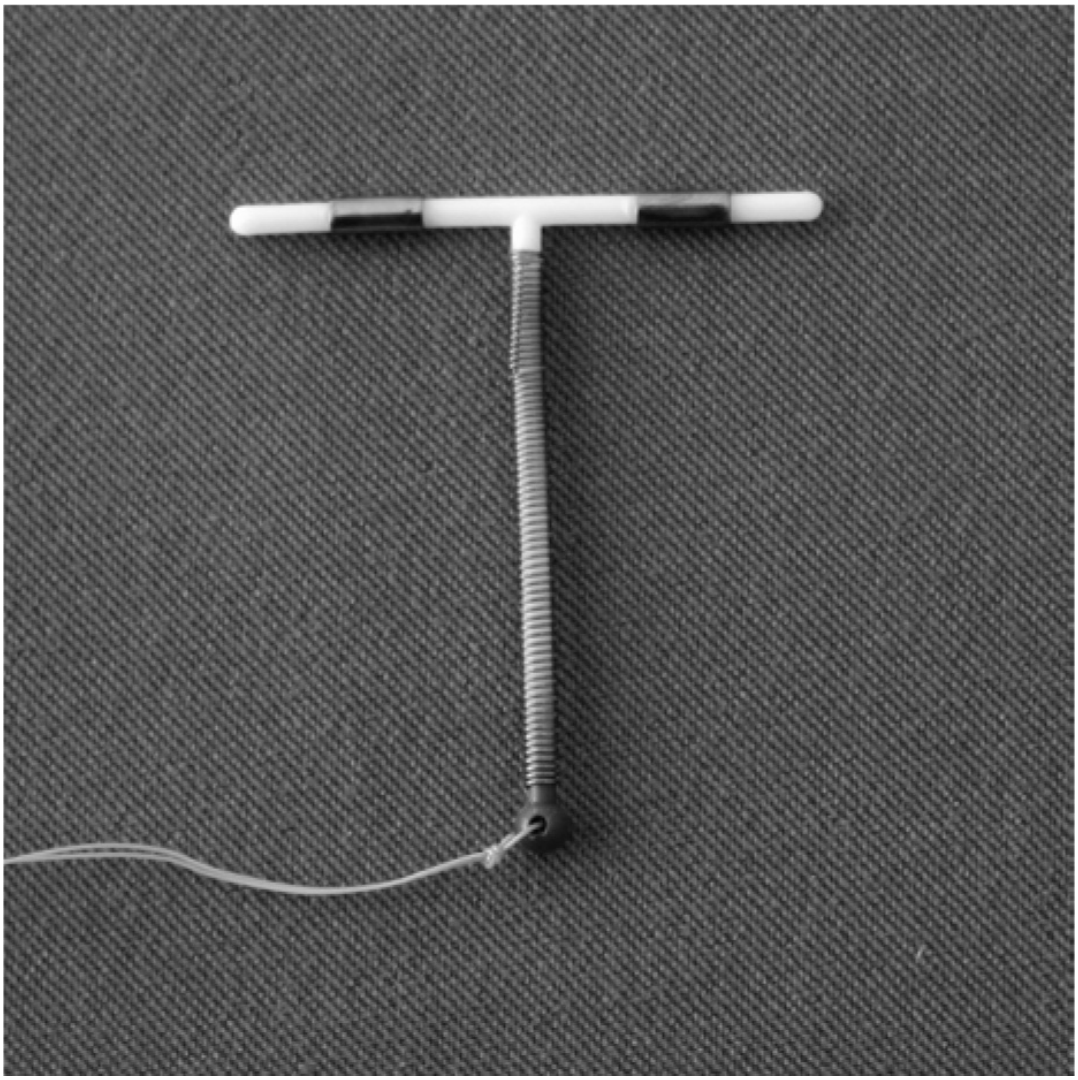


Figure 16-6 ParaGard intrauterine device.

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The effectiveness rate for ParaGard is high, with fewer than 1 in 100 users experiencing an unintended pregnancy in the first year of use.[2] The copper in the IUD alters sperm tubal transport, has toxic effects on the ovum, and impairs normal sperm activity by slowing motility, reducing sperm capacitation, and increasing sperm destruction. In addition, the copper creates a localized reaction[50] in the endometrial tissue that renders the uterine endometrium unfavorable for implantation.[51]

The copper IUD is associated with menstrual changes such as 1 to 3 more days of bleeding per cycle, increased severity of dysmenorrhea, and potential increase in blood loss, which can exacerbate iron-deficiency anemia. Consequently, ParaGard is not a good choice of contraception for women who have heavy menstrual bleeding (menorrhagia). Copper-bearing IUDs appear to convey protection against endometrial cancer (odds ratio [OR], 0.54; 95% confidence interval [CI], 0.47–0.63), although the mechanism of action has not been elucidated.[52]

Use of a copper IUD is noncoital dependent, does not require further consideration of cost

after the initial insertion, and is effective for as long as 10 years. Some women find the IUD-associated side effects such as bleeding too discomfiting, and others do not like the idea of having a foreign object inside their body. When these concerns are not a consideration and for women who do not desire any hormones for contraception, the copper-containing IUD may be a good choice.

Permanent Contraception: Sterilization

Sterilization is the surgical interruption or closure of pathways for sperm or ova to unite, which prevents fertilization. Since the 1970s, female sterilization has become an increasingly available and acceptable contraceptive choice.[53] Sterilization, an elective procedure, is considered a safe, highly effective, and permanent form of contraception. Overall, both female and male sterilization (vasectomy) are more than 99% effective in preventing pregnancy.[2]

Methods of female sterilization include: (1) laparoscopy tubal ligation, (2) tubal interruption, (3) hysteroscopy (transcervical) sterilization, and (4) the chemical agent, quinacrine (Atabrine) that is not available in the United States. Male sterilization, or vasectomy, is both a safer and simpler procedure compared to female sterilization. Vasectomy is also, approximately half the cost of female sterilization, although female sterilization is more common in the United States.[54] In fact, in the United States, female sterilization is one of the most frequently performed surgeries for a woman. The term “tying tubes” is a common phrase, but this wording should be avoided as it may incorrectly connote that the fallopian tubes could later be untied. Fallopian tubes may be surgically cut and ligated with or without a section of the tube being removed, mechanically blocked using clips or rings, electrically coagulated, or blocked by a fibrotic reaction induced by chemicals or micro-inserts. Reversal of a tubal ligation is actually a complicated and costly procedure with a high failure rate. Female sterilization procedures can be performed soon after a vaginal birth (postpartum sterilization), in conjunction with a cesarean section, immediately following an uncomplicated first-trimester abortion, or independent of pregnancy (interval sterilization).

Female Surgical Sterilization

There are two common approaches to female surgical sterilization: laparoscopy, or transcervical. Female sterilization may also be accomplished via a mini-laparotomy but this technique is not commonly used in the United States. Sterilization via laparoscopy is the standard approach for women who desire postpartum sterilization. Women who desire interval sterilization may chose laparoscopy or hysteroscopy tubal interruption

Only 0.5% of women experience an unintended pregnancy within the first year following surgical sterilization, which translates into a 99.5% effectiveness rate.[2] This number includes those women with an unsuspected pregnancy at the time when the procedure is performed. Failure can also happen many years after the procedure is performed. Because spontaneous pregnancy after tubal ligation is rare, the overall risk of ectopic pregnancy is lower in women who undergo surgical sterilization than in the general population. If pregnancy does occur after tubal sterilization, approximately one-third are ectopic.[53]

Overall, there are few, if any, contraindications to sterilization for the individual who desires this form of contraception. Sterilization is a surgical procedure that requires the surgeon’s consent to perform the procedure. It has been reported that some surgeons have personal requirements of age or parity prior to offering sterilization to women. Requirements based on age or nulliparity are not justified.[53] Conversely, reports of specific requirements for male sterilization have not been identified.

Counseling prior to informed consent includes a review of alternatives, risks associated with the procedure and use of anesthesia, permanency of contraception, and the risk of ectopic pregnancy should a pregnancy occur after the procedure. Women also should be informed that sterilization does not prevent transmission of STIs or HIV.

Sterilization is not coital dependent, nor does it require partner agreement or purchasing of supplies. Some studies show a positive effect on sexuality, which is possibly related to reduced worry about unintended pregnancy.[54] Complications are those associated with any surgical procedure.

Regret that a sterilization procedure was performed has been reported.[55] Various studies indicate that the percentage of women who regret their decision to undergo sterilization ranges from 1% to slightly less than 30%.[2,55,56] Higher rates of regret are found in women who were younger than age 30 years at the time of sterilization, are in an unstable relationship, have experienced the death of a child, and are of low parity.[55,56]

Historically women of color were sterilized without consent in some locales. Today, black women are more likely to undergo sterilization than are white women even when confounding factors such as parity and age are controlled for. Although the reasons for this difference have not been fully elucidated, there is some thought that black, Hispanic, and Native American women may be more likely to receive counseling and education about sterilization. Despite a lack of full understanding of these statistics, it is a reminder that healthcare provider overt or implicit bias must not be present in contraceptive counseling.[56]

The midwife must be aware of applicable federal and state regulations about sterilization. Because of abuses related to forced sterilization in the past, strict criteria must be met to verify informed consent, including the requirement that the consenting woman is an adult. Consent is to be obtained within a specified time period (usually 30 days) before giving birth, and the time interval may differ by state, facility, and payer.[53] As with any shared decision-making discussion, the woman should receive information that is at the appropriate health literacy level, in her native language, and she should be informed that she can decline the sterilization even after the consent form has been signed.

Reversal of sterilization is expensive, requiring either costly assisted reproductive technology or highly technical microsurgery, and results cannot be guaranteed. Successful pregnancy following sterilization reversal varies with the type of tubal occlusive method used and with the age of the woman. Rates of successful reanastomosis following reversal procedures range from 30% to 70%. The long-term effects of tubal sterilization on menstrual pattern (post-tubal ligation syndrome) appear to be negligible. Most recent studies have not found any difference in menstrual patterns between women before and after sterilization.[53]

Laparoscopy Sterilization

Early studies conducted when tubal occlusion techniques were relatively new found electrosurgery and micro-clips were associated with higher pregnancy rates compared to tubal ligation. However, recent studies have found all the techniques for occluding or cutting the Fallopian tube have similar effectiveness. Thus, the technique chosen is based on the surgeon’s preference and skill.

Hysteroscopic Sterilization (Transcervical Sterilization)

The transcervical female sterilization method involves gaining access to the fallopian tubes via hysteroscopy that is introduced through the cervix. This method can be accomplished on an outpatient basis under local anesthesia.[53,57] The only currently available transcervical method is marketed under the brand name Essure. The Essure system consists of stainless steel coils that are soft and flexible because they are wrapped in polyethylene terephthalate covered with a nickel–titanium alloy. The small metal springs are placed into the proximal end of each fallopian tube. Upon release, they expand and anchor to the tube, creating scar tissue.[53] Thus, the mechanism of action involves irritation and growth of new tissue that results in permanent occlusion of the fallopian tubes as the tubes respond to the polyester fibers within the micro-inserts. Tubal occlusion occurs approximately 3 months after the procedure is performed. The tissue response has been found to be localized to the insert.

Sterilization does not occur immediately after the procedure, so women need to use additional contraception for 3 months until permanent tubal occlusion is verified by hysterosalpingogram. If this confirmation hysterosalpingogram reveals there is still tubal patency, the woman is instructed to use an alternative contraceptive method. Other methods for verification of tubal occlusion have been suggested, such as ultrasound, but the gold standard for confirmation remains the hysterosalpingogram.

Long-term data on safety, effectiveness, and pregnancy rates for Essure remain controversial, as many of the existing studies were funded by the manufacturer.[57] U.S. and worldwide literature indicates that this sterilization technique has a greater than 98% effectiveness.[57] Other clinical trials and reviews have reported unintended pregnancies that appear to be related to failure to strictly comply with the follow-up protocol, failure to perform a urinary pregnancy test on the day of the procedure, or failure to instruct the woman to return for the hysterosalpingogram follow-up visit to confirm bilateral occlusion via the injection of radiopaque contrast material into the uterus to detect fallopian tube patency.[57] Studies have underscored the importance of the 3-month follow-up, as some women may not develop tubal occlusion until later. In addition, expulsion of the micro-inserts has been observed and can lead to contraceptive failure.[58]

In 2017, the FDA mandated a black box warning for Essure based on reports of adverse effects such as chronic pelvic pain, irregular bleeding, allergic reactions, and autoimmune-like reactions.[53,58-60] Additional study is warranted regarding these reports. Because the Essure coil contains nickel, a known hypersensitivity to nickel confirmed by skin test is a contraindication to placement of the micro-inserts.[58]

Transcervical sterilization may be an option for women who have comorbidities, such as obesity, cardiorespiratory disease, or complex abdominal issues that make other forms of permanent sterilization difficult to accomplish or contraindicated.[59] Like all permanent sterilization options, this procedure is not coital dependent, does not require partner agreement, and avoids the need to buy further supplies, enhancing the convenience factor.

Female Chemical Sterilization

Quinacrine, which was originally approved as an antimalarial drug, is the best-studied chemical agent used for female sterilization. This agent is most commonly used in low-resource countries because it is inexpensive and does not require expensive equipment for placement. However, particularly because of an association with cancer, the World Health Organization (WHO) recommended that until totality of the safety, effectiveness, and epidemiologic data has been reviewed, quinacrine should not be used for nonsurgical sterilization in women.[61] Quinacrine pellets are not currently available in the United States for sterilization.

Male Surgical Sterilization (Vasectomy)

Vasectomy is one of the few contraceptive options currently available for men. Sterilization is achieved by cutting or occluding the vas deferens so that sperm can no longer pass out of the body in the ejaculate. This procedure can be done in an outpatient setting under local anesthetic by a trained provider. Advantages of this method include the short procedure time and the reduced risk of hematoma, infection, and postoperative discomfort when compared to female tubal ligation.[62]

Vasectomy is a highly effective and relatively low-cost permanent method of sterilization with a low morbidity rate and an extremely low mortality rate. The incidence of pregnancy 5 years after the procedure was performed is less than 1%.[2] Vasectomy is not immediately effective, however: It may take 12 weeks or more, or between 12 and 20 ejaculations, before the ejaculate is sperm free. Alternative contraception needs to be used until azoospermia is confirmed.

Individuals should be informed that recanalization does occur, albeit infrequently. Regret about undergoing a vasectomy has been reported among approximately 5% of men who have the procedure done.[62] The majority of men who report regret had the procedure when they were younger than age 30 years, were in an unstable marriage, had no or very young children, or made the decision to have a vasectomy during a time of financial crisis or for reasons related to a pregnancy.[62]

Conclusion

This chapter reviewed currently available nonhormonal methods of contraception for both women and men in the United States. Options for contraception change frequently, as do aspects of a woman's life; therefore, midwives need to remain informed of the latest options so that the persons for whom they provide care can engage in meaningful shared decision making. Each individual has individual needs and a different lifestyle, which make decision making unique. For many, nonhormonal methods can provide the primary or secondary contraceptive method that they desire.

Resources

Organization	Description	Webpage
Center for Disease Control and Prevention (CDC)	U.S. Medical Eligibility Criteria (U.S. MEC) for Contraceptive Use includes recommendations for the use of specific nonhormonal contraceptive methods and addresses how to initiate and use them. Charts, apps, and lists are available from the website.	https://www.cdc.gov/reproductivehealth/contraception/mmwr/mec/summary.html
Association for Reproductive Health Professionals (ARHP)	ARHP is dedicated to improving reproductive health care through a high-quality Continuing Professional Development (CPD) program. ARHP's CPD program is built on its positions, accepted professional standards, peer-reviewed studies, and a commitment to integrity in patient care.	http://www.arhp.org
Bedsider	An online birth control support network for women age 18–29 operated by the National Campaign to Prevent Teen and Unplanned Pregnancy, a private nonprofit organization.	https://www.bedsider.org
Billings Ovulation Method	Emphasis on education in the method. Will connect interested persons with an accredited Billings	http://billings.life/en/

	Ovulation Method tutor for more information.	
Creighton Model FertilityCare System	Emphasis on education in the method. Similar to Billings, but not identical.	http://www.creightonmodel.com
Two-Day Method	Information specific to this method. iOS app available free of charge.	http://www.twodaymethod.com
Point-of-Care Apps		
Multiple fertility apps to be used for pregnancy prevention, attaining pregnancy, or pregnancy spacing	In 2016, a publication ranked several apps as to accuracy. Most of these apps remain current.	Duane M, Contreras A, Jensen ET, White A. The performance of fertility awareness-based method apps marketed to avoid pregnancy. *J Am Board Fam Med*. 2016;29(4):508-511. Available at: http://www.jabfm.org/content/29/4/508.long. Accessed November 28, 2017.

References

1. Sundaram A, Vaughan B, Kost K, et al. Contraceptive failure in the United States: estimates from the 2006–2010 National Survey of Family Growth. *Perspect Sex Reprod Health*. 2017;49(1):7-16.
2. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR*. 2016;65(3):1-106.
3. Trussell J. Contraceptive failure in the United States. *Contraception*. 2011;83(5):397-404.
4. Moniz MH, Spector-Bagdady K, Heisler M, Harris LH. Inpatient postpartum long-acting reversible contraception: care that promotes reproductive justice. *Obstet Gynecol*. 2017;130(4):783-787.
5. Altshuler AL, Blumenthal PD. Behavioral methods of contraception. In: Shoupe D, Mishell D Jr., eds. *The Handbook of Contraception: Current Clinical Practice*. Switzerland: Humana Press Springer; 2016:247-262.
6. United Nations, Department of Economic and Social Affairs, Population Division. Trends in contraceptive use worldwide 2015 (ST/ESA/SER.A/349). Available at: http://www.un.org/en/development/desa/population/publications/pdf/family/trendsContraceptiveUse2015Report.pdf. Accessed November 28, 2017.
7. Arteaga S, Gomez AM. "Is that a method of birth control?": a qualitative exploration of young women's use of withdrawal. *J Sex Res*. 2016;53(4-5):626-632.
8. Killick S, Leary C, Trussell J, Guthrie K. Sperm content of pre-ejaculatory fluid. *Hum Fertil*. 2011;14(1):48-52.
9. Sznitman S, Romer D, Brown L, et al. Prevalence, correlates, and sexually transmitted infection risk related to coitus interruptus among African-American adolescents. *Sex Transm Dis*. 2009;36(4):218-220.
10. King J. Contraception and lactation. *J Midwifery Womens Health*. 2007;52(6):614-620.
11. Labbok MH. Postpartum sexuality and the lactational amenorrhea method for contraception. *Clin Obstet Gynecol*. 2015;58(4):915-927.
12. Kramer MS, Kakuma R. Optimal duration of exclusive breastfeeding. *Cochrane Database Syst Rev*. 2012;8:CD003517. doi:10.1002/14651858.CD003517.pub2.
13. Sandrock N. Share with women: natural family planning. *J Midwifery Womens Health*. 2006;51(6)51:521-522.
14. Malarcher S, Spieler J, Fabic MS, Jordan S, Starbird EH, Kenon C. Fertility awareness methods: distinctive modern contraceptives. *Glob Health Sci Pract*. 2016;25;4(1):13-15.
15. Grimes D, Gallo M, Grigorieva V, Nanda K, Schulz KF. Fertility awareness–based methods for contraception systematic review of randomized controlled trials. *Contraception*. 2005;72:85-90.
16. Mansour D, Inki P, Gemzell Danielsson K. Efficacy of contraceptive methods: a review of the literature. *Eur J Contracept Reprod Health Care*. 2010;15:14-16.
17. Unseld M, Rötzer E, Weigl R, Masel EK, Manhart MD. Use of natural family planning (NFP) and its effect on couple relationships and sexual satisfaction: a multi-country survey of NFP users from US and Europe. *Front Public Health*. 2017;13(5):42.
18. Duane M, Contreras A, Jensen ET, White A. The performance of fertility awareness–based method apps marketed to avoid pregnancy. *J Am Board Fam Med*. 2016;29(4):508-511. Available at: http://www.jabfm.org/content/29/4/508.long. Accessed November 28, 2017.
19. Raymond E, Dominik R. Contraceptive effectiveness of two spermicides: a randomized trial. *Obstet Gynecol*. 1999;93:896-903.
20. Van Damme L, Ramjee G, Alary M, Vuylsteke B, Chandeying V, Rees H. Effectiveness of COL-1492, a nonoxynol-9 vaginal gel, on HIV-1 transmission in female sex workers: a randomized controlled trial. *Lancet*. 2002;360:971-977.
21. Tepper NK, Krashin JW, Curtis KM, Cox S, Whiteman MK. Update to CDC's U.S. Medical Eligibility Criteria for Contraceptive Use, 2016: revised recommendations for the use of hormonal contraception among women at high risk for HIV infection. *MMWR*. 2017;66(37):990-994.
22. Gardella C, Ekert LO, Lentz GM. Genital tract infections: vulva, vagina, cervix, toxic shock syndrome, endometritis, and salpingitis. In: Lobo R, Gershenson D, Lentz G, Valea FA, eds. *Comprehensive Gynecology*. 7th ed. Philadelphia, PA: Elsevier; 2017:524-565.
23. Schwartz B, Brome C. Nonmenstrual toxic shock syndrome associated with barrier contraceptives: report of a case-control study. *Rev Infect Dis*. 1989;1(suppl S43-S48):S48-S49.
24. Eaich G, Pearson K, Fleming D, Sobel S, Anello C. Toxic shock syndrome and the vaginal contraceptive sponge. *JAMA*. 1986;255:216-218.
25. Allen RE. Diaphragm fitting. *Am Fam Physician*. 2004;69(1):97-100.
26. Trussell J, Strickler J, Vaughn B. Contraceptive efficacy of the diaphragm, the sponge, and the cervical cap. *Fam Plann Perspect*. 1993;25:100-105, 135.
27. Amy JJ, Tripathi V. Contraception for women: an evidence based overview. *BMJ*. 2009;339:b2895.
28. Murphy P, Hewett CM, Belew C. Contraception. In: Schuiling K, Likis F, eds. *Women's Gynecologic Health*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2017:209-260.

29. Koeniger-Donohue R. The FemCap: a non-hormonal contraceptive. *Womens Health Care*. 2006;5(4):79-91.
30. Cervical Barrier Advancement Society and Ibis Reproductive Health. Cervical cap (FemCap). Available at: http://www.cervicalbarriers.org/downloads/Images/femcap_(hand).JPG. Accessed November 30, 2017.
31. Gallo MF, Grimes DA, Schulz KF, Lopez LM. Cervical cap versus diaphragm for contraception. *Cochrane Database Syst Rev*. 2002;4:CD003551.
32. Kogan P, Wald M. Male contraception: history and development. *Urol Clin North Am*. 2014;41(1):145-161.
33. Roth MY, Amory JK. Beyond the condom: frontiers in male contraception. *Semin Reprod Med*. 2016;34(3):183-190.
34. Gallo MF, Grimes DA, Lopez LM, Schulz KF. Nonlatex versus latex male condoms for contraception. *Cochrane Database Syst Rev*. 2006;1:CD003550. doi:10.1002/14651858.CD003550.pub2.
35. Steiner M, Dominik R, Rountree R, Nanda K, Dorflinger L. Contraceptive effectiveness of a polyurethane condom and a latex condom: a randomized controlled trial. *Obstet Gynecol*. 2003;101(3):539-547.
36. Higgins J, Cooper A. Dual use of condoms and contraceptives in the US. *Sex Health*. 2012;9(1):73-80.
37. Cervical Barrier Advancement Society and Ibis Reproductive Health. Female condom. Available at: http://www.cervicalbarriers.org/downloads/Images/female_condom_(hand).JPG. Accessed November 30, 2017.
38. Witte SS, MacPhee C, Ginsburg N, Deshmukh N. Medicaid reimbursement for the female condom. *Am J Public Health*. 2017;107(10):1633-1635.
39. Beksinska M, Smit J, Joanis C, Hart C. Practice makes perfect: reduction in female condom failures and user problems with short-term experience in a randomized trial. *Contraception*. 2012;86(2):127-131.
40. Nelson AL, Cohen S, Galitsky A, et al. Women's perceptions and treatment patterns related to contraception: results of a survey of US women. *Contraception*. 2017. [Epub ahead of print]. doi:10.1016/j.contraception.2017.09.010.
41. Bougie O, Singh SS. Dalkon Shield: forgotten but not yet gone. *J Obstet Gynaecol Can*. 2016;38(8):695.
42. Cortessis VK, Barrett M, Brown Wade N, et al. Intrauterine device use and cervical cancer risk: a systematic review and meta-analysis. *Obstet Gynecol*. November 3, 2017. [Epub ahead of print]. doi:10.1097/AOG.0000000000002307.
43. Deans EI, Grimes DA. Intrauterine devices for adolescents: a systematic review. *Contraception*. 2009;79:418-423.
44. Goldthwaite LM, Shaw KA. Immediate postpartum provision of long-acting reversible contraception. *Curr Opin Obstet Gynecol*. 2015;27(6):460-464.
45. Zerden ML, Stuart GS, Charm S, Bryant A, Garrett J, Morse J. Two-week postpartum intrauterine contraception insertion: a study of feasibility, patient acceptability and short-term outcomes. *Contraception*. 2017;95(1):65-70.
46. Ramesh SS, Charm S, Kalinowski A, Liberty AL, Stuart GS. Management of intrauterine contraception in early pregnancy. *South Med J*. 2017;110(8):550-553.
47. Moschos E, Twickler DM. Intrauterine devices in early pregnancy: findings on ultrasound and clinical outcomes. *Am J Obstet Gynecol*. 2011;204(5):427.e1-427.e6.
48. Brahmi D, Steenland M, Renner R-M, Gaffield ME, Curtis KM. Pregnancy outcomes with an IUD in situ: a systematic review. *Contraception*. 2012;85(2):131-139.
49. U.S. Food and Drug Administration. Intrauterine copper contraceptive. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2013/018680s066lbl.pdf. Accessed November 30, 2017.
50. Shimoni N. Intrauterine contraceptives: a review of uses, side effects and candidates. *Semin Reprod Med*. 2010;28(2):118-125.
51. Gemzell-Danielsson K, Berger C, Lalitkumar PGL. Emergency contraception: mechanisms of action. *Contraception*. 2013;87(3):300-308.
52. Beining RM, Dennis LK, Smith EM, Dokras A. Meta-analysis of intrauterine device use and risk of endometrial cancer. *Ann Epidemiol*. 2008;18(6):492-499.
53. Stuart GS, Ramesh SS. Interval female sterilization. *Obstet Gynecol*. 2018;131:117-124.
54. American College of Obstetricians and Gynecologists. Practice Bulletin No. 133: benefits and risks of sterilization. *Obstet Gynecol*. 2013;121:392-404.
55. Shreffler KM, Greil AL, McQuillan J, Gallus KL. Reasons for tubal sterilisation, regret and depressive symptoms. *J Reprod Infant Psychol*. 2016;34(3):304-313.
56. Shreffler KM, McQuillan J, Greil AL, Johnson DR. Surgical sterilization, regret, and race: contemporary patterns. *Soc Sci Res*. 2015;50:31-45.
57. Hurskainen R, Hovi S-L, Gissler M, et al. Hysteroscopic tubal sterilization: a systematic review of the Essure system. *Fertil Steril*. 2010;94(1):16-19.
58. Câmara S, de Castro Coelho F, Freitas C, Remesso L. Essure present controversies and 5 years' learned lessons: a retrospective study with short- and long-term follow-up. *Gynecol Surg*. 2017;14(1):20.
59. Fantasia HC. Update on the Essure system for permanent birth control. *Nurs Womens Health*. 2017;21(5):401-405.
60. Walter JR, Ghobadi CW, Hayman E, Xu S. Hysteroscopic sterilization with Essure: summary of the U.S. Food and Drug Administration actions and policy implications for postmarketing surveillance. *Obstet Gynecol*. 2017;129:10-19.
61. Lippes J. *Quinacrine* sterilization (QS): *time* for *reconsideration*. *Contraception*. 2015;92(2):91-95.
62. Potts J, Pasqualotto F, Nelson D, Thomas AJ, Agarwal A. Patient characteristics associated with vasectomy reversal. *J*

Urol. 1999;161:1835-1839.

APPENDIX

16A

Fertility Awareness Methods

MARY C. BRUCKER

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Although fewer than 1% of women who use contraception in the United States employ fertility awareness methods, in some areas of the world these methods are the predominant form of birth control.[1,2] Some reasons that may account for the limited use of these strategies in the United States include the complexity of the methods, lack of available education about fertility awareness methods, and personal beliefs of consumers and providers that these methods are not effective.[3]

Formal education programs exist for both clinicians and couples. Many midwives complete certification courses for a particular type of fertility awareness method, whereas others prefer to refer interested individuals to a specialist in the area. This appendix provides general information about fertility awareness methods and discusses the five most common types.

Assumptions

Fertility awareness methods are based on regularly occurring biologic events during the menstrual cycle that should be understood by the clinician and the consumer:

1. Ovulation occurs once, approximately 14 days before menses begins. It is important to note that 14 days before menses begins is more reliable than 14 days after the last menstrual period starts because the secretory phase of the menstrual cycle is generally more regular than the follicular phase.
2. The maximum lifespan of the ovum is 12 to 24 hours.[4]
3. Sperm are viable after ejaculation for approximately 3 to 5 days.
4. The most likely time for pregnancy to occur is during the fertile window, which spans from approximately 5 days prior to ovulation until the day after ovulation, for a total of 6 days each month.[5]

Considerations for Use

Table 16A-1 lists the main criteria that a couple should consider before adopting a fertility awareness method for contraception. Although there are no devices required, a strong dedication to consistently and correctly using the fertility awareness method chosen is needed.

Table 16A-1	Considerations for Use of Fertility Awareness Methods
Commitment by both sexual partners to consistently use the method month after month	
Comfort in observing for signs of fertility, including personal touching	
Commitment to recognize and document even subtle signs of fertility	
Ability to practice abstinence for at least one month, during which time baseline signs of fertility are observed and documented before initiating the method	

Contraindications

Relative contraindications to fertility awareness methods include conditions that result in irregular menstrual cycles, including recent childbirth, onset of menarche, perimenopause, breastfeeding, frequent anovulatory cycles, and recent discontinuation of hormonal contraceptives. Women with the following conditions also may have difficulty using fertility awareness methods: persistent vaginitis or other infections that may disrupt signs of cervical changes (for some methods); not being comfortable examining cervical mucus (for some methods); intermenstrual bleeding; and inability to correctly interpret the signs of fertility.

Types of Fertility Awareness Methods

Currently, several fertility awareness methods are in common use. **Table 16A-2** summarizes the majority of these methods using available data.[5-8] Fertility awareness methods can be generally categorized as either calendar methods or ovulation methods. Both are discussed in this section. For methods that are considered proprietary, their developers usually charge and/or restrict use of their educational materials, including provider and consumer charts, to maintain integrity and accuracy. Many of the proprietary organizations also have educational programs for accreditation or certification.

Table 16A-2 **Description, Observations, Fertile Day, and Estimated Effectiveness of Selected Fertility Awareness–Based Methods**

Method	Signs or Symptoms Used by Method	Observations	Days to Avoid Unprotected Intercourse	Efficacy, First Year of Typical Use	Unintended Pregnancies, First Year of Use with Perfect Use[a]
Calendar	Day of cycle	Record cycle lengths for 6–12 cycles Identify shortest and longest cycles Shortest number of cycle days minus 18 = beginning of fertile window Longest number of cycle days minus 11 = end of fertile window	Days identified as fertile by calculations (repeat calculations every cycle) Total: Depends on cycle length variability	87%	3–5
Standard Days	Day of cycle	Track cycle days beginning with first day of menses Note days 8–19 of cycle	Days 8–19 of cycle Total: 12 days per cycle	88%	5
Ovulation	Cervical secretions	Monitor cervical secretions daily Assess quality and quantity of secretions Record data	Menses Preovulatory days following days with intercourse All days with fertile-type secretions Until 4 days past "peak" day Total: Approximately 14–16 days each cycle	80%	3–5
Two-Day	Cervical secretions	Note presence or absence of cervical secretions Record data	All days with secretions One day following days with secretions Total: Approximately 10–14 days each cycle	86%	4
Symptothermal	Cervical secretions Vaginal mucus BBT	Monitor cervical secretions daily Assess quantity and quality of secretions Take BBT daily Record observations on chart	Menses Preovulatory days following days with intercourse All days with fertile-type secretions Until 3 days of higher temperatures, or 4 days past peak Total: Approximately 14–16 days each cycle	80%	2–5

Abbreviation: BBT, basal body temperature.

[a] Number of pregnancies per 100 women per year of use with perfect use.

Based on Arevalo M, Sinai I, Jennings V. A fixed formula to define the fertile window of the menstrual cycle as the basis of a simple method of natural family planning. *Contraception.* 1999;60(6):357-360[5]; Jennings VH, Burke AE. Fertility awareness–based methods. In: Hatcher RA, Trussell J, Nelson A, Cates W Jr, Stewart F, Kowal D, eds. *Contraceptive Technology*. 20th ed. New York, NY: Ardent Media; 2011:432[6]; *Family Planning: A Global Handbook for Providers.* WHO Press; 2011. Accessible at: http://apps.who.int/iris/bitstream/10665/44028/1/9780978856373_eng.pdf. Accessed November 28, 2017[7]; Kambric R, Lamprecht V. Calendar rhythm efficacy: a review. *Adv Contracept.* 1996;12:123-128.[8]

Calendar Methods

Two calendar fertility awareness–based methods are available. The "rhythm" method, also known as the calendar method, is the oldest fertility awareness–based methods method, and was developed in the 1930s. A calendar method, the Standard Days Method, is a more recent adaptation.

Rhythm Method

This method attempts to predict the days in a menstrual cycle during which time a woman is most likely to become pregnant based on the projected time of ovulation. This interval is determined by calculations made from recording the length of the last 8 to 12 menstrual cycles. To use this method, a woman keeps a record of the date on which her menstrual cycle starts for several months to identify the longest and shortest cycles so that all possible fertile days may be projected. Although extremely popular decades ago, the rhythm method is the least used of the fertility awareness–based methods available today.

Standard Days Method

The Standard Days Method is based entirely on tracking the days of the menstrual cycle for women with regular cycles that range from 26 to 32 days.[9-12] The user notes the first day of menstruation and abstains from intercourse or uses another birth control method between days 8 and 19 after the first day that menses start during each menstrual cycle.[10]

The Standard Days Method has been promoted globally, although there is some question regarding standardization and effectiveness of the educational programs. These educational programs often advocate use of a bead-like device called CycleBeads, whose colors a woman uses to determine whether she should be considered fertile or infertile on a particular day. CycleBeads and Standard Days Method programs are proprietary, with the former device being marketed by a for-profit company. A number of apps using Standard Days Method are available.

Ovulation Methods

The three ovulation methods focus on using physical signs that indicate ovulation to identify the days of fertility and infertility for a specific cycle. These signs include changes in amount and characteristics of cervical mucus, basal body temperature (BBT), and the position and consistency of the cervix (**Figure 16A-1**).[11] Although cervical mucus is to be assessed, it is most commonly determined by wetness of the vulva. Ovulation methods that use a BBT require use of an appropriate thermometer—one of the few devices associated with fertility awareness methods.

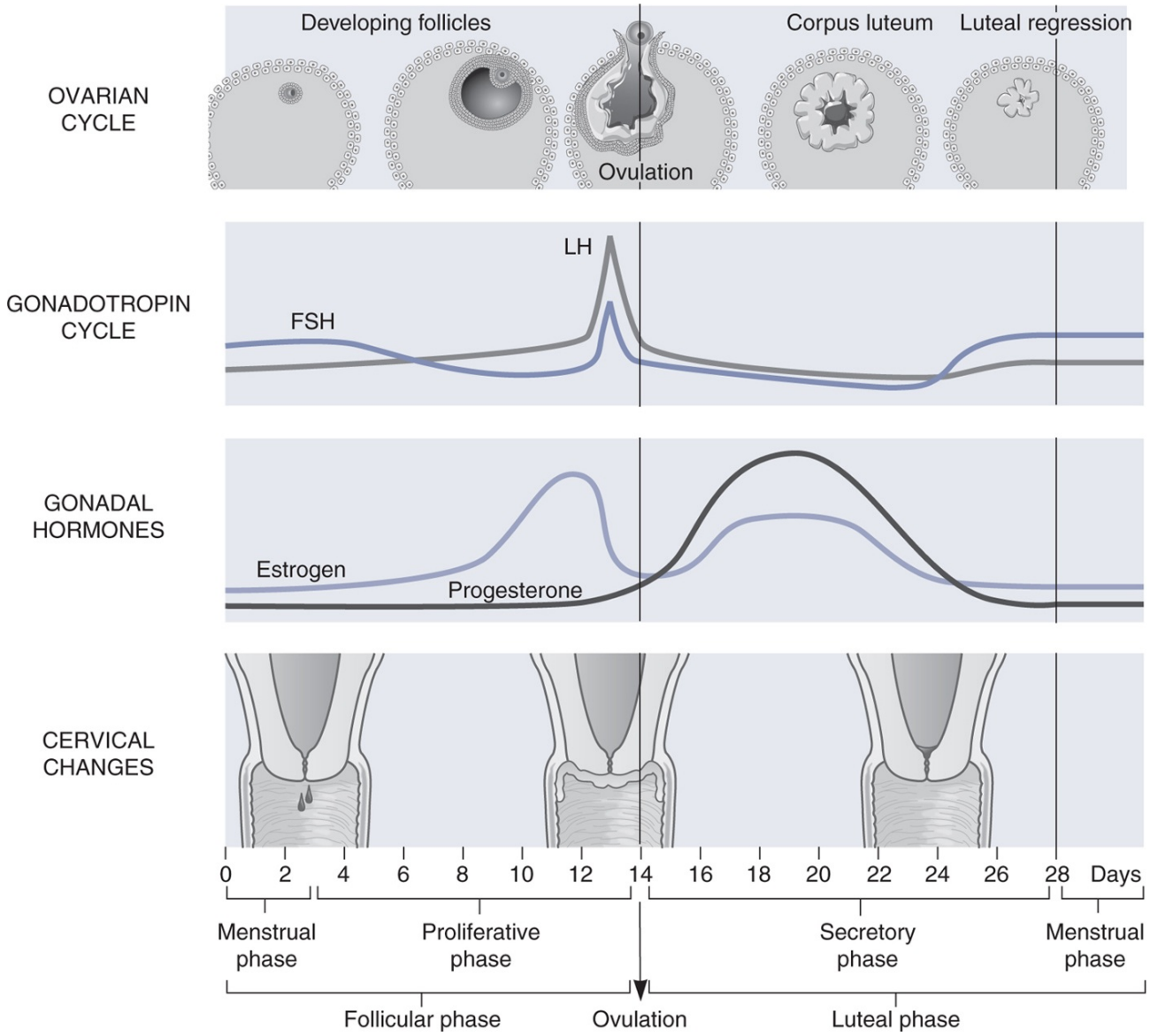


Figure 16A-1 Hormonal, follicular, temperature, secretions, and cervical changes during the menstrual cycle.

Billings Ovulation Method

The Billings Ovulation Method focuses on changes in the cervical mucus. During the infertile period in both the preovulatory phase and the postovulatory phase of the cycle, cervical mucus is absent or results in no sensation of wetness at the vulva. Fertile days are signaled by vulvar wetness or slippery cervical mucus. The woman observes the sensation of wetness at the vulva and the presence of mucus throughout the day and records her observations at the end of the day. During the entire first cycle of charting, abstinence is necessary for her to become familiar with her pattern of cervical mucus and sensations of vulvar wetness. The Billings Ovulation Method uses two patterns to describe monitoring cervical mucus (**Table 16A-3**).

Table 16A-3	**Billings Ovulation Method**

Cervical Mucus Pattern	Comments	Fertility
Basic infertile pattern (BIP)	Days immediately after menstruation Consistent dryness at the vulva	Intercourse every other day to avoid confusing seminal fluid with mucus
Peak pattern	Change in vulvar sensation from wet to slippery Under the influence of estrogen, the mucus increases in volume and becomes clear and stretchy, with an egg-white consistency (spinnbarkeit) Last day of peak is also called “peak day”	Avoid intercourse during peak and for 4 days after the peak day

Use of spermicides, lubricants, vaginal treatments, or barriers using spermicides may interfere with use of this method. Women are also advised to refrain from intercourse on days with heavy menstrual bleeding because it masks the signs of mucus associated with ovulation.

Creighton Model FertilityCare System

This fertility awareness method is a modification of the Billings Ovulation Method, yet a separate method. The Creighton Model FertilityCare System has a standardized teaching plan, is trademarked, and relies on a code system for charting.[13]

Two-Day Method

The Two-Day Method simplifies observation of cervical mucus.[14] This particular method does not require distinguishing the quality of the mucus. Instead, a woman using this method asks herself two specific questions (**Table 16A-4**). Women with irregular cycles can use this method because it relies entirely on observing one physical sign of fertility.

Similar to the Standard Days Method, the Two-Day Method is proprietary. The app associated with this method is available for a free download for a device using an iOS platform.

Table 16A-4	Two-Day Method
Question	**Fertility**
1. Did I note secretions today? 2. Did I note secretions yesterday?	Both questions must be answered. If the answer is “yes” to either one, it should be assumed the woman is fertile.

Symptothermal Method

The symptothermal method combines observation of three physical signs of ovulation: basal

body temperature, cervical mucus, and changes in the cervix itself. **Table 16A-5** lists physical signs of ovulation that suggest fertile days on the basis of ovulation.

Table 16A-5 Patterns of Ovulatory Temperature Rises

Physical Signs of Ovulation	Description	Comments
Rise in basal body temperature (BBT)	BBT rise of at least 0.4°F above the previous six daily BBT measurements	Rise is characterized as a sustained elevation or a plateau of the temperature for 3 days, or when there have been 5 days of progressive increase
Abrupt rise pattern	Rise over 1–2 days	
Saw-tooth pattern	Rise and fall over several days, with overall increase	For example, an increase of 0.4°F, decrease of 0.2°F, increase of 0.4°F, and so on
Slow rise or staircase pattern	Gradual rise over several days	
	Sharp dip in temperature followed by a rise	Occasionally occurs with ovulation but is not required
Changes in cervical mucus	Increase in volume and change to clear and elastic consistency	Same as peak pattern in Billings Ovulation Method
Cervical changes	From firm to soft and moves higher into vaginal canal	

The rise in BBT occurs as more progesterone is released by the corpus luteum, signaling that ovulation has occurred. The pattern of the rise in temperature may vary both from woman to woman and from cycle to cycle for the same woman and can be altered by inconsistent schedules or illness. Observing cervical mucus changes augments the BBT pattern to help identify fertile and infertile days. As with the Billings Ovulation Method, fertile days are characterized with elastic, thin, clear mucus.

In addition to BBT and cervical mucus observation, the third component of this method is changes in the cervix itself, which can be felt by palpating the cervix. As ovulation approaches, the cervix softens, the os dilates slightly, and the cervix is positioned higher in the vaginal canal. After ovulation, the cervix returns to being firm, closed, and lower in the vaginal canal. Detection of the cervical position is an optional sign. A woman using this method documents observation of the three parts of the method—BBT, cervical mucus, and cervical characteristics.

References

1. Sundaram A, Vaughan B, Kost K, et al. Contraceptive failure in the United States: estimates from the 2006–2010 National Survey of Family Growth. *Perspect Sex Reprod Health*. 2017;49(1):7-16.
2. Mosher WD, Jones J. Use of contraception in the United States: 1982–2008. National Center for Health Statistics. *Vital Health Stat*. 2010;23(29).
3. Han L, Taub R, Jensen JT. Cervical mucus and *contraception*: what we know and what we don't. *Contraception*. 2017;96(5):310-321.

4. Pyper C. Fertility awareness and natural family planning. *Eur J Contracept Reprod Health Care*. 1997;2:131-146.
5. Arevalo M, Sinai I, Jennings V. A fixed formula to define the fertile window of the menstrual cycle as the basis of a simple method of natural family planning. *Contraception*. 1999;60(6):357-360.
6. Jennings VH, Burke AE. Fertility awareness–based methods. In: Hatcher RA, Trussell J, Nelson A, Cates W Jr, Stewart F, Kowal D, eds. *Contraceptive Technology*. 20th ed. New York, NY: Ardent Media; 2011:432.
7. World Health Organization. *Family Planning: A Global Handbook for Providers*. WHO Press; 2011. Accessible at: http://apps.who.int/iris/bitstream/10665/44028/1/9780978856373_eng.pdf. Accessed November 28, 2017.
8. Kambric R, Lamprecht V. Calendar rhythm efficacy: a review. *Adv Contracept*. 1996;12:123-128.
9. Pallone SR, Bergus GR. Fertility awareness–based methods: another option for family planning. *J Am Board Fam Med*. 2009;22(2):147-157.
10. Arevalo M, Jennings V, Sinai I. The Standard Days method. *Contraception*. 2002;65:333-338.
11. Germano E, Jennings V. New approaches to fertility awareness–based methods: incorporating the Standard Days and TwoDay methods into practice. *J Midwifery Womens Health*. 2006;51:471-477.
12. Marston CA, Church K. Does the evidence support global promotion of the calendar-based Standard Days Method of contraception? *Contraception*. 2016;93(6):492-497.
13. Hilgers TW, Daly KD, Hilgers SK, Prebil AM. *Creighton Model Fertility Care System: A Standardized, Case Management Approach to Teaching, Book 1*. Omaha, NE: Pope Paul VI Institute Press; 2002.
14. Dunson D, Sinai I, Colombo B. The relationship between cervical secretions and the daily probabilities of pregnancy: effectiveness of the TwoDay algorithm. *Hum Reprod*. 2001;16:2278-2282.

APPENDIX

16B

Fitting Diaphragms

MARY C. BRUCKER

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Introduction

A diaphragm is a soft silicone dome that covers the cervix during intercourse. The rim has a spring inside that ensures the diaphragm fits between the posterior symphysis and the posterior vagina, covering the cervix. This appendix describes how to fit or verify the fit of a diaphragm. The diaphragm is always to be used with a spermicide. The spermicide is placed in the dome, which is designed directly to face the cervix, and spread upward to around the rim of the diaphragm.

The contraceptive mechanism of action for a diaphragm is twofold: (1) The spring in the rim forms a seal against the vaginal wall, providing a physical barrier to prevent sperm from entering the cervix, and (2) during vaginal intercourse, the diaphragm moves slightly and coats the cervix and vaginal wall with spermicide. Current types of diaphragms are listed in **Table 16B-1**. A silicon wide-seal diaphragm is illustrated in **Figure 16B-1**, and a one-size diaphragm is depicted in **Figure 16B-2**.

Table 16B-1 Types of Diaphragms

Type	Description	Indications
Wide-seal rim	Has a flexible 1½-cm-wide "skirt" extending from the inner edge of the rim	Latex-free diaphragm composed of silicon. The small skirt is designed to hold gel in place and improve the seal. May have one of two springs.
	Arcing spring	Strongest type of rim. May be used by women who have cystocele, rectocele, uterine prolapse, retroverted uterus, or an anteverted or retroverted cervix.
	Omniflex	Has a distortion-free spring that provides arc no matter where the rim is compressed. This type of rim is proposed to provide increased suction for added protection.

		The wide-seal rim omniflex replaced the “all-flex” diaphragm, which was once the most popular diaphragm but was discontinued by the manufacturer in approximately 2014. However, it folds on only two axes.
Silicon single size	Purple diaphragm with rim dimples for gripping during insertion and a finger dome for ease of removal	Marketed with the “Caya” brand name, this silicon diaphragm is designed to fit most women. It is a single size and no measuring is required. Like all diaphragms, it is available by prescription only. Some providers suggest that proper placement should be verified by a clinician because the diaphragm is not marketed as fitting all women.

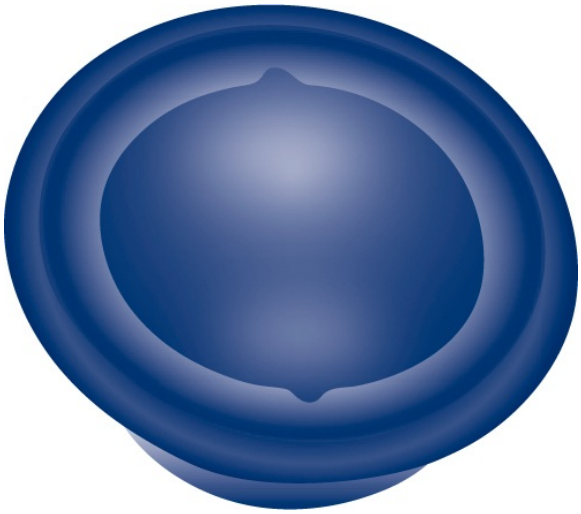


Figure 16B-1 Wide-seal diaphragm.

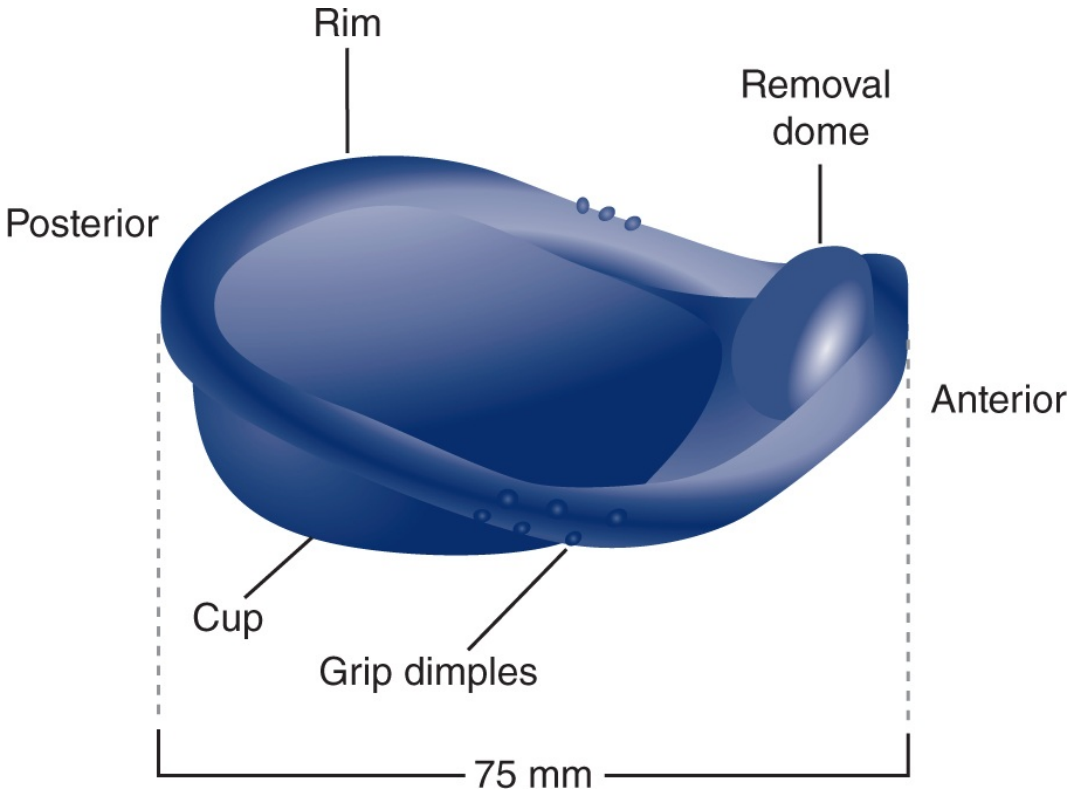


Figure 16B-2 Single-size contoured diaphragm.

Contraindications (U.S. MEC for Contraceptive Use Category 3 or 4)[1]

- History of toxic shock syndrome
- High risk for human immunodeficiency virus (HIV) infection
- HIV infection or acquired immunodeficiency syndrome (AIDS)

Procedure for Fitting for Diaphragms That Have Different Sizes

Equipment and Supplies

- A complete set of fitting rings or fitting diaphragms, from 50 mm to 95 mm
- Nonsterile gloves
- Lubricant

Preparation

1. Encourage the woman to have an empty bladder prior to examination.
2. Gather all needed equipment and arrange it so it is in easy reach.
3. Perform a pelvic examination to rule out any infections or abnormalities.
4. The initial choice of a fitting diaphragm or fitting ring usually begins with a size in the center of the probable range for the woman's parity. Generally a nulliparous woman will be fitted with size 65, 70, or 75, and a multiparous woman with size 75, 80, or 85.

Procedure[2]

1. Insert a gloved index and middle finger into the vagina until the middle finger reaches the posterior wall of the vagina, to determine the distance between the posterior symphysis and the posterior vagina behind the cervix.
2. Mark the point at which the index or middle finger touches the posterior inferior sympthysis pubis.
3. Remove the fingers and place a diaphragm rim on the tip of the middle finger. The opposite rim of the correct size of diaphragm should lie approximately in front of the place on the examiner's finger that touched the posterior inferior symphysis pubis.
4. Lubricate or moisten the diaphragm or fitting ring, compress the sides together with the fingers and thumb of one hand, and introduce it into the vagina (**Figure 16B-3**). Be certain to direct the diaphragm or fitting ring downward and inward, thereby applying pressure against the posterior vaginal wall and avoiding the more sensitive anterior structures.
5. Once the diaphragm is inserted, check its placement with a gloved finger. It is the

correct size if:

a. The rim is behind the cervix in the posterior fornix.
b. The circumference rests easily against the lateral vaginal walls.
c. A fingertip can be inserted between the diaphragm and the posterior surface of the symphysis pubis.
d. The cervix is covered.

6. The diaphragm is *too small* if:
   a. There is more than enough space for the flat portion of a fingertip to be inserted between the rim of the diaphragm and the posterior surface of the sympthysis pubis.
   b. It moves about freely in the vagina.
   c. It dislodges when the woman coughs or bears down.
7. The diaphragm is *too large* if:
   a. It fits tightly against the symphysis pubis.
   b. The rim buckles forward against the lateral vaginal walls.
   c. It dislodges or protrudes out the vagina when the woman performs a Valsalva maneuver.
   d. The woman feels discomfort when the diaphragm is in place.
8. Many midwives will verify the appropriate size by inserting diaphragms of successive size until the correct size is ascertained and then inserting one size larger for confirmation, although this last step is not required.
9. Return demonstration:
   a. Have the woman wash her hands. Remind her in real life she should verify the integrity of the device before inserting it.
   b. Have the woman assume a comfortable position that will allow her to insert two fingers into her vagina and reach her cervix. Optional positions include the following:
      i. Lying supine with head elevated and knees flexed
      ii. Standing with one leg on a stool, bending forward
   c. As she inserts her fingers into her vagina, verbally guide the woman to locate and feel the symphysis pubis and then her cervix.
   d. Have the woman hold the lightly lubricated diaphragm in one hand, dome side down. Then have her compress the sides of the diaphragm between her thumb and fingers, with her hand on top of the diaphragm, and spread her labia with her other hand and introduce the end of the folded diaphragm into her vagina in a downward and backward direction.
   e. Once the diaphragm has been introduced into the vagina, the woman can use the hand that has been separating the labia to complete insertion until the rim is under the posterior symphysis pubis. Instruct her to then use a finger to tuck the rim upward, behind the pubis.
   f. Have the woman check the placement, feeling the rim behind the pubis and the cervix covered by the diaphragm.
   g. The midwife may ask the woman to walk around the room to note any sensation or discomfort. When correctly fitted, the diaphragm will not be felt.

h. Have the woman lie down in modified lithotomy position; insert a gloved finger into the vagina and check that the diaphragm is fitted correctly.

i. To remove the diaphragm, have the woman resume the position she chose for insertion. She inserts a finger into her vagina, bears down, grasps the rim behind the symphysis pubis with two fingers or thumb and index finger, and gently slides the diaphragm down and out.

j. Have her repeat this practice insertion and removal until she is confident she can insert the diaphragm, check its correct placement, and remove it.

k. Demonstrate use of the spermicide. Squeeze 1 to 2 teaspoons of the gel from the tube into the dome, spreading it evenly over the inside and around the rim of the diaphragm.

For a woman who is using a single-size diaphragm, some providers suggest that the return demonstration as outlined here be performed to verify correct placement.

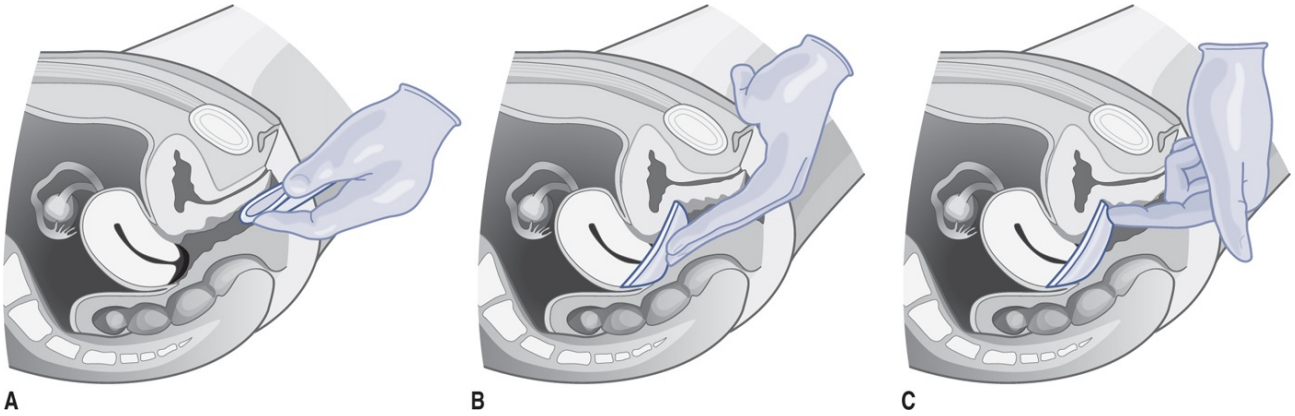


Figure 16B-3 Diaphragm fitting. **A.** Insert the folded diaphragm into the posterior vaginal fornix. **B.** Place the anterior wall behind the symphysis pubis. **C.** Check placement of the diaphragm posteriorly, anteriorly, and laterally to confirm it fits without undue pressure or too much space between the rim and the vaginal walls.

Health Education: Key Points

1. The diaphragm ensures the best protection when inserted correctly and used consistently with a spermicide.
2. Always wash hands and check for integrity of the diaphragm before using it. Even pinholes in the device invalidate this barrier method.
3. The diaphragm can be inserted up to a maximum of 6 hours prior to intercourse, although evidence-based data on timing are lacking. Some clinicians suggest an additional applicator of spermicide be inserted if more than 1–3 hours have passed after the initial insertion.
4. It is recommended that the diaphragm be left in place a minimum of 6 hours after intercourse, although there are no studies that document the optimal time needed for full contraceptive protection. Additional spermicide should be added without removing the

diaphragm before another act of coitus.

5. Do not leave the diaphragm in situ for more than 24 hours after intercourse, secondary to the theoretical risk of toxic shock syndrome.
6. When a diaphragm fits correctly, the woman will not feel it inside her vagina.
7. Symptoms of toxic shock syndrome—sudden high fever, muscle aches, chills, and malaise—should be reported to the midwife immediately.

References

1. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use 2016. *MMWR*. 2016;65(3):1-106.
2. Allen RE. Diaphragm fitting. *Am Fam Physician*. 2004;69(1):97-100.

APPENDIX

16C

Intrauterine Device Insertion and Removal

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Introduction

Intrauterine devices are long-acting reversible contraceptive (LARC) agents that require little, if any action, by the woman after they are inserted, resulting in a very effective contraceptive method. For the purposes of this appendix, the terms "intrauterine device" and "IUD" will be used throughout. Five different brands of IUDs are currently available in the United States, one of which is wrapped in copper (copper IUD) and four of which are impregnated with levonorgestrel (LNG-IUD). More information about the effectiveness and use of these devices is found in the *Family Planning*, *Nonhormonal Contraception*, and *Hormonal Contraception* chapters.

This appendix can be used as a guide for insertion of any of the current IUDs. In addition to its use as a contraceptive, the LNG-IUD can be used for the treatment of women with menorrhagia; more information about this non-contraceptive usage is found in the *Menstrual Cycle Abnormalities* chapter.

Health Counseling for Women Considering an Intrauterine Device

In advance of IUD insertion, the clinician should examine personal biases toward contraceptive options, as this is essential to providing evidence-based information that is free of judgment.[1] A thorough reproductive life plan and health history, health education, contraindications, risks, contraceptive and noncontraceptive benefits, side effects and adverse effects are reviewed as part of a shared decision-making process.[1-7] These topics are presented briefly in this appendix. The reader is referred to the chapter on *Hormonal*

Contraceptives, resources and references for a more detailed description of health counseling prior to IUD insertion.

A written consent form granting permission to the clinician for IUD insertion, signed by the woman either on paper or by electronic means, is part of the shared decision making and legal informed consent. Health education should include the costs involved with IUD insertion. Although the initial cost may be higher than for other contraceptive methods, the IUD becomes cost neutral after three years of continuous use and has the added cost savings associated with reduction in unintended pregnancies.[6,7] For women without insurance coverage, some financial support for IUD use may be available directly through the pharmaceutical companies or their associated philanthropic programs.

Contraindications for Intrauterine Device Insertion

The initial step prior to consideration of an intrauterine device is to determine if the woman has any contraindications as listed in **Table 16-4**. In brief, absolute or relative contraindications for IUD use include: pregnancy, unexplained vaginal bleeding, solid organ transplantation, cervical cancer awaiting treatment, distorted uterine cavity, endometrial cancer, gestational trophoblastic disease, and infections such as pelvic tuberculosis, pelvic inflammatory disease (PID), post-septic abortion, sexually transmitted infections (STIs) (e.g., chlamydia, syphilis, gonorrhea), purulent cervicitis, or high risk for STIs.[3,4]

Contraindications specifically for use of the copper-containing IUD include Wilson's disease, allergy to copper, or systemic lupus erythematosus with severe thrombocyteopenia.[3,4]

Contraindications specifically for use of the LNG-IUD are current breast cancer, history of breast cancer, current heart disease, history of heart disease, systemic lupus erythematosus, and liver disease such as cirrhosis.[3,4] The LNG-IUD is not recommended for use as an emergency contraceptive, perhaps because of ethical issues in conducting a rigorous study or lack of compelling need because the copper IUD has well recognized spermicidal effects, and has been on the market for approximately four decades with a body of research demonstrating effectiveness as an emergency contraceptive.

Risks and Benefits Associated with Intrauterine Devices

The benefits of IUDs include ease of use in that these are non-coital dependent, long-acting reversible method, and a limited number of side effects. IUDs are associated with a decreased risk of cervical cancer. The copper-containing IUD is associated with menstrual regularity and possibly a decreased risk of endometrial cancer. The LNG-IUD is associated with amenorrhea, reduction of symptoms of dysmenorrhea and heavy menstrual bleeding, symptoms of endometriosis, and a reduced risk for PID.

Side Effects and Adverse Effects Associated with Intrauterine Devices

Many myths and misconceptions exist regarding IUDs that can be addressed during pre-insertion counseling.[5] IUDs are not associated with an increased risk for infertility, ectopic

pregnancy, or infections such as PID or HIV. Side effects are primarily changed vaginal bleeding patterns. The copper-containing IUD is associated with increased menstrual flow and menstrual cramping. In contrast, the LNG-IUD gradually induces amenorrhea. Adverse effects include a vasovagal response and uterine perforation, which are associated with insertion.

Vasovagal Response

During IUD insertion, some women may experience a vasovagal reaction secondary to cervical manipulation. Common symptoms include syncope, hypotension, bradycardia, tunnel vision, vertigo, dyspnea, nausea, and diaphoresis. This response usually resolves spontaneously if the clinician stops any manipulation and waits for the symptoms to resolve. Usually insertion can be resumed without incident. It is rare that an IUD cannot be inserted due to a vasovagal reaction.

Uterine Perforation

Perforation of the uterus at the time of IUD insertion is a rare event with an incidence estimated at 0.4 per 1000 insertions. If uterine perforation occurs during placement of the IUD, the clinician may find that during fundal placement, the IUD continues to travel beyond the original uterine depth and the threads begin to travel upward. Women most at risk for perforation are those who are breastfeeding, who have recently given birth, or both.[8] When perforation is suspected, the IUD should be removed, physician consultation obtained, and the woman should be evaluated closely. Most perforations are small and require little treatment.[9],[10] Any woman demonstrating symptoms of bleeding or shock post insertion should be transferred to an appropriate care facility for prompt treatment.

Choice of an Intrauterine Device

Several IUDs are currently available in the United States. Both the copper and levonorgestrel (LNG) IUDs provide highly effective contraception, can be identified on ultrasound if needed, and are easily reversible. Nevertheless, certain differences between them must be taken into consideration.

The copper IUD (ParaGard) is a nonhormonal option and has been available for decades in the United States. This IUD is associated with irregular bleeding, dysmenorrhea, and heavier menses, which on rare occasions may lead to anemia. Although the copper IUD is currently approved by the U.S. Food and Drug Administration (FDA) as having 10 years of effectiveness, emerging research suggests a longer duration of effectiveness exists.

Four LNG-IUDs (Mirena, Liletta, Skyla, and Kyleena) exist; they are described in more detail in the *Hormonal Contraception* chapter. While all of these devices contain the same hormone (levonorgestrel), they differ in size, duration of effectiveness, and dose of hormone. Additionally, Mirena and Liletta each have unique FDA approvals: Mirena is approved for the treatment of individuals with heavy menstrual bleeding who also desire an IUD, whereas Liletta is approved specifically for use in nulliparous women. When considering side effects

and the therapeutic benefit of amenorrhea conferred by the presence of LNG in these devices, it may be helpful to consider the person's baseline menstrual flow. Those who report light to moderate bleeding may be more likely to experience amenorrhea within the first year of use.[11]

Timing of the Intrauterine Device Insertion

IUDs may be inserted at any time if the clinician is reasonably sure that the woman is not pregnant, including postpartum (including after cesarean birth) and post abortion (either spontaneous or induced).[3] In both cases, sepsis precludes use of the method until resolution of infection. In an effort to reduce the number of unplanned pregnancies, insertion in the immediate postpartum period is becoming more common in the United States, despite an increased rate of expulsion associated with this timing.[12,13] Postabortion insertion continues to be a common practice and has been found to be safe and effective.[3] The copper IUD also can be used as a type of emergency contraception; its use for that indication is discussed in the *Family Planning* chapter.

In years past, IUDs for contraceptive use were almost exclusively inserted during menses to assure that the woman was not pregnant at the time of insertion. This practice was also based on the belief that insertion would be easier during menses. A theory existed that the cervix might be slightly open and softer at this time. In reality, little evidence exists to support this timing.[14] Current guidelines state that insertion can occur at any time for those women without preexisting contraindications to the method, assuming there is reliable evidence that the woman is not currently pregnant.

For a woman who already is using a hormonal contraceptive method such as combined oral contraceptives, the IUD can be inserted at any time during the cycle but should be in place 7 days before the pills are discontinued when an LNG-IUD is inserted and 5 days before the pills are discontinued if a copper IUD is used.

Screening for Sexually Transmitted Infections

According to national screening guidelines, screening for STIs should be conducted on a regular basis.[15] However, requiring that negative results of recent STI screening be available prior to IUD insertion imposes an unnecessary barrier to access for this method. Studies have found that the rate of PID is no different in women who screened positive for chlamydia infection or gonorrhea at the time of insertion versus those for whom IUD insertion was deferred.[16] An exception is those women who have active disease or signs and symptoms of purulent cervicitis, chlamydia, or gonorrhea infection; in these women, insertion should be deferred until the infection is resolved.[3,4,17] Screening for human papillomavirus infection or cervical cancer is not necessary and, therefore, is not required.[18]

Day of Intrauterine Device Insertion

The insertion procedures for copper IUDs and LNG-containing IUDs have minor differences based on the configuration of both the inserter and the device. Insertion in the immediate postpartum period also requires a different set of steps. In all cases, the intention is to place the IUD in the fundus of the uterus. If the IUD placement is not fundal, there is a higher likelihood of unintended pregnancy as well as expulsion of the IUD.

Several training programs are available in which clinicians can learn how to insert an IUD. Most of these programs are sponsored by the manufacturers of the devices, and these vendors may restrict use of their products until such education is completed. Multiple resources for IUD insertion procedures exist online, including video recordings that help the clinician review the process. In addition, the FDA-approved prescribing information and package label for each device include insertion instructions and images. The following steps for insertion are general guidelines and cannot replace hands-on experiences.

Premedication

It is common practice to administer a prophylactic nonsteroidal anti-inflammatory agent such as ibuprofen to women prior to insertion of an IUD. These drugs effectively treat the pain associated with IUD use, such as copper IUD–associated dysmenorrhea, but there is no evidence suggesting that they diminish discomfort at the time of IUD insertion.[3,19] Anesthesia such as topical lidocaine may or may not decrease pain, but published studies on this topic lack enough rigor to draw any conclusion.[3,19] By comparison, lidocaine, when delivered as a paracervical nerve block, may reduce pain associated with tenaculum placement or IUD insertion.[3,19] Only limited evidence supports the use of nitrous oxide as a possibly effective analgesia option for women during an IUD insertion; however, high satisfaction rates among users were noted.[3,19]

Using misoprostol (Cytotec) to soften the cervix does not significantly improve ease of IUD insertion and may even increase pain.[3,19] Thus, misoprostol is not recommended for regular use as part of the insertion procedure. However, there may be some benefit to using this agent when attempting a second insertion after a previous failed attempt.[20]

The use of antimicrobial agents has been shown to have limited effectiveness in reducing infection, including PID and endocarditis, despite a theoretical risk of these complications.[16,21] Although the overall risk of IUD-related infection is low, one systematic review found a small, but significant, reduction in infections among women receiving such treatment; the authors cautioned that the number of women in the studies was small and routine use was likely not to be cost-effective.[22]

Required Equipment for Intrauterine Device Insertion

General equipment includes the following items that are clean, albeit not necessarily sterile:

- Clean, nonsterile gloves for a bimanual examination.

- Lubricant for a bimanual pelvic examination.
- Adequate lighting for visualization.
- Speculum of appropriate shape and size.
- Scissors to cut the IUD threads after insertion.
- Antiseptic solution (povidone–iodine or 4% chlorhexidine gluconate).
- Cotton-tipped swab (drumstick swab) and/or a cotton ball or 4 × 4 gauze pads. These may be used to apply the antiseptic to the cervix. The swab may also be inserted to the level of the cervix against the uterine sound and then removed simultaneously for easy visualization of the measurement.
- Ring forceps to hold the 4 × 4 gauze pads if utilized, to remove the IUD if needed, or to remove the cut strings from the vaginal vault.
- A sanitary pad for the woman in the event of post-procedure spotting or bleeding.

A *sterile field* includes the following sterile equipment:

- Uterine sound to verify the size of the uterine cavity and the appropriateness of a specific size of IUD.
- Tenaculum (single tooth) to grasp the cervix, straighten the cervical canal, and stabilize the uterus during insertion. Some evidence suggests that an Allis clamp may be equally effective and reduce the risk of tenaculum-related bleeding.[23]
- Sterile gloves of appropriate size.

Optional Equipment for Intrauterine Device Insertion

- Os finder or other such dilator
- Monsel's solution or silver nitrate sticks to control tenaculum-related bleeding
- Supplies for administering a paracervical block, including trumpet, needle, syringe, and anesthesia
- 1% lidocaine, to be injected 3–5 minutes prior to IUD insertion
- 10-cc syringe with 22-gauge needle with extender or a spinal needle for the lidocaine

The IUD of choice may be placed on the field or handed to the clinician by an assistant. At some sites, a secondary IUD may be available should contamination occur with the first one. At other sites, consumers must purchase their own IUD. In those situations, it is likely that only one device will be available.

Key Points for Insertion of Any Intrauterine Device

1. Optimally the clinician should have another individual to assist with the procedure.
2. A bimanual pelvic examination is performed to ascertain the position of the uterus, rule out any abnormalities, and determine the position of the uterus. Note that a woman immediately postpartum or breastfeeding who has a softer uterus may have a higher risk

of perforation, but these conditions are not contraindications to the procedure.

3. The woman should have signed legal consent for documentation and verification of understanding of the procedure.
4. The woman is assisted into a physical position of comfort for the procedure. The examiner establishes adequate light for visualization of the vagina and cervix.
5. The woman is reassured that the procedure can be stopped at any time should she make this request.
6. The insertion is begun using sterile technique. Insert the speculum so that the cervical os is visualized.
7. The cervix and vagina is cleansed with the antiseptic. Cervical anesthesia administered if indicated.
8. Apply the tenaculum to the anterior lip of the cervix, with the teeth placed parallel to the plane of the speculum blades. If the uterus is retroverted, it may be more effective to place the tenaculum on the posterior lip. Gentle traction is exerted toward the clinician to straighten the cervical canal in alignment with the uterine cavity. Usually the clinician uses the nondominant hand to hold the tenaculum.
9. While performing these actions, the clinician should observe for a vasovagal response caused by manipulation of the cervix. If the woman feels faint or nauseated, stop the procedure and wait. Usually the second attempt at insertion is successful, assuming several minutes have elapsed.
10. While the clinician's nondominant hand holds the tenaculum in place, the dominant hand gently slides the uterine sound into the uterus to measure the depth of the uterine cavity (**Figure 16C-1**). Caution must be taken not to aggressively force the sound. Most of the currently available IUDs are designed for a uterine cavity that sounds between 6 and 9 cm, while the minimal sounding depth for Liletta is lower, at 5.5 cm. Skyla and Kyleena are physically smaller and may be appropriate for the uterine cavities of nulliparous women which are typically smaller than the uterine cavity in a multiparous woman. The depth of the uterus in centimeters is noted. If the sound does not confirm a depth within the required range, the insertion procedure should be aborted and an alternative method of contraception discussed.
11. At this point, the steps for the insertion vary based on the device. However, for all IUDs, the clinician maintains sterile technique. The device is not opened until it has been deemed appropriate for immediate insertion.

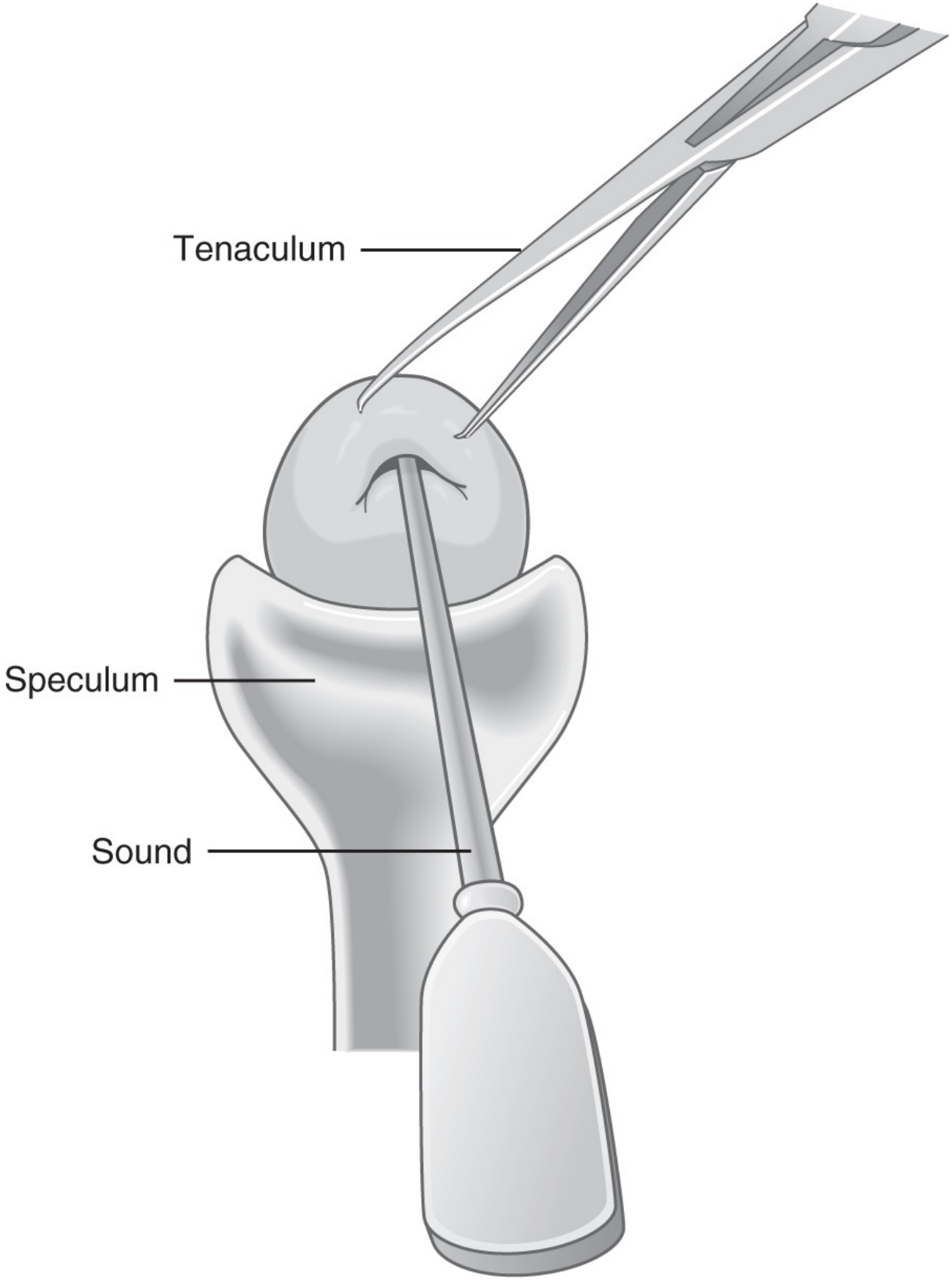


Figure 16C-1 Cervix with tenaculum and uterine sound.

Insertion of an LNG-IUD

The Mirena, Skyla, Kyleena, and Liletta IUDs are inserted in a similar manner. However, the anatomy of the Liletta device differs, particularly the loading mechanism and slider.

1. The IUD package is opened, usually by the assistant, and the clinician reaches for the device in a sterile manner. Alternatively, the entire package can be opened by the clinician and placed on the sterile field, who then dons sterile gloves and proceeds with the insertion. **Figure 16C-2** provides an illustration of the device in the packaging and the anatomy of the various LNG devices.
2. The threads of the Mirena, Skyla, and Kyleena are self-contained within the device. The threads of the Liletta are secured on the handle of the inserter and are ultimately released from the groove so they can hang freely.
3. Placement of the clinician's thumb or forefinger on the slider will stabilize the inserter and should be maintained until the insertion is complete. The slider should *not* be moved downward at this time, as this action may prematurely release the threads of the IUD. This is particularly relevant to the Mirena, Skyla, and Kyleena IUDs: Once the slider is moved below the mark, the IUD cannot be reloaded. The Liletta, however, may be reloaded.
4. The arms of the device should be in a horizontal position. If they are not, they can be realigned on a flat sterile area, such as the inside of the sterile package (**Figure 16C-3**).
5. To load the strings into the Mirena, Skyla, and Kyleena, push the slider forward until a "click" is heard or felt, indicating it is in place, while maintaining forward pressure on the slider. For Liletta, while continuing forward pressure on the blue slider, pull both strings straight back toward the clinician until a hard stop is achieved. This two-handed action retracts the IUD arms in the tubing (**Figure 16C-4**). The Liletta may be reloaded by releasing the thread from the cleft and pulling back on the blue slider until the groove becomes aligned with the green. By returning the blue slider to the forward position, the loading steps can be repeated. Do not load the IUD too far in advance of insertion so as to allow the device to retain its memory and allow optimal expansion of the arms to occur; prolonged time in the inserter may cause the device to lose structural memory.
6. Based on the uterine size determined by the sound, the flange on the insertion device is set at that number by sliding the flange over the marked increments on the IUD insertion tube. Sterile gloves must be used when touching the flange. The Liletta packaging has a built-in tray notch that enables the clinician to adjust the flange without touching it (**Figure 16C-5**).
7. The clinician's nondominant hand exerts gentle traction on the tenaculum or Allis clamp. Simultaneously, the clinician applies continued pressure to the slider on the IUD handle. These actions place the insertion tubing into the vagina at the level of the external cervical os.
8. Gently advance until the flange is approximately 1.5 to 2 cm from the external cervical os. The IUD will be into the uterus but should not be in the fundus at this time (**Figure 16C-6**).
9. For Mirena, Skyla, and Kyleena, the slider on the handle is pulled back toward the

clinician until the level of the raised mark on the insertion handle is reached. For Liletta, pull the blue slider back until resistance is reached; a common thumb recess is created once merged with the green slider. At that time, the IUD arms are expelled from the insertion tubing (**Figure 16C-7**). The clinician must wait 10 to 15 seconds to allow the arms to open completely.

10. Advance the insertion tubing until the flange is at the external cervical os. At that point, the IUD is in the uterine fundus (**Figure 16C-8**).
11. Move the slider(s) toward the clinician to release the IUD. A green indicator in the thread cleft of Liletta will confirm that the threads have been released (**Figure 16C-9**).
12. Gently remove the IUD handle and insertion tubing from the uterus and cervix and appropriately dispose of them.
13. The threads will remain in place.

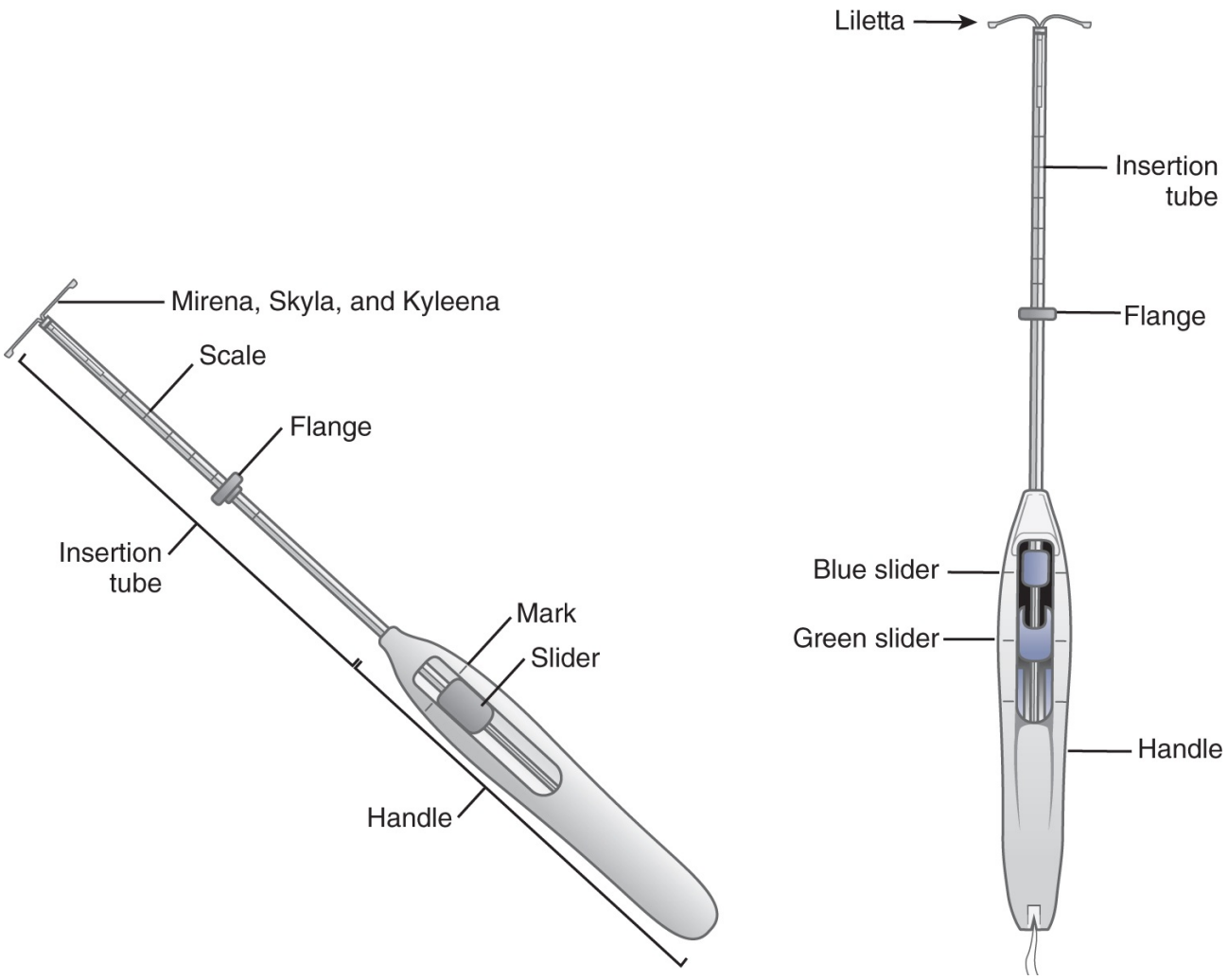


Figure 16C-2 Anatomy of LNG-IUDs: Mirena, Skyla, Kyleena, and Liletta.

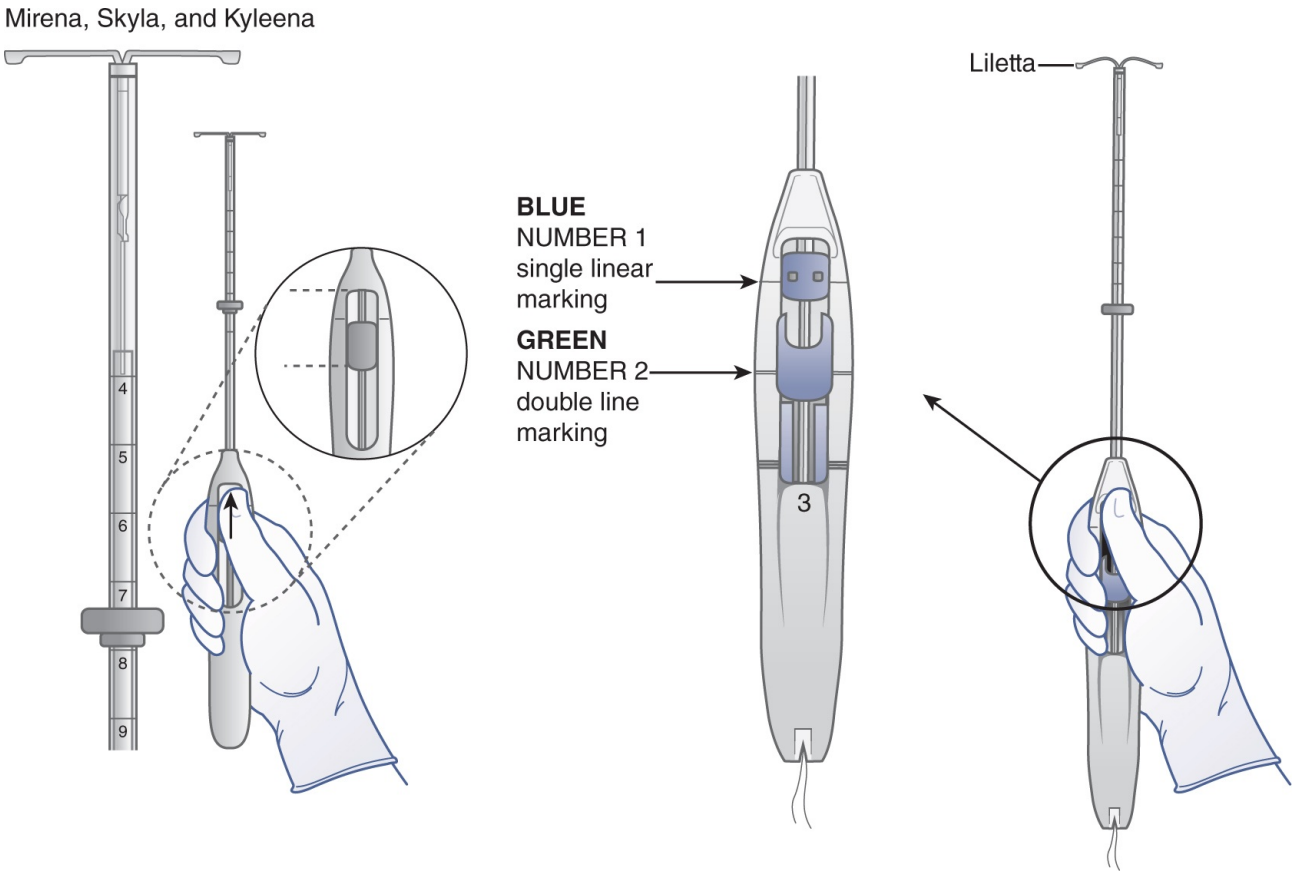


Figure 16C-3 Stabilization of slider and verification of position of IUD.

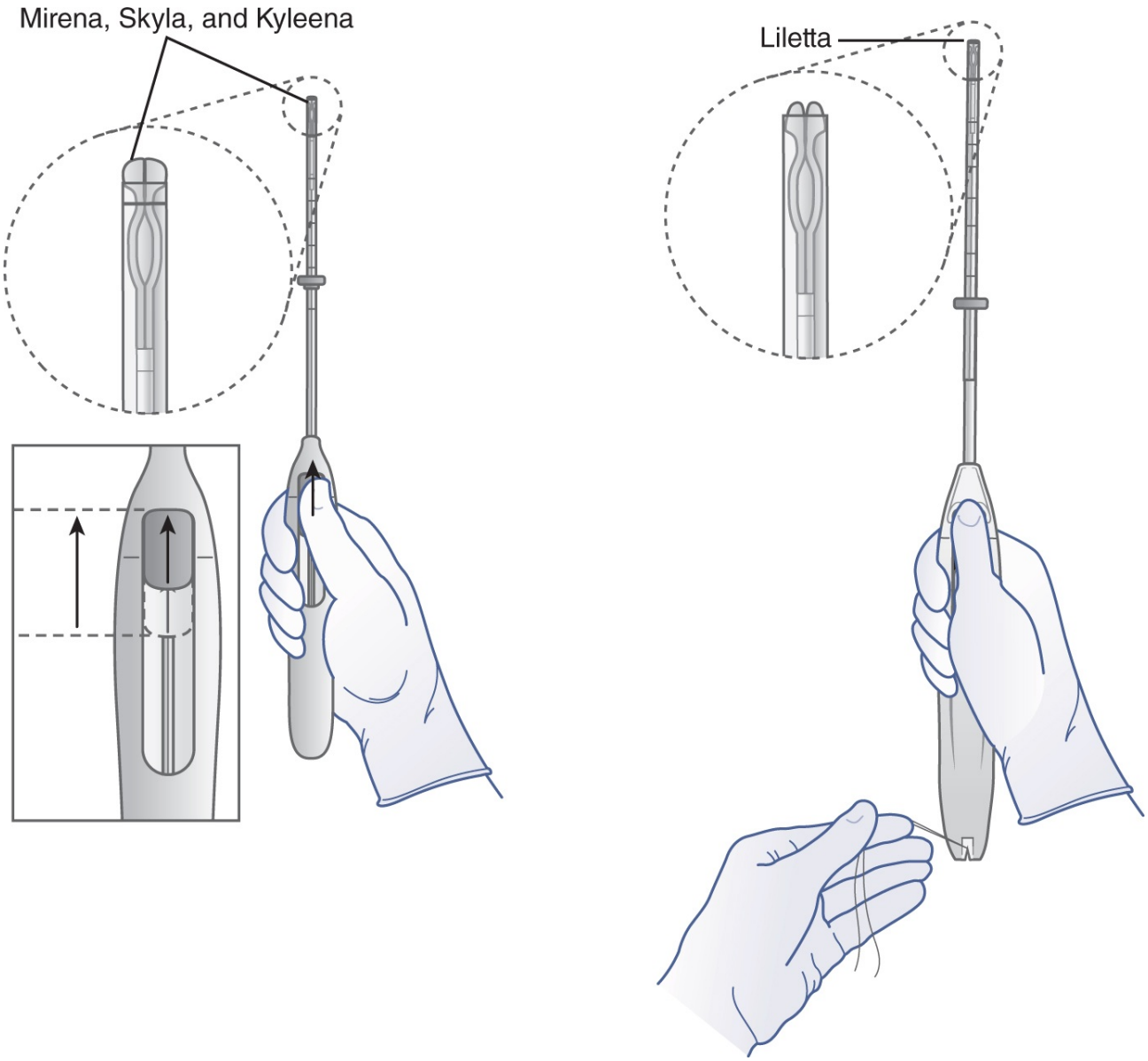


Figure 16C-4 Retraction of LNG-IUD into tubing.

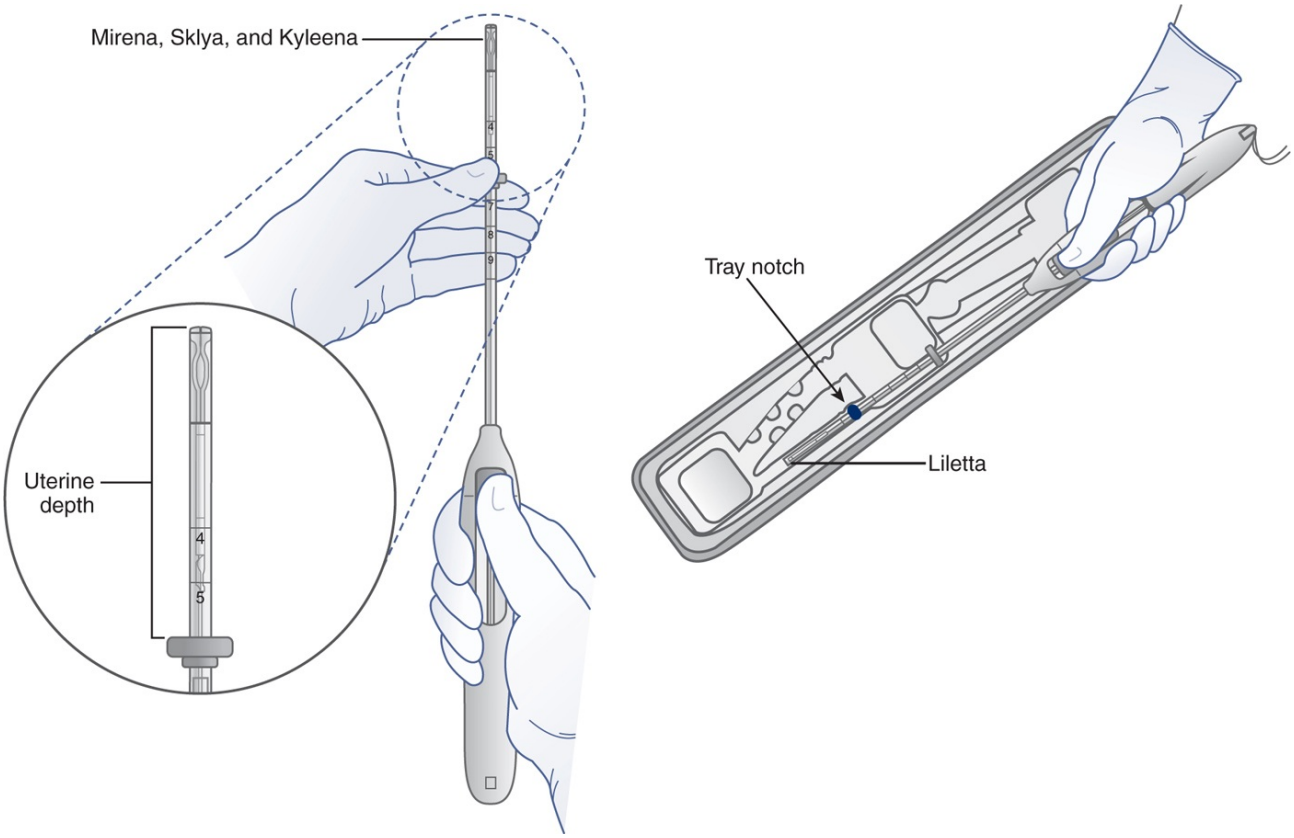


Figure 16C-5 LNG-IUD flange moved to uterine depth.

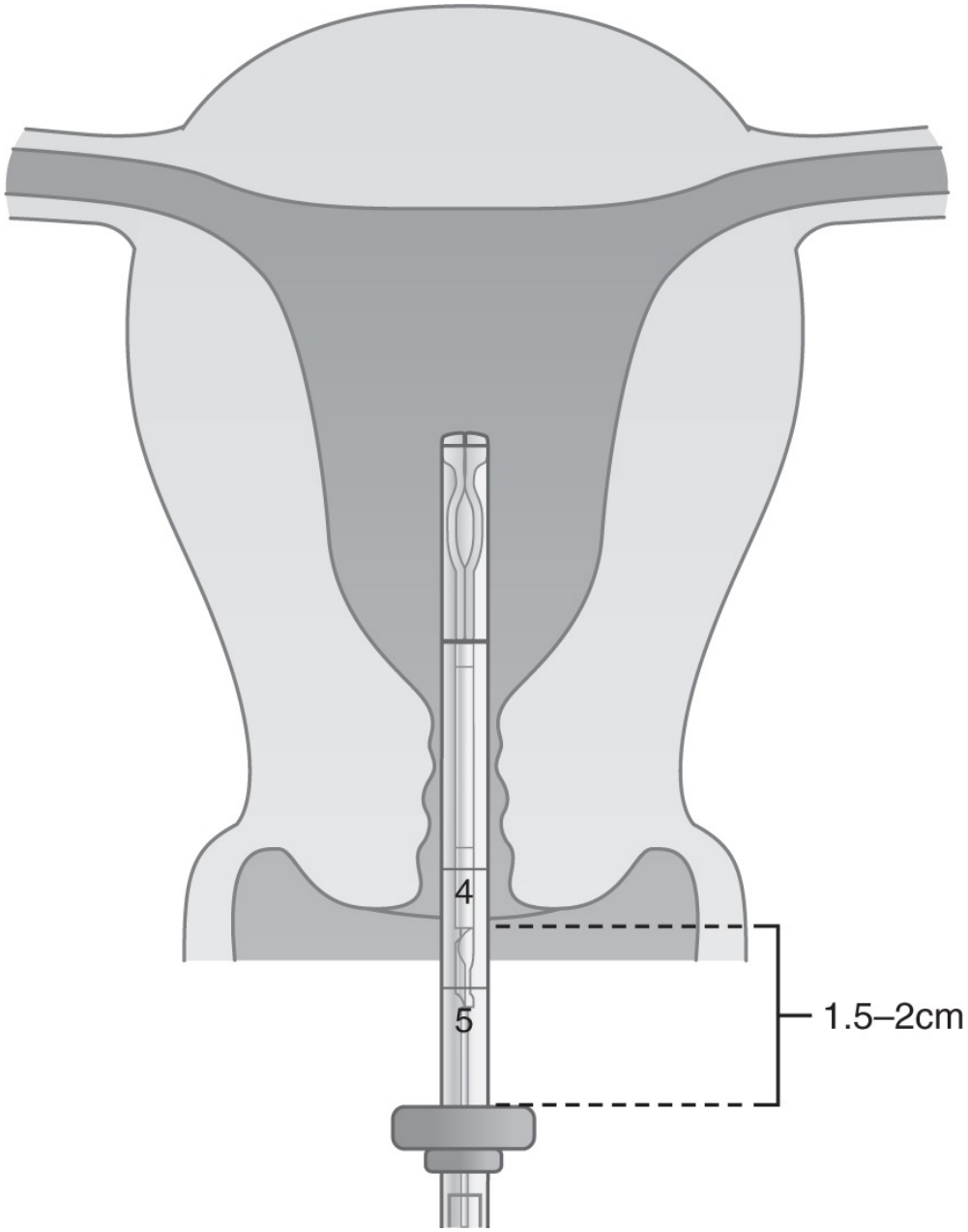


Figure 16C-6 LNG-IUD inserter guided through the cervix.

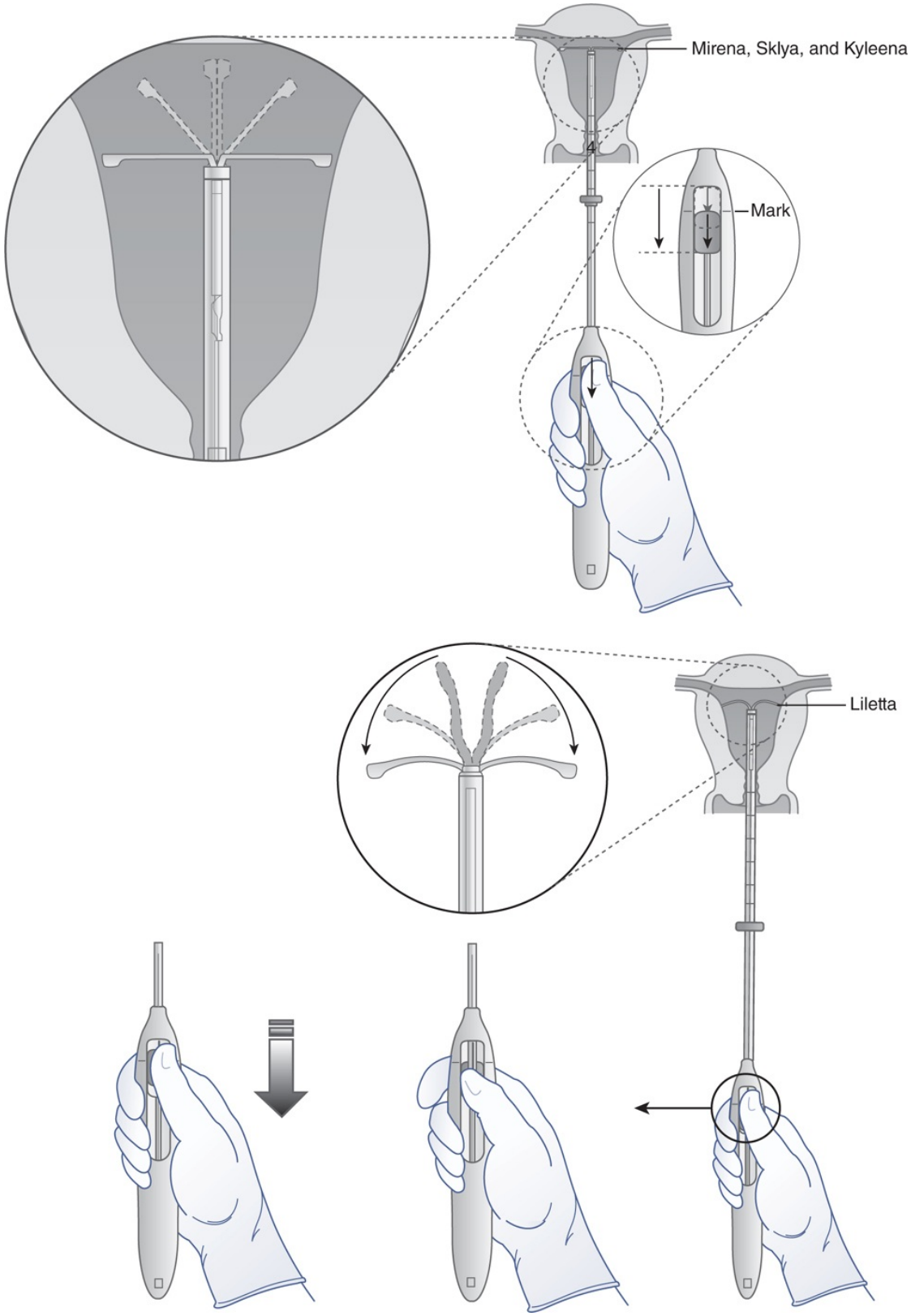


Figure 16C-7 Opening of the LNG-IUD arms via moving the slider to the preset mark.

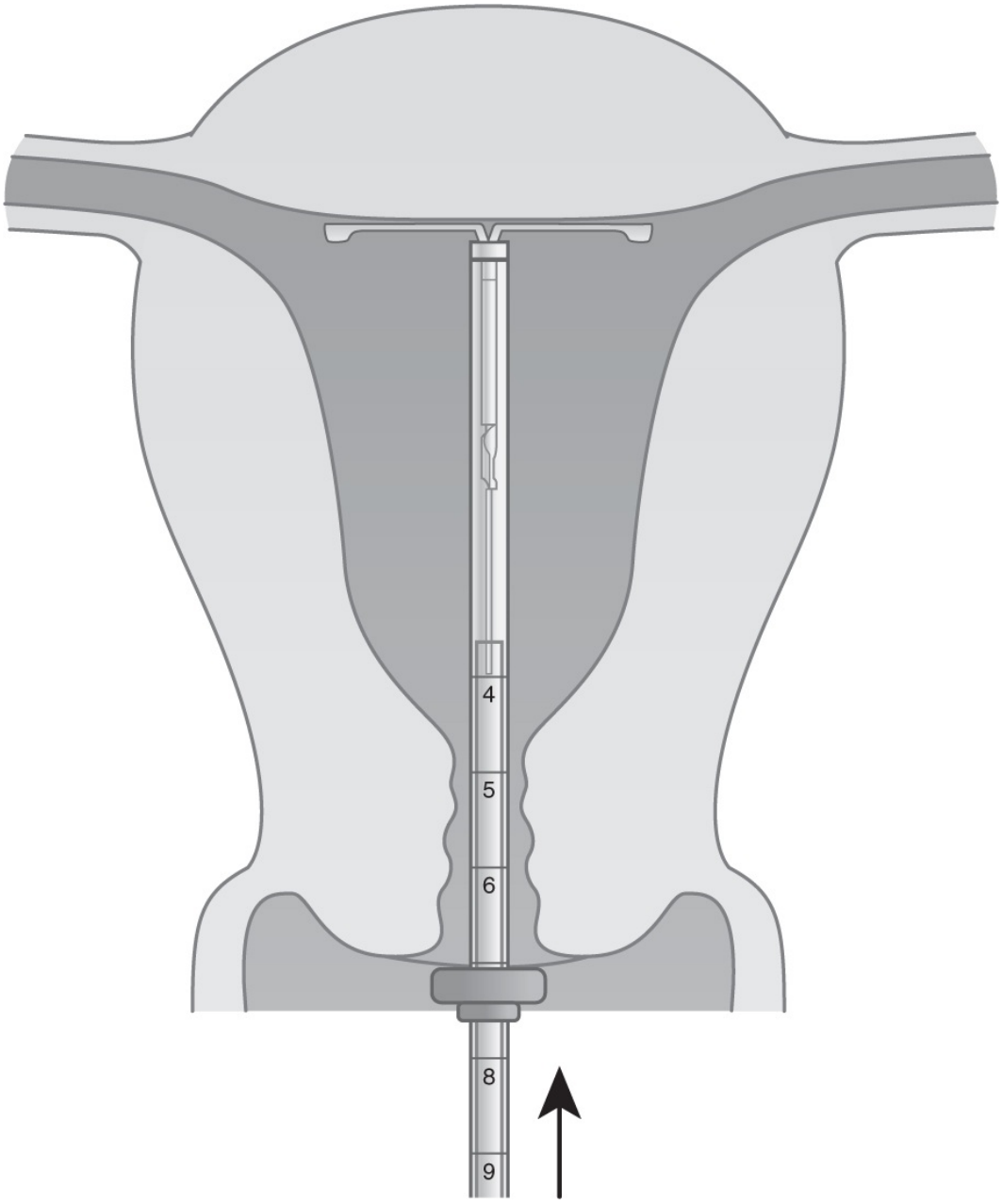


Figure 16C-8 LNG-IUD in the fundus with the flange at the level of the external cervical os.

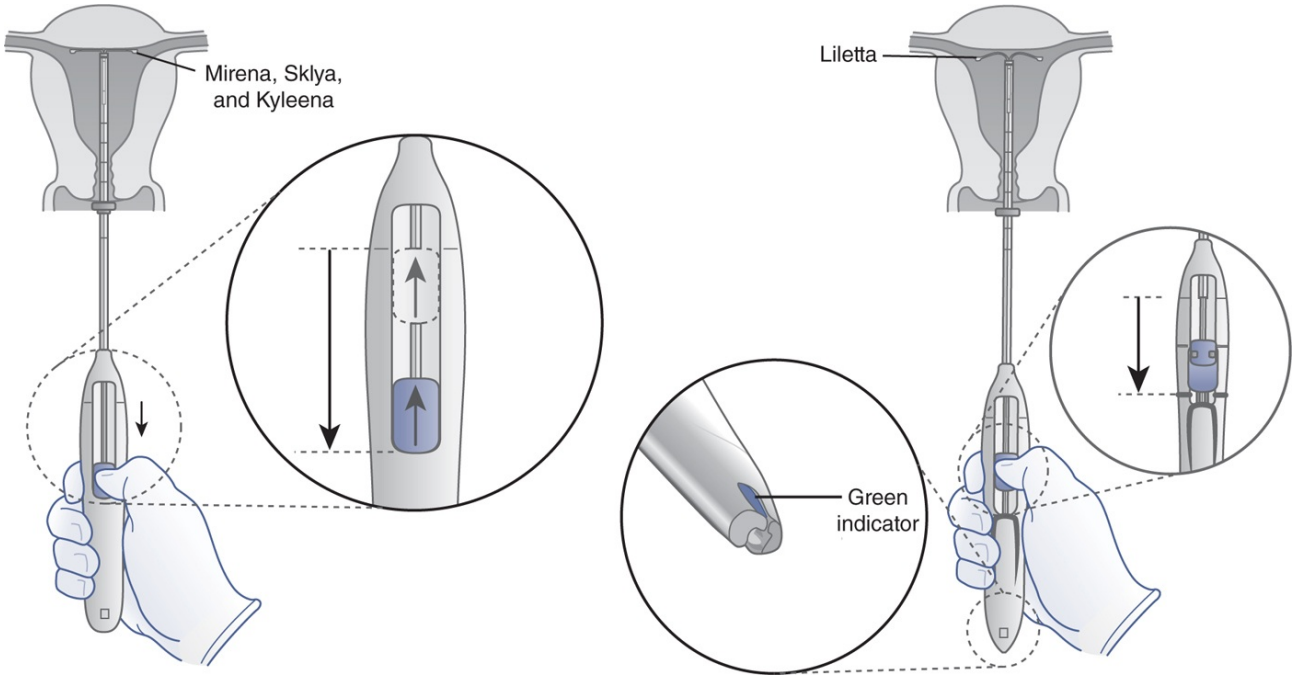


Figure 16C-9 Release of the LNG-IUD slider.

Insertion of a Copper Intrauterine Device (ParaGard T CU 380A IUD)

1. The IUD package (**Figure 16C-10**) is opened in the same manner for any IUD, usually by the assistant, and the clinician reaches for the device in a sterile manner. The clinician should be aware of the components of this IUD (**Figure 16C-11**).
2. Load the IUD into the insertion tubing by slightly withdrawing the insertion tubing and folding the horizontal arms of the IUD down along the vertical arm using the thumb and forefinger. This can be accomplished through the packaging (**Figure 16C-12**) or by using an open sterile technique (**Figure 16C-13**). Take precautions to ensure that the arms are not bent into the tubing until immediately before insertion. If the arms are folded for more than 5 minutes, the device may lose its memory and may not completely unfold in the uterus.
3. Advance the insertion tubing so that the horizontal arms sit securely within the insertion tubing.
4. Place the solid white rod into the bottom of the insertion tubing, advancing it until it touches the bottom of the IUD.
5. Grasp the insertion tube at the open end, and set the flange to the centimeter level predetermined by sounding the uterus.
6. Rotate the insertion tubing so that the horizontal arms of the IUD are parallel to the long axis of the flange.
7. Use a tenaculum or Allis clamp to stabilize the cervix. This stabilization usually is accomplished by the clinician's nondominant hand holding the tenaculum or Allis clamp as the loaded insertion tube is passed through the cervical canal guided by the dominant hand. When resistance is met at the uterine fundus, the flange should be at the external

cervical os (**Figure 16C-14**).

8. While the clinician's nondominant hand stabilizes the solid white rod, withdraw the insertion tubing with the clinician's other hand for a distance of approximately 1 cm. At this time, the IUD is released from the inserter.
9. Gently advance the insertion tube to ensure the IUD is placed in the fundus (**Figure 16C-15**).
10. Gently, the insertion tubing is withdrawn (**Figures 16C-16** and **Figure 16C-17**), and both the rod and the tubing are appropriately discarded.

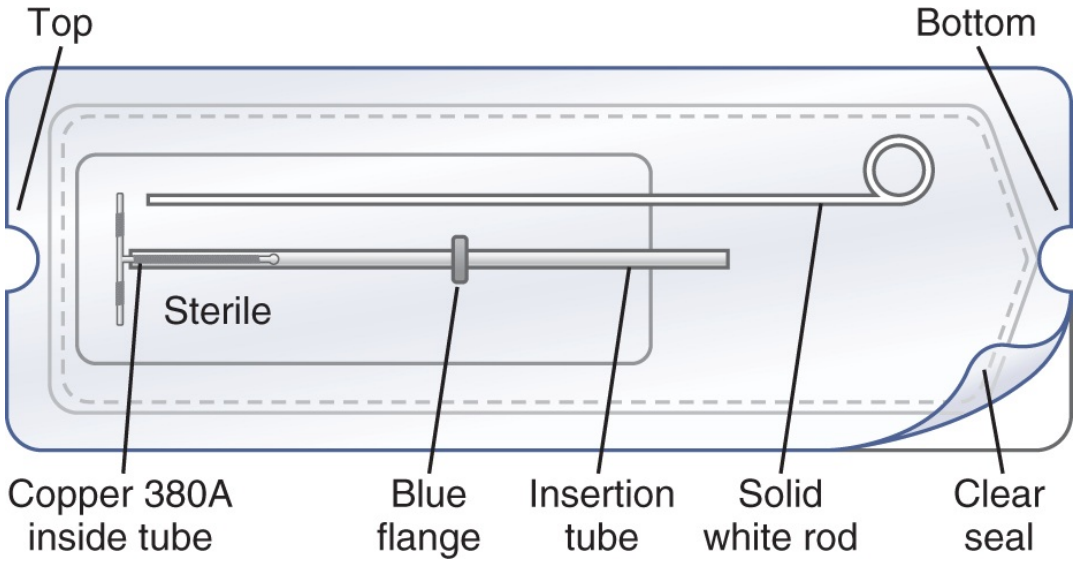


Figure 16C-10 Packaging of copper IUD.

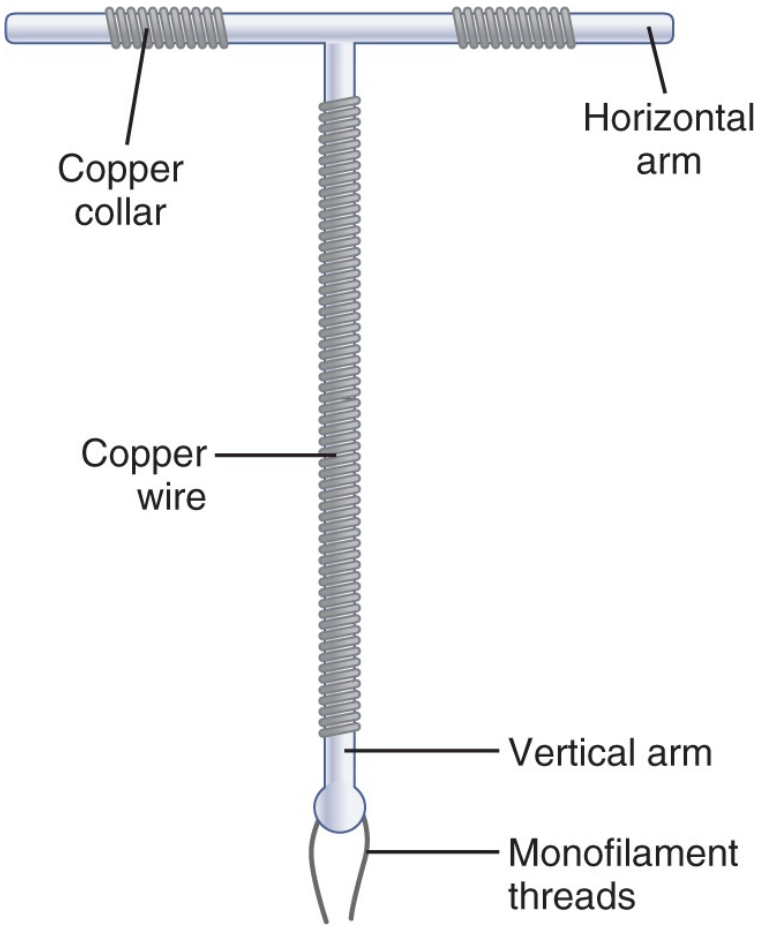


Figure 16C-11 Anatomy of a copper IUD.

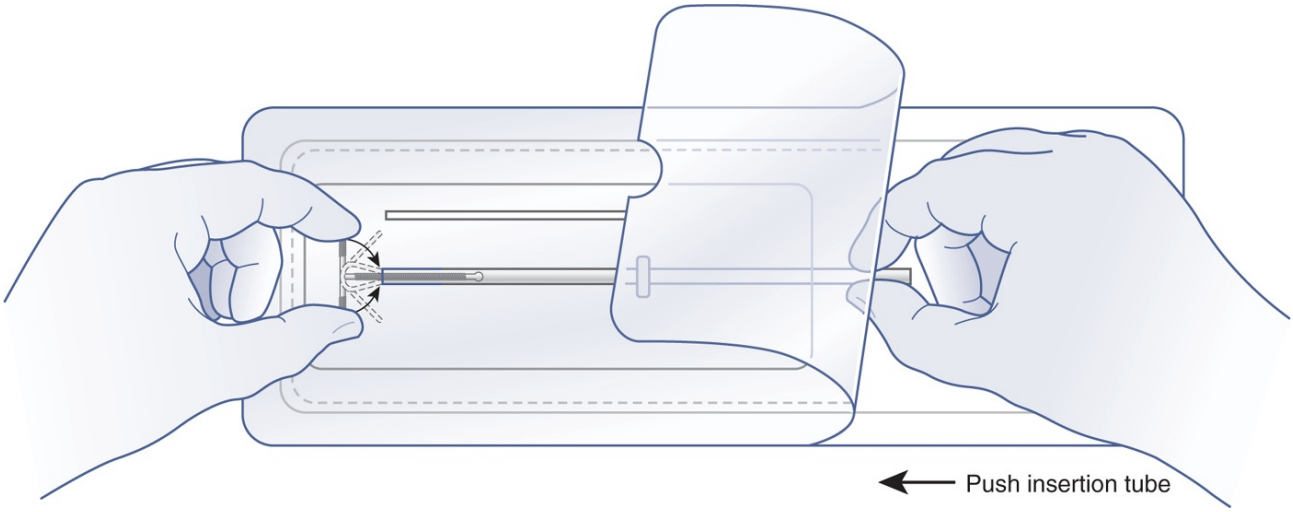


Figure 16C-12 Placement of the arms of a copper IUD into the tubing through the packaging.

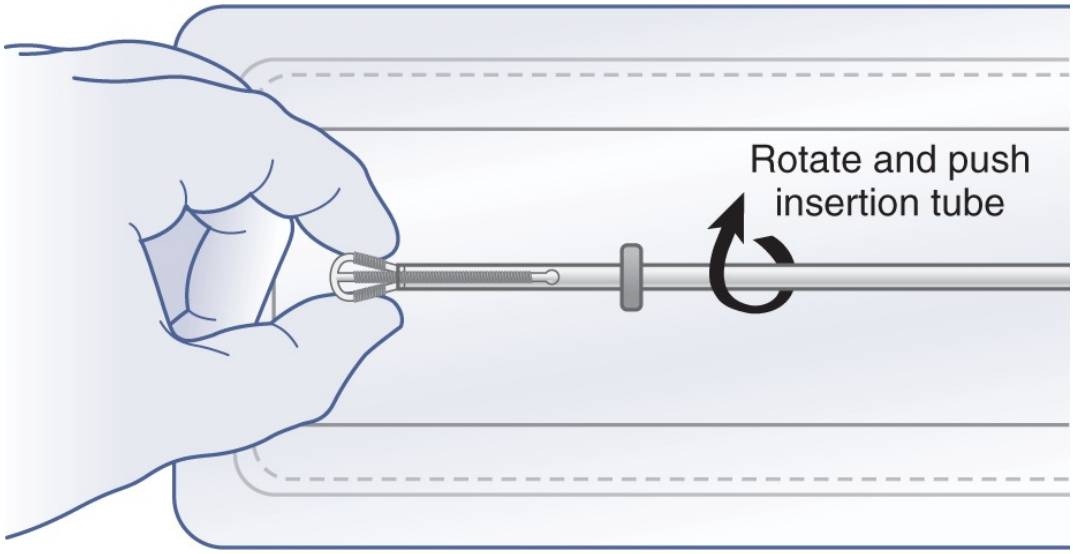


Figure 16C-13 Placement of the arms of a copper IUD into the tubing by open sterile technique.

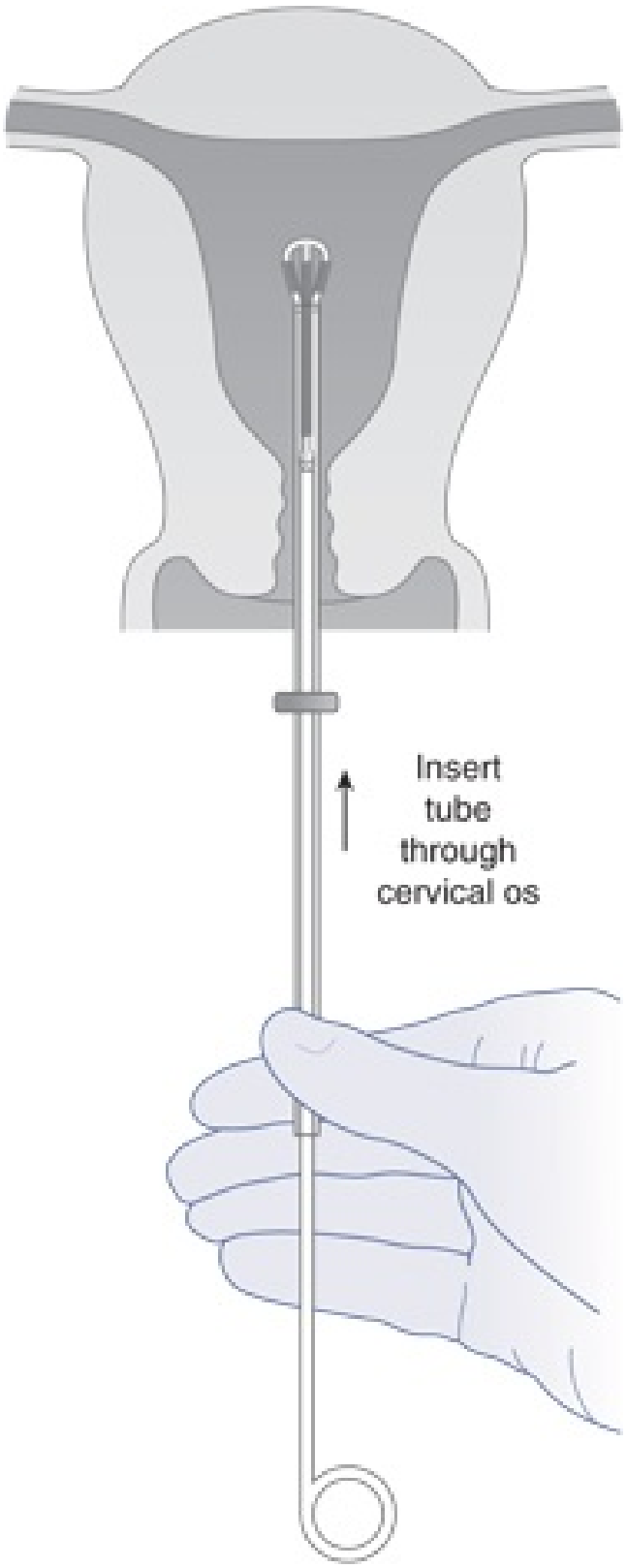


Figure 16C-14 Insertion of a copper IUD and inserter through the cervical os.

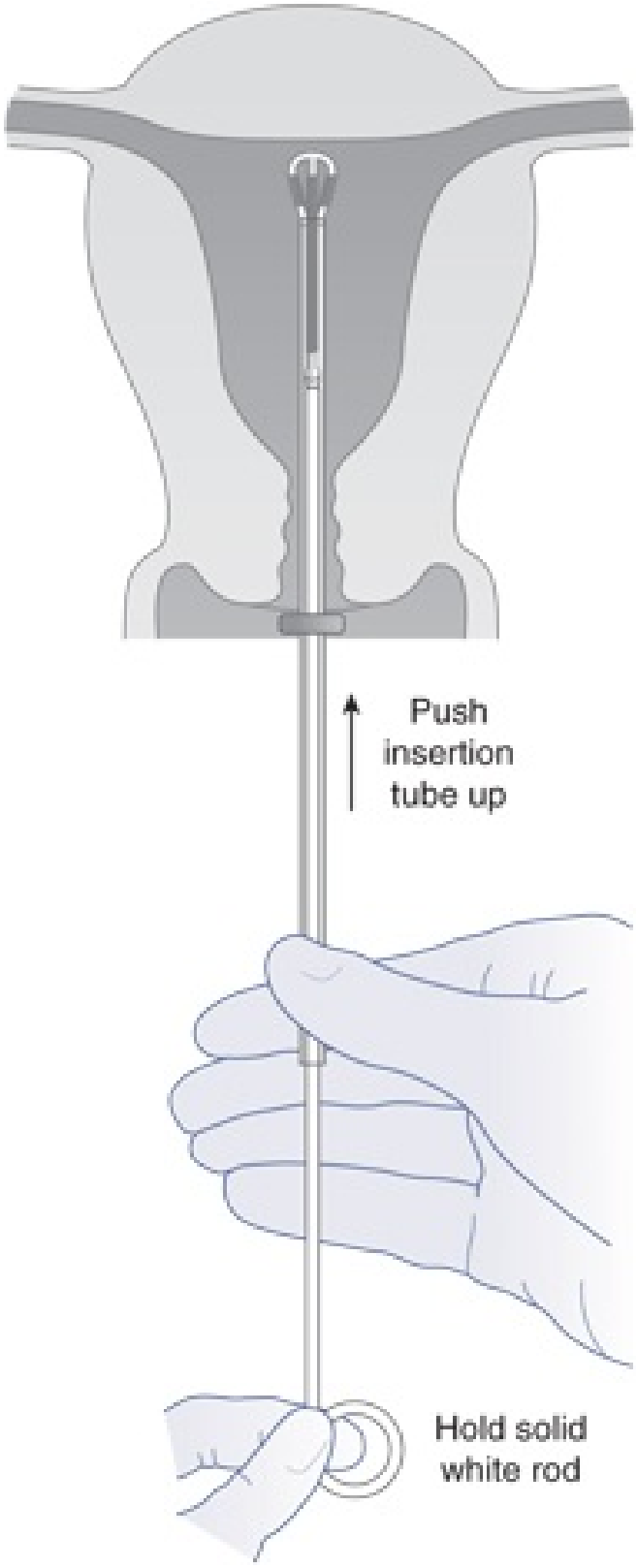


Figure 16C-15 Placement of a copper IUD through the inserter in the fundus.

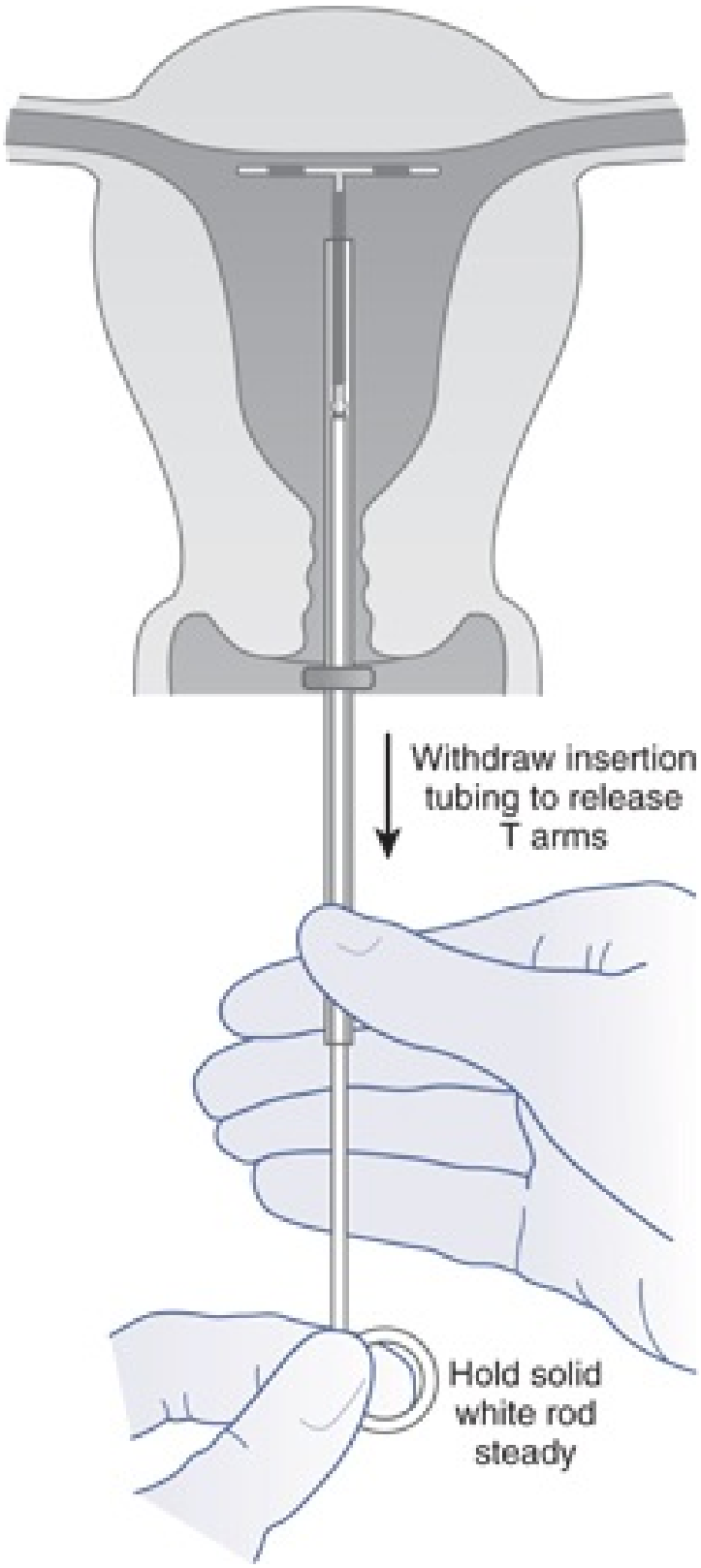


Figure 16C-16 Withdrawal of insertion tubing to release the arms for a copper IUD.

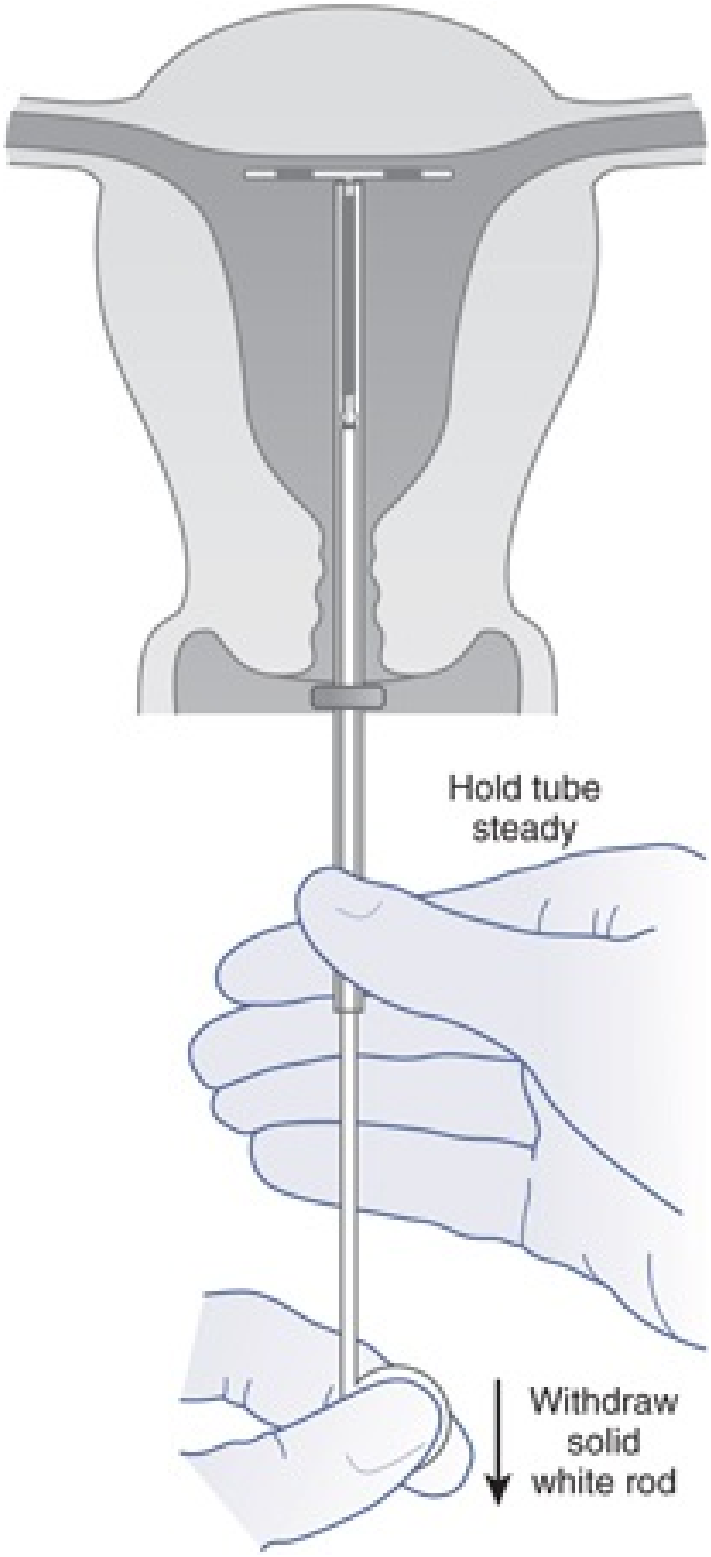


Figure 16C-17 Withdrawal of rod and insertion tubing for a copper IUD.

Postinsertion Care

The clinician should be able to visualize the IUD threads or strings in the vagina after IUD insertion is complete and the inserter materials removed. Using sharp scissors, threads are

trimmed to a length of approximately 3 cm from the external os after which the speculum can be removed. If the woman is overweight or obese, she may need a longer thread length than 3 cm to assure that she can easily feel the strings. Caution should be taken not to cut the threads too short, as they may migrate into the os, whereas longer strings can be shortened at a subsequent visit (**Figure 16C-18**). The exception to post-insertion cutting of the threads occurs when inserting an IUD in the immediate postpartum period, as discussed in that specific section of the *Nonhormonal Contraception* chapter.

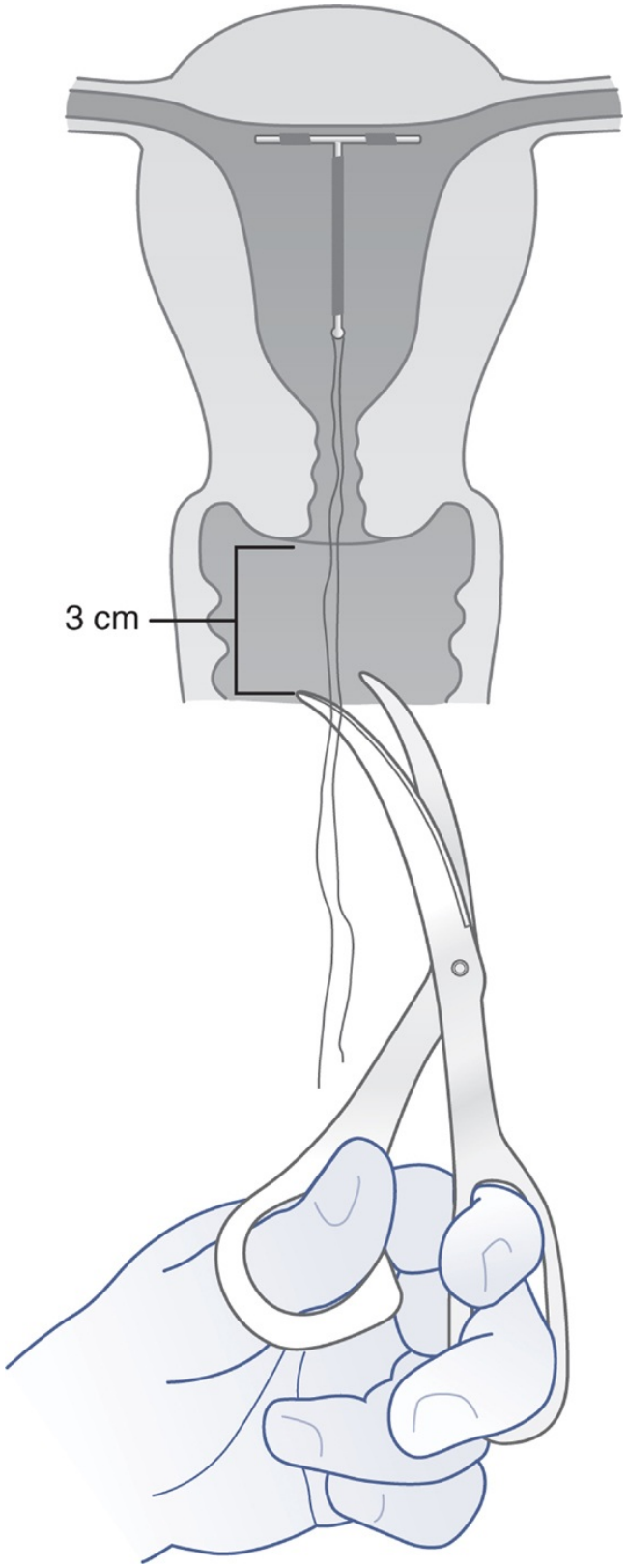


Figure 16C-18 Cutting threads for any IUD.

Immediate Postpartum Insertion of an Intrauterine

Device After Vaginal Birth

Placement of an IUD in the immediate postpartum period is contraindicated for women with peripartum chorioamnionitis, endometritis, or puerperal sepsis.[12] Alternative methods of contraception should be considered and placement of the IUD deferred in such a case. As with any contraceptive method, a woman should be engaged in shared decision making and the decision should be made prior to labor.

Ring Forceps Technique

1. Ensure that the device is placed within 10 minutes of when the placenta is expelled.
2. Clean the external genitalia and apply sterile drapes over the abdomen and under the buttocks.
3. Retract the vagina with a gloved hand, speculum, or retractor.
4. Swab the cervix and vagina with an antiseptic if desired; similarly, anesthesia can be administered as appropriate.
5. Using a ring forceps, stabilize the cervix by grasping the anterior lip. Do not use a tenaculum to avoid tearing the cervix.
6. Remove the IUD from the inserter and grasp the device by the vertical arm, with the horizontal arms slightly outside the ring but on the same plane (**Figure 16C-19**).
7. Cut the strings to 10 cm in length. Note that this is in contrast to cutting strings after insertion among women who are not postpartum.
8. Apply gentle traction to the ring forceps to stabilize the cervix.
9. Insert the device through the cervix and into the lower uterine segment (**Figure 16C-20**).
10. With the clinician's nondominant hand placed on the fundus to stabilize the uterus, advance the device to the uterine fundus, with ring forceps still grasping it, in an upward angle toward the umbilicus (**Figure 16C-21**).
11. If desired, confirm that the device is in place. This may be done under ultrasound guidance.
12. Release the IUD by opening the ring forceps and carefully remove the forceps from the uterus and vagina. Reduce the risk of inadvertently removing the newly device by leaving the forceps slightly open and sweeping laterally to avoid pulling the threads.
13. Examine the cervix and remove the ring forceps.

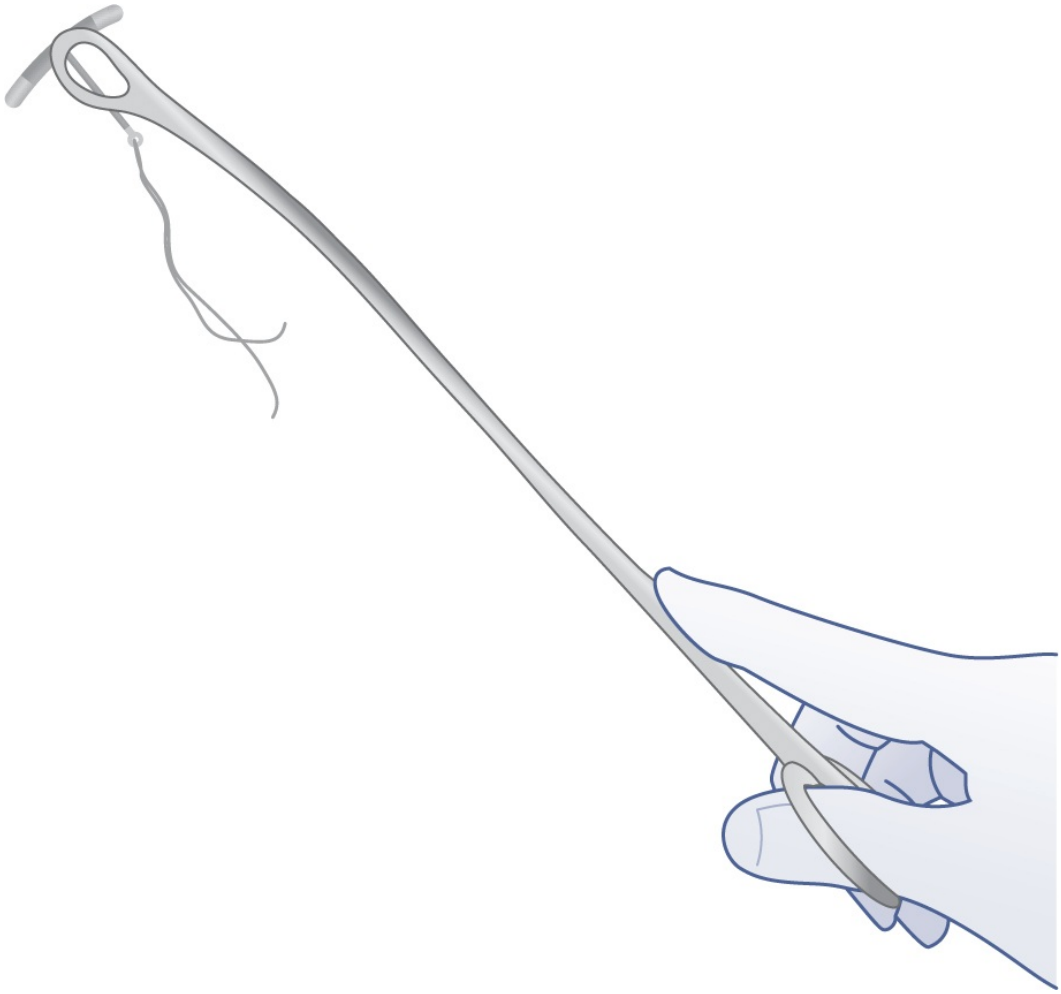


Figure 16C-19 Grasping the IUD with ring forceps.

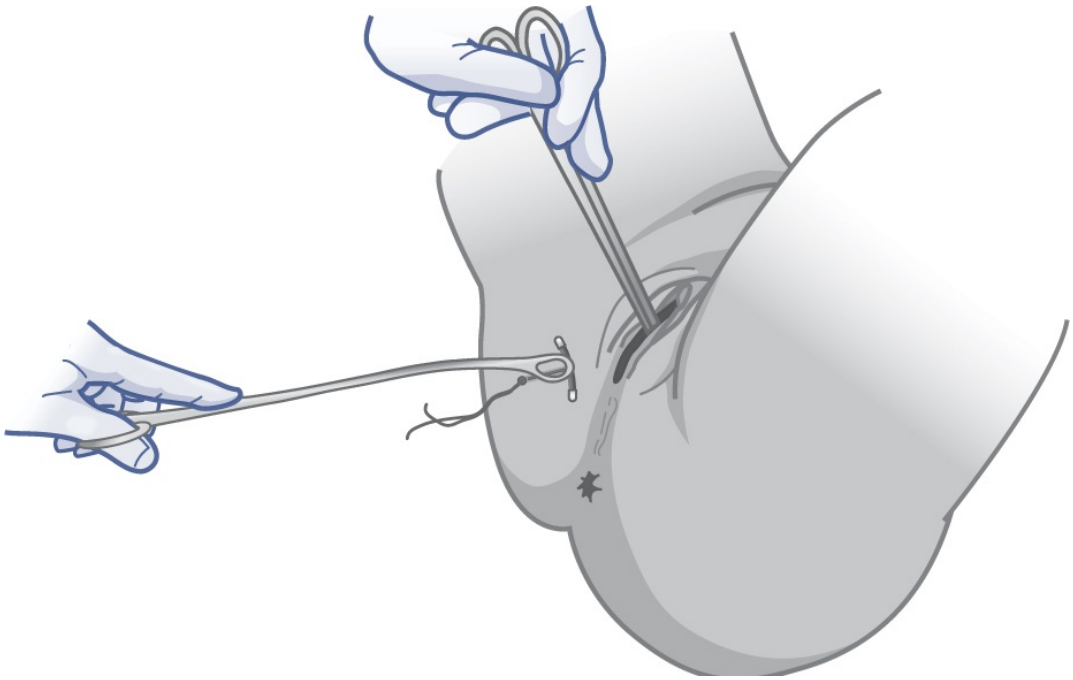


Figure 16C-20 Inserting the IUD with ring forceps while placing traction on the cervix.

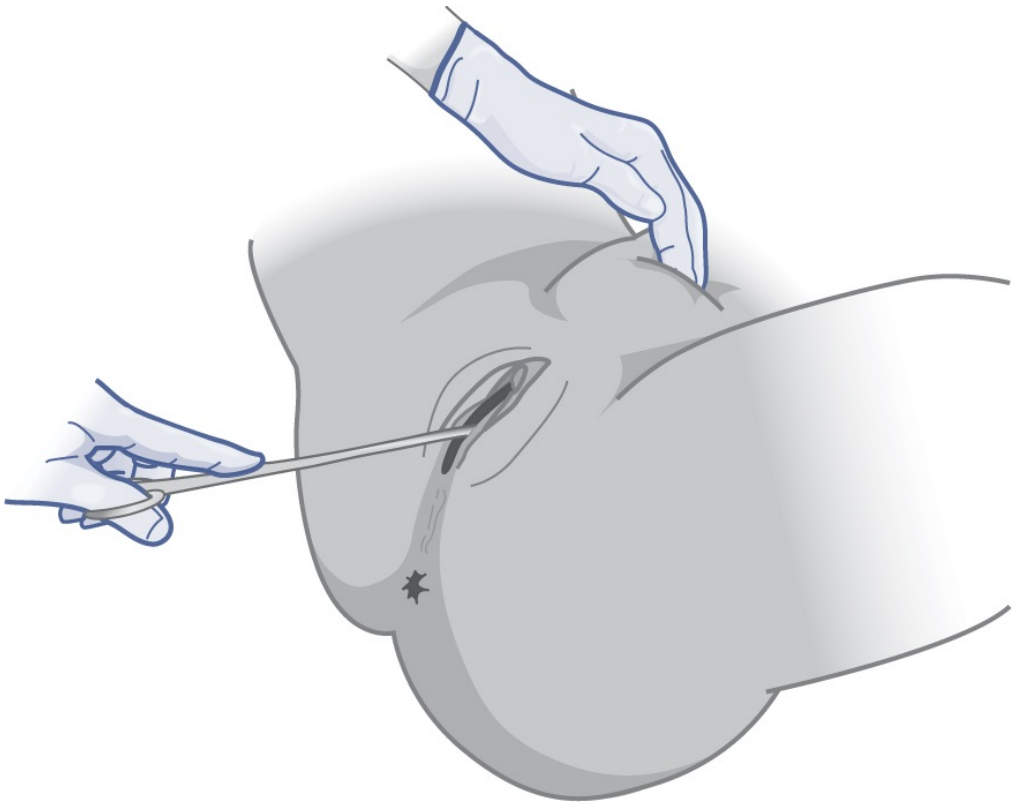


Figure 16C-21 Stabilizing the uterus while placing the IUD in the fundus.

Manual Technique

1. Ensure that the device is placed within 10 minutes of the birth of the placenta.
2. Clean the external genitalia and apply sterile drapes over the abdomen and under the bottom.
3. Swab the cervix and vagina with the antiseptic if desired; similarly, anesthesia can be administered as appropriate.
4. Visualize the cervix with a gloved hand, speculum, or retractor.
5. Remove the IUD from the inserter and grasp the vertical rod between the index and middle fingers of the dominant hand, noting the plane of the horizontal arms (**Figure 16C-22**).
6. Cut the strings to 10 cm in length. Note that this is in contrast to cutting strings after insertion among women who are not postpartum.
7. Insert the device through the cervix and into the lower uterine segment (**Figure 16C-23**).
8. With the clinician's nondominant hand placed fundally to stabilize the uterus, advance the device to the uterine fundus manually at an upward angle toward the umbilicus (**Figure 16C-24**).
9. If desired, confirm that the device is in place. This may be done under ultrasound guidance.
10. Release the IUD and carefully remove the dominant hand from the uterus and vagina, avoiding dislodging the device or inadvertently applying traction on the threads.

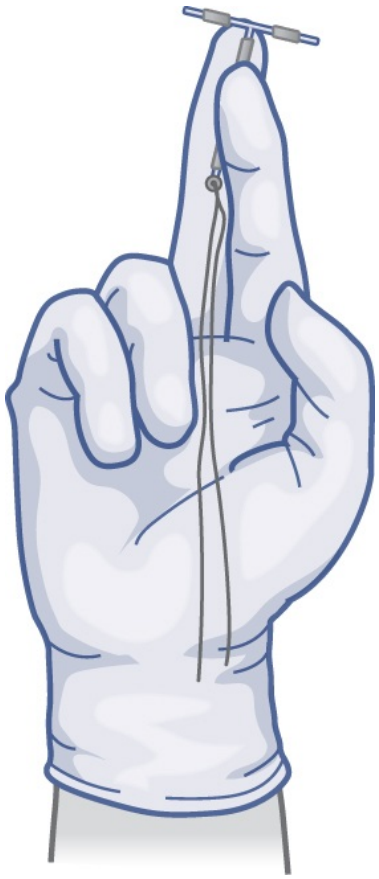


Figure 16C-22 Grasping the IUD prior to manual insertion.

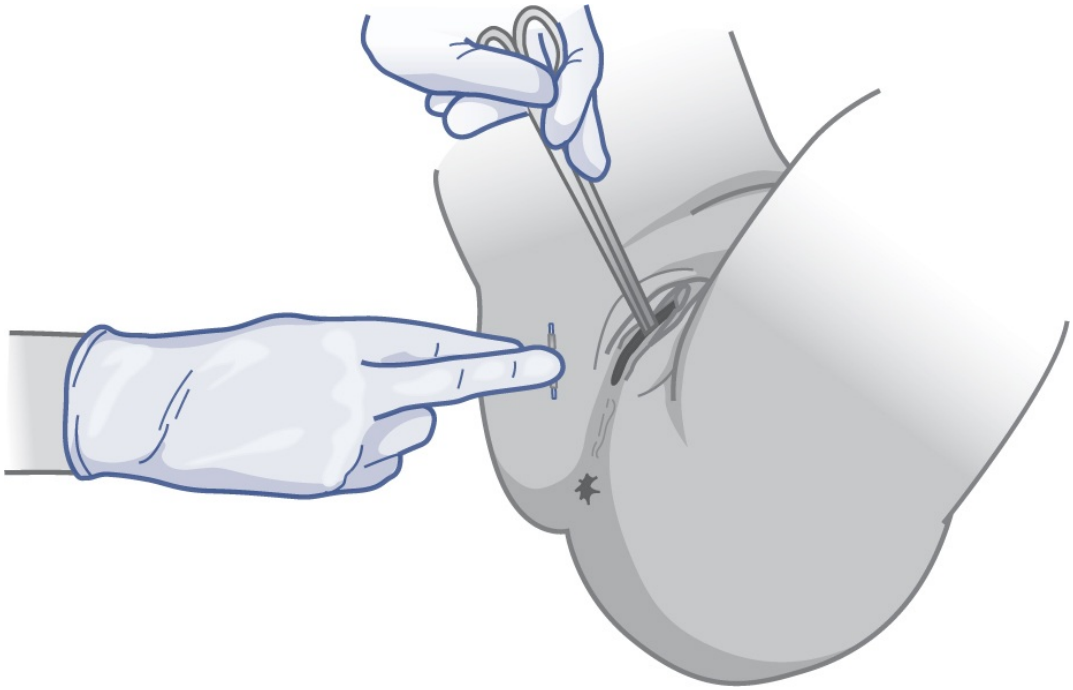


Figure 16C-23 Manually inserting the IUD with a retractor in place if needed.

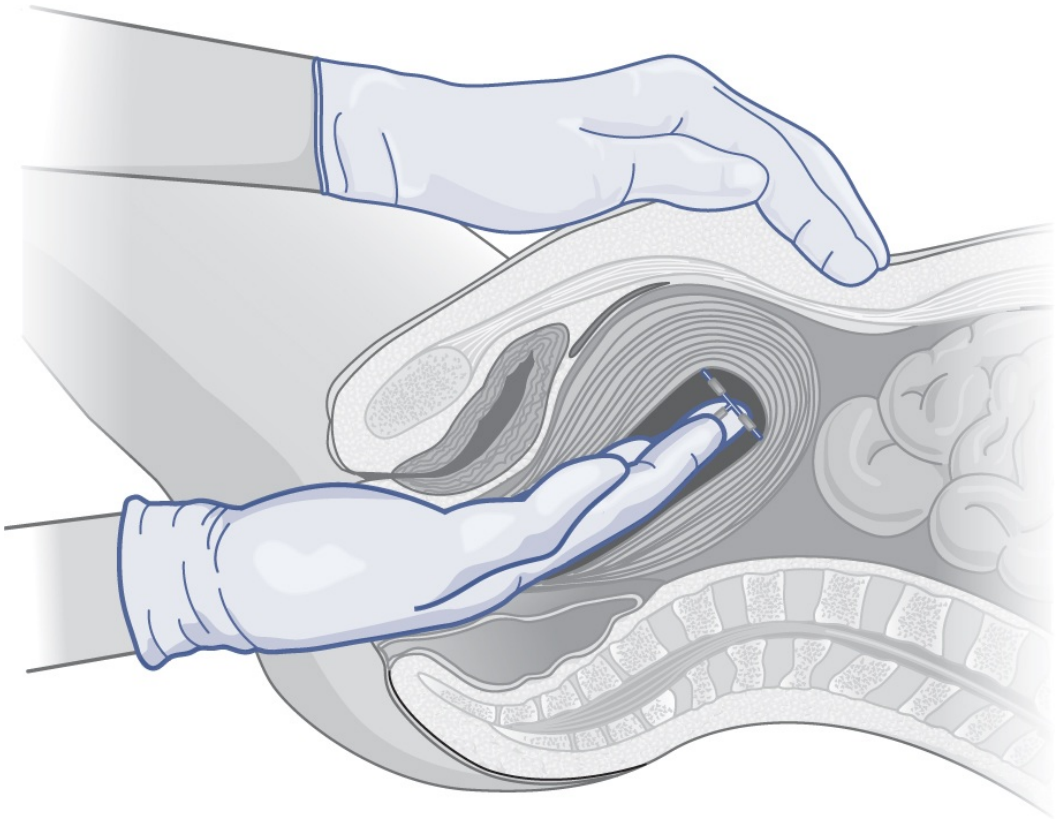


Figure 16C-24 Stabilizing the uterus while manually placing the IUD in the fundus.

Health Education

After insertion of an IUD, health education includes teaching about how to feel for and identify the threads in the vagina (**Figure 16C-25**). A sample IUD with strings the woman can feel may be used as a teaching aid. Any significant variations such as unusual lengthening of the strings, which may indicate nonfundal placement, or feeling any part of the plastic of the device should be reported, as these findings may suggest partial expulsion of the IUD. Some new IUD users may be concerned that either tampon or menstrual cup use will increase the rate of expulsion, though this has not been found to be the case.[24]

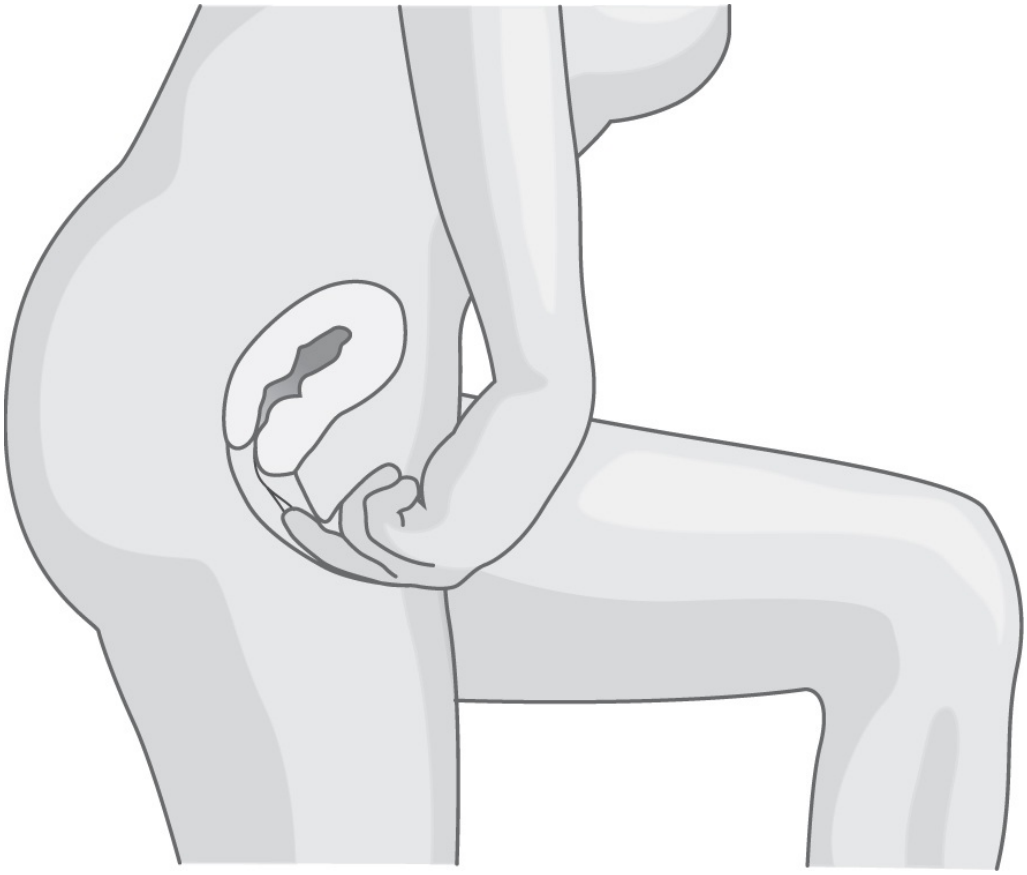


Figure 16C-25 How to feel for IUD threads or strings.

After insertion of the copper IUD, no no additional contraception is required, as this method is immediately effective.[3] The same is true for the LNG-IUD if inserted within 7 days of the onset of menses. However, abstinence or additional contraception should be used for 7 days after initiation of LNG-IUD if inserted more than 7 days after the onset of menses.[3] All women should be counseled that IUDs do not provide protection from STIs.

The mnemonic "PAINS" is often shared after IUD insertion as an easy-to-use reminder of potentially significant deviations that should be reported (**Table 16-5**). In all cases, women should be encouraged to contact their provider with questions or concerns, although most women using an IUD do not experience major side effects. Among the most common side effects of all IUDs are menstrual irregularities. In some women, especially those using a copper IUD, dysmenorrhea or heavy bleeding may occur; the use of nonsteroidal anti-inflammatory drugs (NSAIDs) provides relief from either or both of these symptoms. Conversely, those women using an LNG-containing IUD may experience decreased menstrual flow or amenorrhea. However, sudden amenorrhea for any woman with an IUD suggests pregnancy, and she should be evaluated for this condition.

Follow-Up Care

Usually a follow-up visit is scheduled for women 4 to 6 weeks post IUD insertion. At that

time, any concerns the woman has can be addressed and a speculum examination can be performed to confirm that the threads are visible. After the initial post-insertion visit, future appointments are made based on routine screening guidelines or other risk factors. The only other IUD-related visit should be at the time when the duration of the IUD expires (e.g., Skyla at 3 years); at that point, a new device can be inserted if desired.

Removal of an Intrauterine Device

Common reasons for requesting removal of an IUD include desire for a pregnancy, unnecessary troublesome side effects of menstrual irregularities, establishment of menopause, or end of duration of effectiveness. The removal procedure is the same for all of the IUDs.

Procedure for Removing an Intrauterine Device

1. Removal of an IUD requires clean, but not sterile technique.
2. Removal is a rapid procedure.
3. Constant communication should occur between the clinician and the woman during the removal.
4. After the woman assumes a lithotomy or dorsal position of comfort, perform a bimanual pelvic examination to determine the position of the uterus. Next, insert a speculum; it may be warmed and/or lubricated prior to insertion.
5. Visualize the IUD threads, and use an instrument (e.g., ring forceps) to grasp the threads.
6. Guide the IUD threads downward toward the clinician until the IUD emerges through the cervix and vagina. Some women may feel a sharp cramp as the device passes the cervix, but most report no discomfort or little sensation.
7. Most clinicians show the device to the woman to reassure her it has been removed.
8. The removal is now completed. If the woman desires a new device, it can be immediately placed by following the procedure presented earlier in this appendix.

References

1. Dole DM, Martin J. What nurses need to know about immediate postpartum initiation of long-acting reversible contraception. *Nurs Womens Health*. 2017;21(3):186-195.
2. Callegari LS, Aiken AR, Dehlendorf C, Cason P, Borrero S. Addressing potential pitfalls of reproductive life planning with patient-centered counseling. *Am J Obstet Gynecol*. 2017;216(2):129-134.
3. Curtis KM, Jatlaoui TC, Tepper NK, et al. U.S. Selected Practice Recommendations for Contraceptive Use, 2016. *MMWR Recomm Rep*. 2016;65(RR-4):1-66
4. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR Recomm Rep*. 2016;65(RR-3):1-104.
5. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 539: adolescents and long-acting reversible contraception: implants and intrauterine devices. *Obstet Gynecol*. 2014;120;983-988.
6. Trussell J, Hassan F, Lowin J, Law A, Filonenko A. Achieving cost-neutrality with long-acting reversible contraceptive methods. *Contraception*. 2015;91(1):49-56.
7. Bearak JM, Finer LB, Jerman J, Kavanaugh ML. Changes in out-of-pocket costs for hormonal IUDs after implementation of the Affordable Care Act: an analysis of insurance benefit inquiries. *Contraception*. 2016;93(2):139-144.

8. Heinemann K, Barnett C, Reed S, Möhner S, Minh TD. IUD use among parous women and risk of uterine perforation: a secondary analysis. *Contraception*. 2017;95(6):605-607.
9. Kaislasuo J, Suhonen S, Gissler M, Lähteenmäki P, Heikinheimo O. Intrauterine contraception: incidence and factors associated with uterine perforation: a population-based study. *Hum Reprod*. 2012;27(9):2658-2663.
10. Kaislasuo J, Suhonen S, Gissler M, Lähteenmäki P, Heikinheimo O. Uterine perforation caused by intrauterine devices: clinical course and treatment. *Hum Reprod*. 2013;28(6):1546-1551.
11. Mejia M, McNicholas C, Madden T, Peipert JF. Association of baseline bleeding pattern on amenorrhea with levonorgestrel intrauterine system use. *Contraception*. 2016;94(5):556-560.
12. American College of Obstetricians and Gynecologists. Committee Opinion No. 670: immediate postpartum long-acting reversible contraception. *Obstet Gynecol*. 2016;128:e32-e37.
13. Lopez LM, Bernholc A, Hubacher D, Stuart G, Van Vliet HAAM. Immediate postpartum insertion of intrauterine device for contraception. *Cochrane Database Syst Rev*. 2015;6:CD003036. doi:10.1002/14651858.CD003036.pub3.
14. Whiteman MK, Tyler CP, Folger SG, Gaffield ME, Curtis KM. When can a woman have an intrauterine device inserted? A systematic review. *Contraception*. 2013;87(5):666-673.
15. Workowski KA, Bolan GA. Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep*. 2015;64(RR-3):1-137.
16. Sufrin CB, Postlethwaite D, Armstrong MA, Merchant M, Wendt JM, Steinauer JE. *Neisseria gonorrhea* and *Chlamydia trachomatis* screening at intrauterine device insertion and pelvic inflammatory disease. *Obstet Gynecol*. 2012;120(6):1314-1321.
17. World Health Organization. Medical Eligibility Criteria for Contraceptive Use. 5th ed. Geneva, Switzerland: World Health Organization; 2015. Available at: http://apps.who.int/iris/bitstream/10665/181468/1/9789241549158_eng.pdf?ua=1. Accessed February 21, 2018.
18. Tepper NK, Steenland MW, Marchbanks PA, Curtis KM. Laboratory screening prior to initiating contraception: a systematic review. *Contraception*. 2013;87:645-649.
19. Lopez LM, Bernholc A, Zeng Y, et al. Interventions for pain with intrauterine device insertion. *Cochrane Database Syst Rev*. 2015;7:CD007373. doi:10.1002/14651858.CD007373.pub3.
20. Bahamondes MV, Espejo-Arce X, Bahamondes L. Effect of vaginal administration of misoprostol before intrauterine contraceptive insertion following previous insertion failure: a double blind RCT. *Hum Reprod*. 2015;30:1861-1866.
21. Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation*. 2007;116:1736-1754.
22. Grimes DA, Lopez LM, Schulz KF. Antibiotic prophylaxis for intrauterine contraceptive device insertion. *Cochrane Database Syst Rev*. 1999;3:CD001327. doi:10.1002/14651858.CD001327.
23. Johnson LT, Johnson IM, Heineck RJ, Lara-Torre E. Allis compared with tenaculum for stabilization of the cervix during IUD placement. *Obstet Gynecol*. 2015;125. doi:10.1097/01.AOG.0000463550.01588.fd.
24. Wiebe ER, Trouton KJ. Does using tampons or menstrual cups increase early IUD insertion rates? *Contraception*. 2012;86(2):119-121.

CHAPTER

17

Hormonal Contraception

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Introduction

In 1960, the first oral contraceptive was approved by the U.S. Food and Drug Administration (FDA), and thus began the story of modern hormonal contraception in the United States.[1] The "pill," as it came to be known colloquially, enabled women to make choices regarding pregnancy, when and if they desired. Several clinical sites participated in the initial testing of the "pill." One of these clinical sites was the Frontier Nursing Service, where over a 3-year period in the late 1950s, oral contraception was investigated and research demonstrated the advantages of the midwife as the primary provider of contraceptive care.[2]

Hormonal methods of contraception include both combined oral contraceptives (COCs) and progestin-only pills (POPs), and non-oral hormonal formulations, including rings, patches, and subdermal implants. In addition, intrauterine contraceptive devices impregnated with a progestin are marketed; because of the presence of the hormone in these devices, they are discussed in this chapter, rather than in the *Nonhormonal Contraception* chapter. The manufacturers of these agents—which have the brand names Liletta, Mirena, Skyla, and Kyleena—use the terms intrauterine system (IUS) and intrauterine device (IUD) interchangeably in their descriptions. Other sources use the term intrauterine contraceptive device (IUCD). For consistency, IUD is the abbreviation used throughout this chapter for intrauterine devices.

Hormonal contraceptives generally are subclassified as either a short-acting reversible contraceptive (SARC) or a long-acting reversible contraceptive (LARC).[3]

Evolution of Hormonal Contraceptive Methods

For the last several decades, modifications of the types of the hormones and doses in contraceptives have been made in attempts to decrease the adverse effects associated with these agents while maintaining their contraceptive effectiveness. It has been estimated that 1 million pregnancies occur annually among women who say they are taking oral contraceptives, with many of these pregnancies attributable to inconsistent use of the agents. More than 50% of women using COCs miss two or more pills during the second month they use them.[4] Methods that are less dependent upon daily user action are more effective as a contraceptive. Therefore, in recent years, the focus in contraception has shifted away from development of new estrogens or progestins for oral contraceptives and toward development of alternative routes for delivery of hormones. Newer novel approaches include transdermal patches, IUDs, subdermal implants, and injectable products. Contraceptive options other than the oral formulations offer women a wider variety of choices for the hormone delivery method and duration of action with lower maintenance.

Several hormonal methods combine estrogen and a progestin, whereas others contain only progestin (**Table 17-1**). Appreciating which hormones are present in a particular contraceptive method can assist the provider and woman in anticipating both the common side effects and the rare adverse effects associated with that method.

Table 17-1	Contraceptive Methods and Types of Hormones	
Hormonal Method	**Estrogen and Progestin**	**Progestin Only**
Combined oral contraceptives (COCs)	X	
Progestin-only contraceptives (POPs)		X
Transdermal contraceptive patch	X	
Intravaginal contraceptive ring	X	
Depot medroxyprogesterone acetate injections (DMPA)		X
Subdermal implants		X
Levonorgestrel IUD (LNG-IUD)		X

Mechanism of Action of Hormonal Contraceptives

Hormonal contraceptives have many biologic effects.[5-8] The primary mechanism of action with regard to contraception is inhibition of ovulation and changes to cervical mucus so that sperm transport is impaired. Fallopian tube motility is also altered, which inhibits sperm motility and the endometrium becomes atrophic. Hormonal methods of contraception contain synthetic steroidal hormones that act centrally, altering the functions of the pituitary gland and hypothalamus. Although it may seem counterintuitive that supplementing natural progesterone and estrogen with synthetic analogues of the same hormones can prevent ovulation and impede sperm transport, this remains the most common mechanism of action for hormonal contraceptives.

Mechanism of Action of Estrogen

A major action of estrogen in hormonal contraception is to stabilize the endometrium, thereby providing cycle control and minimizing breakthrough bleeding.[5] Estrogen also suppresses gonadotropin-releasing hormone (GnRH) and pituitary release of both GnRH and follicle-stimulating hormone (FSH). These actions inhibit development of a dominant follicle and subsequent ovulation. Several pharmacologic formulations of estrogen exist. The major estrogen used in hormonal contraception is ethinyl estradiol. Although popular in years past, mestranol is a less potent type of estrogen that is now found solely in the few remaining 50 microgram COCs.[6]

Estrogen is a lipophilic intracellular hormone that has many physiologic effects. For example, estrogen increases the plasma concentrations of clotting factors, which is why it is associated with an increased risk for venous thromboembolism (VTE).[7] Estrogen also increases insulin resistance, very low-density lipids, high-density lipids and may cause a slight rise in blood pressure. Although these estrogen effects could be potentially dangerous for women who have an a priori risk for VTE or cardiovascular disease, today's COCs have forms of estrogen and low doses that are associated with markedly lowered to negligible risks.

Ethinyl estradiol is metabolized by the cytochrome P450 (CYP450) enzymes, which are highly polymorphic. Many drugs can inhibit or accelerate the function of CYP450 enzymes. More than 150 drugs have been suggested as agents that have drug interactions with ethinyl estradiol. These drug interactions potentiate or inhibit the effects of either estrogen or the effect of the interacting drug. Concomitant use of ethinyl estradiol and any other pharmacologic agent should be individually evaluated. Clinically relevant drug interactions are discussed later in this chapter.

Mechanism of Action of Progestins

Unlike estrogen, in which a single type (ethinyl estradiol) is used almost exclusively for contraception, several progestins are prescribed. Progestins include both synthetic and natural progesterone. Progestins include estranes, gonanes, and pregnanes that are derived from 19-nortestosterone or 19-norprogesterone as well as the first non-testosterone agent drospirenone

(**Figure 17-1**). All synthetic progestins bind to the progesterone receptor. In addition, they have varying affinities for androgen, glucocorticoid, mineralocorticoid, and estrogen receptors. The side effects of different progestins are largely related to their individual affinity for these other receptors. In contraceptive methods, progestins inhibit the release of luteinizing hormone (LH), thereby preventing the LH surge necessary for ovulation. Additional mechanisms of action are theorized to include thickening of the cervical mucus and delayed sperm transport. Progestin-only agents lack the endometrial stabilization from estrogen, which increases irregular vaginal bleeding.

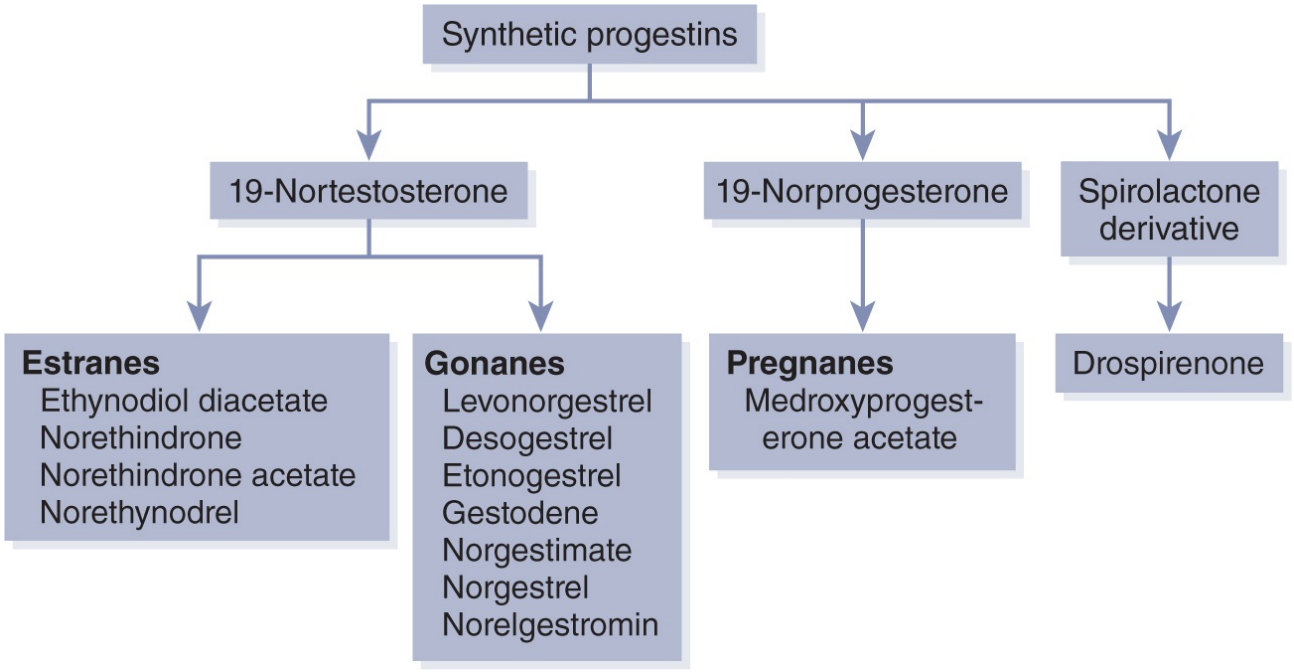


Figure 17-1 Progestins used for contraception.

General Considerations for Hormonal Contraceptives

When providing women with the information they need to make a choice about hormonal contraception, several topics are reviewed including: effectiveness, how long it takes for fertility to return when the agent is discontinued, non-contraceptive benefits, possible drug interactions, danger signs and contraindications.

Effectiveness

Hormonal contraceptives are the most effective method of all reversible contraception products. **Table 17-2** summarizes the effectiveness of the hormonal methods, with the least effective options listed first.[9] Because of their lack of dependency upon user actions, LARCs —unsurprisingly—are more effective than SARCs.

Table 17-2	Effectiveness of Hormonal Contraceptive Methods	
Contraceptive Method	**Type of Method**	**Unintentional Pregnancy (%)[a]**
Oral method (pills containing either combined estrogen/progestin or progestin only)	SARC	9
Transdermal patch	SARC	9
Contraceptive ring	SARC	9
Contraceptive injections	SARC	6
Levonorgestrel IUD	LARC	0.2–0.8[b]
Subdermal implants	LARC	0.05[c]

Abbreviations: IUD, intrauterine device; LARC, long-acting reversible contraceptive; SARC, short-acting reversible contraceptive.

[a] Approximate percentage of women experiencing an unintentional pregnancy during the first year of use.

[b] Based on which IUD is studied.

[c] Based on studies of Implanon. Nexplanon is expected to have similar effectiveness.

Data from Trussell J, Guthrie KA. Choosing a contraceptive: efficacy, safety and personal considerations. In: Hatcher RA, Trussell J, Nelson AL, Cates W, Kowal D, Policar MS, eds. *Contraceptive Technology.* 20th ed. New York, NY: Ardent Media; 2011:45-75.[9]

Return to Fertility

For most hormonal methods, including LARCs, return to fertility is relatively rapid, rarely taking more than a few months.[10] The exception is depot medroxyprogesterone acetate (DMPA), for which an average of 9 months usually passes after the last injection before normal fertility resumes. **Table 17-3** lists the length of time for the average woman to attain fertility after discontinuation of specific hormonal methods.[9,10]

Table 17-3	Average Length of Time for Return to Fertility After Discontinuation of Contraceptive Method
Contraceptive Method	**Average Length of Time for Return to Fertility**
Combined oral contraceptives	1–3 months
Progestin-only pills	1–3 months
Transdermal patch	1–3 months
Contraceptive ring	1 week–1 month
Contraceptive injections	9 months after last injection
Levonorgestrel IUD	Immediately upon removal
Subdermal implants[a]	1–3 weeks after removal[a]

[a] Based on studies of Implanon. Nexplanon is expected to have similar effectiveness.

Based on Trussell J, Guthrie KA. Choosing a contraceptive: efficacy, safety and personal considerations. In: Hatcher RA, Trussell J, Nelson AL, Cates W, Kowal D, Policar MS, eds. *Contraceptive Technology.* 20th ed. New York, NY: Ardent Media; 2011:45-75[9]; Speroff L, Darney P. *A Clinical Guide for Contraception*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.[10]

Noncontraceptive Benefits

Discussion of hormonal contraceptives often focuses on side effects and adverse effects associated with their use, rather than on their benefits. Although adverse effects are always reviewed, most of the side effects are minor, and serious adverse effects are rare. However, a number of noncontraceptive benefits also are associated with these agents, as described in **Table 17-4**. Counseling about the noncontraceptive benefits of these products should be balanced with discussion of their untoward effects.[11,12]

Table 17-4	Noncontraceptive Benefits of Hormonal Contraceptives
Reduction of menstrual-associated conditions:	
Dysmenorrhea	
Heavy menstrual flow	
Irregularity	
Menstrual migraines	
Perimenopausal symptoms (e.g., hot flashes)[a]	
Premenstrual syndrome[a]	
Therapeutic use—treatment of women with the following conditions:	
Bleeding associated with fibroids	
Pelvic pain associated with endometriosis	
Acne[a]	
Decreased risk of the following conditions:	
Anemia (from uterine bleeding)	

Colorectal cancer[a]
Endometrial cancer[a]
Hirsutism[a]
Osteoporosis[a]
Ovarian cancer[a]
Lifestyle (drug-induced) amenorrhea[b]

[a] Associated with estrogen and progestin hormonal contraceptives rather than progestin-only contraceptives.

[b] Menstrual suppression may be seen as a benefit to some women, although other women desire a monthly menses as personal assurance that they are not pregnant.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 110: noncontraceptive uses of hormonal contraceptives. *Obstet Gynecol.* 2010;115:206-218. [Reaffirmed 2016][11]; Hilliard PA. Menstrual suppression: current perspective. *Int J Womens Health*. 2014;6:631-637.[12]

Contraindications to Use of Hormonal Contraceptives

Although most women can use hormonal contraceptives, there are some situations in which these agents are not recommended or are even contraindicated. Contraindications to hormonal contraceptive use are primarily contraindications to estrogen, progestins, or both, and include —most notably—breast cancer and pregnancy. The list of contraindications to estrogen is longer than the list of contraindications to progestins. For example, women with a history of cardiovascular disease or coagulopathy should not use estrogen-containing products. Contraindications defined as Category 3 or 4 in the U.S. Medical Eligibility Criteria for Contraceptive Use are found in **Table 17-5**.[13]

Table 17-5	**Contraindications for Hormonal Contraceptives Based on Category 3 or Category 4 Status in U.S. Medical Eligibility Criteria for Contraceptive Use**
Absolute Contraindications (U.S. MEC for Contraceptive Use Category 4)	**Relative Contraindications (U.S. MEC for Contraceptive Use Category 3)**
Combined Oral Contraceptives (COCs)	
Current breast cancer	Anticonvulsant therapy
Cirrhosis (severe)	Antiviral therapy with ritonavir-boosted protease inhibitor
DVT history of and not on an anticoagulant or acute DVT or PE or DVT/PE and established anticoagulation for at least 3 months but still high risk	Breast cancer history and no evidence of current disease for 5 years
Diabetes mellitus: nephropathy/retinopathy/neuropathy[a] or other vascular disease or diabetes of > 20 years' duration[a]	*Breastfeeding and < 1 month postpartum*
Headache (migraine) with aura	DVT history and low risk for recurrent DVT/PE
History of stroke	Gallbladder disease
Hypertension with systolic ≥ 160 mm Hg or diastolic ≥ 100 mm Hg or vascular disease	*Headaches (migraine) without aura < 35 years[b]*
Ischemic heart disease (current or history)	History of bariatric surgery with malabsorptive procedures
	Hypertension adequately controlled or systolic 140–159 mm Hg or diastolic 90–99 mm Hg
	Inflammatory bowel disease[b]
	Postpartum cardiomyopathy ≥ 6 months

Known thrombogenic mutations Liver tumors: hepatocellular or malignant *Multiple risk factors for atherosclerotic cardiovascular disease*[a] *Peripartum cardiomyopathy (< 6 months) or moderate impaired cardiac function* *Postpartum < 21 days* *Smoking at age ≥ 35 years and ≥ 15 cigarettes a day* [c] Solid organ transplant, complicated Stroke: history of cerebrovascular accident Systemic lupus erythematosus positive or unknown antibodies Valvular disease, complicated *Viral hepatitis acute*[a,b]	Postpartum and breastfeeding with additional risk factors for venous thromboembolism Rifampin or rifabutin therapy *Smoking at age ≥ 35 years and < 15 cigarettes a day* [c]
Contraceptive Implant	
Current breast cancer	Breast cancer history and no evidence of current disease for 5 years Cirrhosis (severe) *Headaches (migraine) without aura < 35 years*[b] History of bariatric surgery with malabsorptive procedures Ischemic heart disease (current or history) Liver tumors: hepatocellular or malignant Solid organ transplant, complicated Systemic lupus erythematosus positive or unknown antibodies Unexplained vaginal bleeding suspicious for serious condition before evaluation
Depot Medroxyprogesterone Acetate (DMPA)[d]	
Current breast cancer *Long-term use of corticosteroid therapy in women with a history or risk of nontraumatic fractures*	Breast cancer history and no evidence of current disease for 5 years Cirrhosis (severe) *Diabetes mellitus: nephropathy/retinopathy/neuropathy or other vascular disease or diabetes of > 20 years' duration*[a]
Depot Medroxyprogesterone Acetate (DMPA)[d]	
	Headaches (migraine) without aura < 35 years[b] *Hypertension with systolic ≥ 160 mm Hg or diastolic ≥ 100 mm Hg or vascular disease* Ischemic heart disease (current or history) Liver tumors: hepatocellular or malignant *Multiple risk factors for atherosclerotic cardiovascular disease*[a] *Rheumatoid arthritis on immunosuppressive therapy*[b] Stroke: history of cerebrovascular accident Systemic lupus erythematosus positive or unknown

	antibodies or severe thrombocytopenia Unexplained vaginal bleeding suspicious for serious condition before evaluation
Levonorgestrel Intrauterine Device	
Current breast cancer Cervical cancer awaiting treatment Gestational trophoblastic disease with persistency elevated beta-hCG levels or malignant disease, with evidence of or suspicion of intrauterine disease *Pelvic inflammatory disease, current*[b] Pregnancy *Sexually transmitted disease with current purulent cervicitis or chlamydial infection or gonococcal infection*[b] *Post septic abortion or birth*[b] *Pelvic tuberculosis*[b] *Unexplained vaginal bleeding*[b]	*Anticonvulsant therapy*[b] *Antiviral therapy with ritonavir-boosted protease inhibitor*[b] Breast cancer history and no evidence of current disease for 5 years Cirrhosis (severe) *Headaches (migraine) without aura < 35 years*[b] *Ischemic heart disease (current or history)*[b] Liver tumors: hepatocellular or malignant *Sexually transmitted infections increased risk*[b] Solid organ transplantation complicated for initiation Stroke: history of cerebrovascular accident Systemic lupus erythematosus positive or unknown antibodies
Progestin-Only Pills (POPs)	
Current breast cancer	Anticonvulsant therapy Antiviral therapy with ritonavir-boosted protease inhibitor Breast cancer history and no evidence of current disease for 5 years *Headaches (migraine) without aura < 35 years*[b] History of bariatric surgery with malabsorptive procedures Ischemic heart disease (current or history) Liver tumors: hepatocellular or malignant Rifampin or rifabutin therapy *Stroke: history of cerebrovascular accident*[b] Systemic lupus erythematosus or unknown antibodies

Abbreviations: DVT, deep vein thrombosis; hCG, human chorionic gonadotropin PE, pulmonary embolus.

[a] Listed as both category 3 and 4.

[b] For initiation of the method, Category 2 or 3 for continuation.

[c] Because smoking is often underreported, the American College of Obstetricians and Gynecologists recommends any woman older than 35 years who smokes should not take COCs.

[d] DMPA is not recommended for women who plan a pregnancy within one year because the return of ovulation after discontinuing this agent can be delayed.

Based on Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep.* 2016;65(3):1-103. Available at: https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf. Accessed August 27, 2017.[13]

Women seeking to use hormonal contraceptives should always be screened for the presence of any contraindications first; if any are present, alternative contraceptive methods should be discussed and recommended. The U.S. Medical Eligibility Criteria (U.S. MEC)

recommendations, which are summarized in the *Family Planning* chapter, include specific contraceptive eligibility guidelines for women who have a variety of disorders and health conditions.[13] The U.S. MEC for Contraceptive Use are also available as an application for smartphones, and on the Centers for Disease Control and Prevention (CDC) website.

Side Effects and Adverse Effects of Hormonal Contraceptives

In general, most randomized controlled trials of drug methods are placebo controlled; that is, these studies explore the risk of use of an agent compared to the risk of not using that agent. One of the challenges in the study of hormonal contraceptives is that ethics precludes such experimentation. Therefore, the majority of studies on the effects of hormonal contraceptives lack the rigor of randomized controlled trials. In addition, contraceptive clinical trials generally focus on healthy women and usually exclude adolescents, women with pre-existing conditions, and even women who are outside the normal parameters for weight. As a result, application of data from studies to everyday clinical practice is challenging. Because the participants in clinical trials are healthy women, some risks may not become apparent until large postmarketing populations adopt the methods. Despite these limitations, hormonal contraceptives have been the subject of extensive research and are considered quite safe for healthy women. The U.S. MEC for Contraceptive Use, as described in the *Family Planning* chapter, provide guidelines for use of the different hormonal methods based on clinical trials as well as national and international studies.[13] In 2016, the CDC published a set of recommendations on some common issues related to use of contraceptives as a companion to the U.S. MEC for Contraceptive Use, as discussed in the *Family Planning* chapter.[13]

Side Effects of Estrogen and Progestins in Contraceptives

Both estrogens and progestins have multiple biologic effects.[5,14] Given the different forms, doses, and routes of administration available, it can be difficult to determine which side effect is related to estrogen and which is related to progestin in a particular product. In general, the most common side effects of estrogen are headaches and nausea. Bloating, leukorrhea, chloasma, and increased cholesterol in bile (which increases the risk of gallstones) are also reported.

All progestin-only methods are safer than the combined products but progestins are associated with bleeding irregularities including amenorrhea. Common side effects of progestins include acne, decreased libido, pruritus, mood changes, premenstrual-type symptoms such as edema, and transient depression, which is usually mild. DMPA is associated with a decrease in bone mass or bone mineral density that is thought to be reversible in women who are not in the perimenopausal period. Studies that have evaluated the effect of progestin on mood changes and depression have conflicting findings, and depression is not a contraindication for use of progestin containing contraceptives. That said, when a woman reports mood changes associated with progestin, an alternate form of contraception should be considered.

Both estrogen and progestin methods may have similar or overlapping side effects,

especially during the first few months of use. Headaches and nausea may be more common with estrogen containing pills, but nausea is often encountered with any oral method. Other problems that women may report with hormonal contraceptives include breast tenderness, acne, and premenstrual symptoms. The latter two are most likely to occur among women using progestin-only methods.

Adverse Effects of Hormonal Contraceptives

Estrogen use increases the risk for some rare, but severe, adverse effects. Notably, estrogen is associated with an increased risk for cardiovascular events such as stroke and myocardial infarction, and thromboembolic events such as deep vein thrombosis (DVT). In addition, estrogen use slightly increases the risk of developing breast cancer. Women who smoke, are older than age 35 years and who use estrogen are at a higher risk for myocardial infarction. The risk of breast cancer is low among reproductive-age women, but evidence indicates that estrogen and progestin contraceptives increase that risk; thus, use of these agents should be carefully considered for women with a personal history of breast cancer. Some progestin-only methods also list breast cancer as a contraindication for use, even though the evidence is not as strong for this proscription. An increased risk of VTE may be associated with use of certain progestins. For example, the risk of venous thrombosis associated with combined oral contraceptives containing the progestins gestodene, desogestrel, and drospirenone plus 30–35 micrograms of ethinyl estradiol is approximately 50% to 80% greater than the risk associated with the same estrogen combined with levonorgestrel.[8] Some studies have suggested that progestins also are associated with an increased risk of breast cancer, although these studies were primarily conducted among perimenopausal/postmenopausal women and the proposed risk is the product of extrapolation. Relationships between progestins and cardiovascular risk and bone health continue to be studied.

Counseling Women About Side Effects and Adverse Effects of Hormonal Contraceptives

The midwife can assist the woman in viewing her individualized risks and benefits. It is important to remember that hormonal contraceptives do not protect a person from acquiring a sexually transmitted infection (STI). Condoms are the only contraceptive method that serves the dual purpose of contraception and STI protection. For example, a woman may decide that the benefits in terms of the reliability and effectiveness of a hormonal method for preventing pregnancy outweigh a relatively minor increase in her risk for a specific condition. Consideration of risk of a condition also should be viewed in light of risk of pregnancy with a less reliable method. For example, the risk of DVT is known to increase with use of estrogen-containing contraceptives. However, for women who do not have a history of coagulopathy or medical disorder that increases her a priori risk for coagulopathy, the risk of DVT associated with estrogen-containing contraceptives is less than the risk of developing DVT during pregnancy. Thus, shared decision making and a careful cost–benefit assessment are essential steps prior to prescribing any hormonal contraception unless an absolute contraindication

exists.

Drug–Drug Interactions

All hormonal contraceptive methods are considered to have potential drug interactions, including implants and IUDs, although their relationships are controversial. The effect of one or both of the agents can be potentiated; one or both can be inhibited; one can be inhibited and the other potentiated; or nothing clinically relevant can occur. Most of these effects occur because the same liver enzymes in the CYP450 family metabolize the two drugs, and one or the other drug alters the function of the enzymes, thereby affecting metabolism of the other agent.

Many drug interactions occur secondary to use of pharmacologic agents that are CYP450 enzyme inducers (i.e., they potentiate the effect of the enzyme, which leads to rapid metabolism of the other drug). Fortunately, healthy women do not commonly use most of the drugs that decrease contraceptive effectiveness. The majority of known drug interactions occur with oral (rather than non-oral hormonal) contraceptives. Nevertheless, the midwife should be particularly alert for potential interactions if a woman is taking medications to treat tuberculosis, a seizure disorder, a clotting disorder, human immunodeficiency virus (HIV), or mild depression.

Among the drugs that have been suggested to decrease the effectiveness of COCs are anti-infectives (specifically rifampin [Rifadin]), anticonvulsants (e.g., carbamazepine [Tegretol], phenytoin [Dilantin], phenobarbital [Luminal]), antifungals (specifically griseofulvin [Fulvicin]), protease inhibitors (e.g., saquinavir [Invirase], ritonavir [Norvir]), and non-nucleoside reverse transcriptase inhibitors (e.g., efavirenz [Sustiva], nevirapine [Viramune]).[13] St. John's wort—a nutritional supplement marketed as a treatment for mild depression—also has been found to decrease the effectiveness of COCs. Simultaneous administration of oral contraceptives with an over-the-counter (OTC) antacid (e.g., Maalox, Mylanta) can result in decreased contraceptive effectiveness due to impaired absorption of the hormones.

Several of these agents also have been suggested to affect the effectiveness of POPs—specifically, rifampin, anticoagulants, antiretrovirals, and St. John's wort. The majority of these drug interactions are associated with breakthrough bleeding and not necessarily pregnancy, but a secondary contraceptive method is recommended for women using any of the aforementioned agents. In addition, anticoagulants may impair steroid metabolism and increase the side effects of oral contraceptives.

Pharmacogenomics also may play a role in drug interactions with contraceptives, and more information is needed in this area. A history of problems with a particular method in the past can direct the choice of a new method. It is always a good practice to obtain a complete history from a woman regarding medications (both prescribed and OTC agents), botanicals, and nutritional supplements and to verify whether any potential drug interaction exists, as information about such interactions is constantly evolving.

Clinical Evaluation for Women Initiating or Changing a Hormonal Contraceptive Method

This section reviews clinical considerations that apply to all hormonal contraceptives. **Table 17-6** summarizes the components of management for any woman initiating or changing a contraceptive method.

Table 17-6 Care of Women Initiating or Changing a Hormonal Contraceptive Method

Assure that no contraindications exist for the chosen method.
If the method is Category 3 according to the U.S. Medical Eligibility Criteria, discuss both risks and benefits with the woman so she can make an informed choice.
Obtain reasonable assurance that the woman is not pregnant.
Verify that the method to be initiated is one that the woman desires and is likely to use correctly. Because hormonal methods influence menstrual flow, it is wise to explore the woman's feelings about decreased flow, irregular bleeding, and amenorrhea.
Verify the woman is not taking any drugs likely to interact with hormonal contraceptives.
For all women using hormonal contraceptives, review the following points:
Signs or symptoms indicating serious complications (danger or warning signs)
Common side effects
Effect on menstrual flow
Noncontraceptive benefits
Average time for return to fertility
Lack of protection from sexually transmitted infections
Date and time for follow-up appointment
Health promotion or screening activities as appropriate, separate from contraceptive needs
For all women using a short-acting reversible contraceptive (e.g., daily pills, weekly patch) that is woman dependent, review the following points:
When and how to initiate the method
Back-up or secondary protection when indicated (and additional supplies for occasional use if needed)
How to use the method correctly and continuously
Common mistakes to avoid
What to do if she fails to use the method correctly (e.g., missed pills)
Safe disposal of used contraceptives and packages
Specific information regarding prescriptions, including when to be renewed and general cost
Provide opportunities for the woman to ask questions.

Special Considerations for Hormonal Contraception

Adolescents and Young Women

In most locales, women younger than 18 years are considered minors and cannot give consent for care. However, legal provisions typically exist that allow these individuals to obtain contraception if desired. All methods of hormonal contraception can be appropriate for

adolescents. The FDA has a black box warning that states adolescents should not use DMPA longer than 2 years given the effects on bone density at a time when bone mineralization is maximal. This recommendation is not supported by all professional associations but should be considered when discussing this option with an adolescent who is considering use of DMPA. Some noncontraceptive benefits, such as decrease in acne, menstrual bleeding, and dysmenorrhea, associated with several options can be of particular value to adolescents.[15] The sections of this chapter that review the different methods contain information that is generally appropriate to persons in this age group. Moreover, there is no evidence that adolescents should be denied implants or IUDs. Some evidence suggests that depot medroxyprogesterone may be associated with reversible bone loss or lack of bone growth, but it does not preclude its use in younger women.[16] Parental consent may be required before prescribing hormonal contraception for treatment of acne.

Lesbian, Gay, Bisexual, Queer, Transgender Men

When offering contraceptive services for a person who identifies as lesbian, queer, gay, bisexual, queer (LGBQ) or transgender or gender nonconforming (TGNC), one should focus on sexual behaviors not identity. Gender identity does not predict sexual orientation. Data on the number of transgendered youth are elusive, but these individuals are almost certainly underserved. A transgender male is biologically female and should be offered contraception and preventive care as indicated. Although gender affirming hormone therapy (testosterone) limits fertility, it is not a reliable contraceptive.[15,16] Therefore transgender men who have not undergone gender-affirming surgery such as hysterectomy and who are sexually active may desire contraception. Contraceptive methods such as IUDs and implants that tend to promote amenorrhea may provide pregnancy protection as well as provide an added benefit of privacy for these individuals.

Women Who Are Postpartum and/or Lactating

Hormonal contraception is not contraindicated for a healthy postpartum woman, although the timing at which such contraception is initiated or resumed is controversial for postpartum women who are lactating. Additional discussion of this subject can be found in the *Postpartum Care* chapter.

In general, women who recently have given birth, regardless of whether they are breastfeeding, should delay use of contraceptive methods containing estrogen until at least 21 days after the birth because of the increased risk for venous thrombosis resulting from the hypercoagulable state during pregnancy and the early postpartum period. Progestin-only methods, including implants, injections, and pills, may be initiated earlier than estrogen-containing methods because the former are not associated with thrombotic activity. Timing of postpartum use of DMPA is controversial, although some sites initiate the injections within 5 days of birth or abortion/miscarriage.

The American College of Obstetricians and Gynecologists suggests that either an implant or an IUD can be inserted within 10 minutes after birth.[17] Although there is evidence of a higher

rate of expulsion of the latter, this risk may be outweighed by the benefit.[18,19] The insertion procedure immediately postpartum is the same as that used with a nonpregnant uterus, although the midwife should use caution because the uterine isthmus is much softer and more prone to perforation at this time. IUDs also may be placed following spontaneous or elective abortion, either immediately or at 4 to 8 weeks post abortion/miscarriage.[20,21] The only contraindication to immediate postpartum insertion of an IUD is for women with postpartum sepsis, who should wait until the infection has resolved.[13,17]

Effect of BMI on Contraceptive Effectiveness

Some evidence has been published suggesting that hormonal contraception—particularly pills, patches, and implants—may be less effective among women who are obese compared to their thinner counterparts. However, study results are conflicting. Body weight may be more important than body mass index (BMI) calculations in determining the effectiveness of hormonal contraception, perhaps indicating a metabolic effect associated with the amount of adiposity, circulating volume of blood, or increased metabolic rate found among women who are obese. Additional research is needed in this area.

Studies of women using oral contraceptives suggest that women weighing more than 70 kg (154 pounds) may be at an increased risk of contraceptive failure, although this is not a current contraindication for use.[22,23] Women who desire to use the patch should be informed that a body weight of 90 kg (198 pounds) or more is considered a concern, because clinical trials found a higher risk of pregnancy in women in this weight group, even if they were not obese according to their BMI.[24] Based on this finding, the FDA has published a precaution for women of this weight or greater who use the patch contraceptive method.

No evidence exists that hormonal contraceptive methods cause consistent weight gain in general.[22,23] Adolescents who are overweight prior to using DMPA may be more likely to gain weight while using the progestin-only DMPA, as are adult women who are either of normal weight or overweight. Conversely, adult women who are obese are less likely to gain weight when using DMPA.[25] Adolescents who experience a weight gain of more than 5% of total body weight after 6 months of DMPA use are at risk for continued excessive weight gain; counseling about food intake and regular exercise should be provided to these young women. For any woman, if the weight gain becomes excessive, she should consider alternative methods of contraception if she is unable to control her weight through diet and exercise.

Based on pharmacokinetic studies of serum concentration of etonogestrel, women who are 130% of their ideal body weight may be at risk for decreased contraceptive effectiveness when using subdermal implants, although studies have not found such a decrease in clinical practice.[26] Even if hormonal methods are less effective for this population of women, implants and IUDs remain the most effective reversible methods of contraception.

Women with HIV or at Risk for HIV

More than 250,000 women in the United States are living with HIV infection, approximately 11% of whom are unaware of their status.[27] For those who are aware of their HIV-positive

status, contraception often is a paramount concern due to the risk of perinatal transmission. All hormonal methods are classified into category 1 or 2 in the U.S. MEC for Contraceptive Use, even for women taking antiviral agents that have potential drug–drug interactions with COCs, because the benefits of contraception outweigh the risks of drug–drug interactions.[13]

For women who are HIV negative, there is no evidence that any specific method of contraception is associated with an increased risk of HIV acquisition.[28,29] However, all women who use a non-barrier method, such as a hormonal method, should be counseled that their form of birth control does not offer protection from acquiring or transferring the infection.

Women with Disabilities

Care of women with cognitive or physical disabilities should be comprehensive, and no contraceptive method is medically contraindicated for women in these populations. The provider's goal, regardless of the woman's age, should be to conduct care in a manner that shows respect for the woman during an assessment of her abilities, acknowledges her autonomy, promotes her comfort, and listens to her desires.

Women with cognitive disabilities cannot be characterized based on a single stereotype. Many women have mental health conditions; others are intellectually limited to varying degrees. These women often are vulnerable to sexual coercion or assault. In years past, some women were sterilized against their will essentially for eugenic reasons. Today, the topics of sexuality, menstruation, and contraception need to be customized based on the individual's desires and ability to understand and share in decision making.[30]

Physical disabilities do not indicate a lack of sexual desire. Some changes may need to be made in facilities to accommodate provision of care to women with special needs. Women with some physical challenges may have difficulty with use of vaginal rings or even patches. Menstrual suppression may be desired by women with special cognitive or physical disabilities, but selection of a method that achieves that goal should be in concert with the wishes of an individual woman.[31,32]

Women in the Military

It was reported that in 2015, approximately 200,000 women—almost 15% of the total U.S. female workforce—are active-duty military personnel.[33] Contraception should be available to all of these individuals based on Department of Defense policies. Some of these women are deployed in areas where the working situation presents unique circumstances related to contraception. For example, combat areas tend not to stock methods that require regular use, such as pills, injections, or rings. Extremely hot climates may interfere with patch adherence. Implants and IUDs may be of particular use to these women, especially since they can be at risk for sexual coercion or assault and desire effective contraception. The side effect of amenorrhea may also be a benefit in an area where hygiene becomes a challenge.

Women Who Are Incarcerated

According to the U.S. Bureau of Justice, the fastest-growing correctional population in the United States is women. More than 100,000 women are currently incarcerated in local jails, and the overwhelming majority are women who have been victims of sexual abuse.[34] In addition, sexual victimization can occur within correctional facilities. Thus, women who are incarcerated are at risk for both STIs and pregnancy. Although hormonal contraceptives cannot provide protection from infections, LARCS in particular are the most effective reversible methods and should be offered as a contraceptive option.[35]

Women in the Perimenopausal Period

Age is not a contraindication for use of hormonal contraceptive methods. The exception is any prescription of estrogen-containing pills for the woman who smokes more than 15 cigarettes per day and is 35 years or older. Such a woman has a risk of myocardial infarction far greater than her nonsmoking counterpart. In contrast, COCs can safely be used by nonsmokers who are perimenopausal.

Healthy women in their 40s continue to be sexually active and fertile, even if their degree of fertility is less than when they were younger. An early perimenopausal symptom is irregular menses, which may give women false assurance that they are not ovulating and, therefore, not at risk of pregnancy. In reality, escape ovulation and a subsequent pregnancy can occur unpredictably. Clearly, these women need effective contraception if pregnancy is not desired.

Some perimenopausal women opt for permanent sterilization, but some forms of hormonal contraception provide additional non-contraceptive benefits. COCs can provide relief from many perimenopausal discomforts such as irregular bleeding, hot flashes, and dyspareunia. The levonorgestrel IUD (LNG-IUD) has an FDA indication as a therapy for women experiencing heavy menses. Other contraceptive methods associated with the side effect of amenorrhea also may be of value for perimenopausal women. Notably, there is some controversy regarding use of DMPA for this population because of the potential for decreased bone mass density immediately prior to entering the postmenopausal period, a time during which bone loss is more likely.

For the woman who is in the perimenopausal period, hormonal methods usually mask menopause because of the changes in bleeding patterns. No evidence exists to guide the midwife in deciding when it is best to discontinue a hormonal method; however, many clinicians discontinue the hormone contraceptive method when a woman is 55 years old.[36] At that time, the vast majority of woman are postmenopausal. If desired, a woman at that time might consider hormone therapy. The hormone therapy used to treat perimenopausal symptoms belongs to the same drug categories as hormonal contraceptives (estrogen and progestin), but is not of sufficient potency to provide effective contraception.

Initiation of Hormonal Contraceptives

For decades, midwives and other providers delayed initiation of a hormonal contraceptive method until after a woman's next menstrual period to reassure both the provider and the woman that the woman was not pregnant. Timing in tandem with a menstrual period also represented an attempt to minimize breakthrough bleeding, although this connection has not been proven. Some providers advocated starting a method on the first day of menses, others within 5 days of a menses, and yet others within a week of menses. For years, many clinicians advised women to start oral contraceptives on the Sunday after a menses started. This method was promoted as a strategy to prevent subsequent menses from beginning on a weekend. Although all these approaches to initiation were based on opinion and not evidence, they continue to be used in some practices today.

Quick Start or Same-Day Start

In 2002, a study was published advocating a new initiation method called *Quick Start*. Quick Start is the term used to describe an initiation method in which the woman begins taking oral contraceptives as soon as she gets them, regardless of where she is in her menstrual cycle. The original Quick Start technique was designed for combined oral contraceptives and had the woman take the first pill in the office when the pills were prescribed.

Quick Start was developed in response to consumer desires. As many as 25% of women who were provided with a prescription for contraceptive pills never had it filled, often using another, less reliable method or no method of contraception at all.[37,38] Women were found to be more satisfied when they were able to obtain the contraceptive method they desired in a timely manner. In addition, waiting for a menses is a questionable practice often deemed unnecessary because of the improved reliability of pregnancy tests. Even in the rare event that a woman is pregnant and is inadvertently exposed to contraceptive hormones, there is no evidence of an increase in the risk of miscarriage or any teratogenic effects from hormonal contraceptives. Quick Start was also initially promoted as a way to ensure longer continuation rates when compared to traditional approaches, although only limited evidence supports this effect.

The Quick Start practice has since expanded from its first use at the time oral contraceptives are dispensed. Studies suggest it can be used for initiation of patches, rings, and injections as well as pills.[38,17] **Figure 17-2** provides an algorithm for Quick Start and various other hormonal methods.[39]

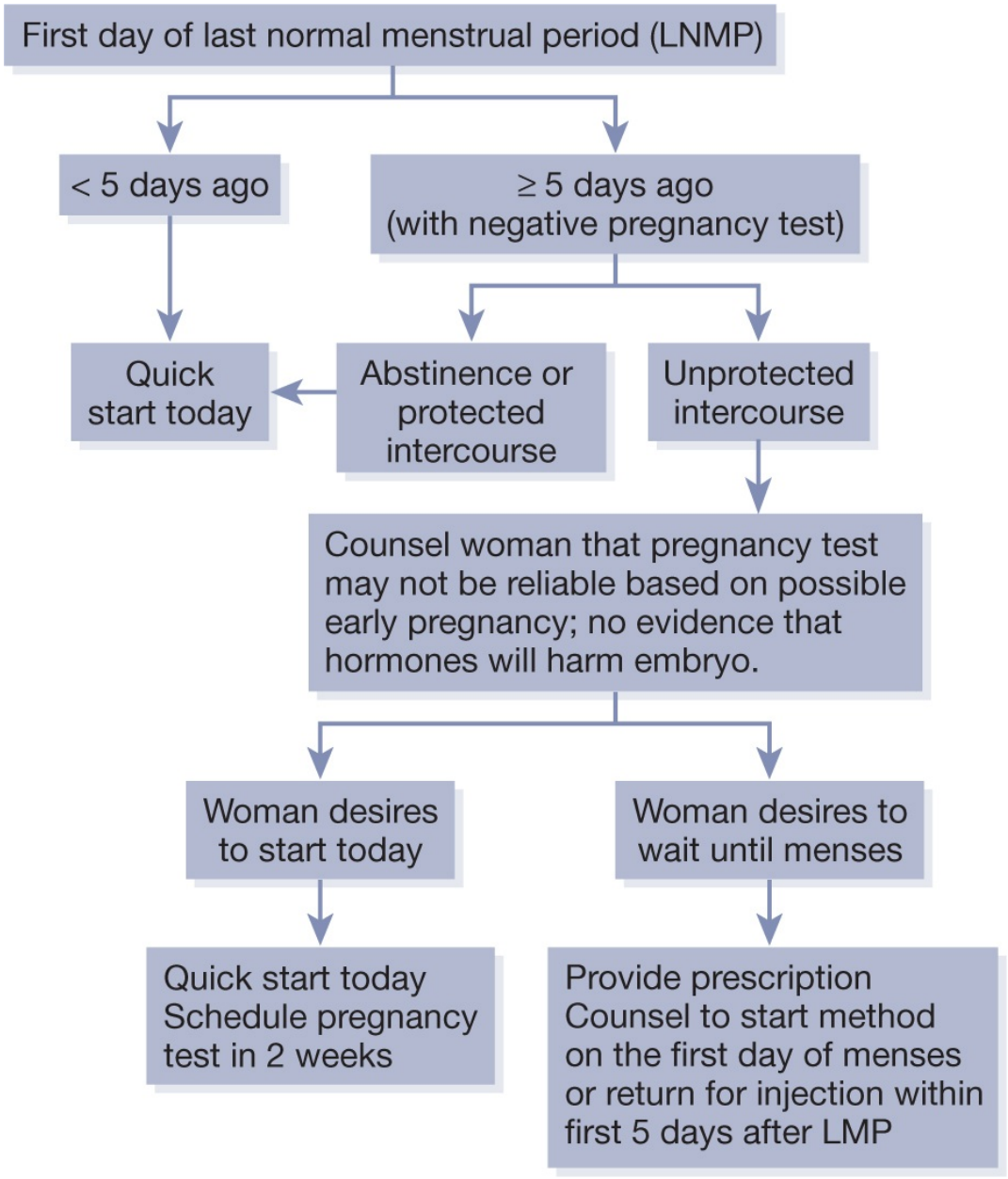


Figure 17-2 Quick Start for pills, patches, rings, and injections.

Modified with permission from The Center for Reproductive Health Education in Family Medicine. Quick Start Algorithm. 2017. www.rhedi.org.[39] Used by permission of RHEDI/Center for Reproductive Health Education in Family Medicine, Montefiore Medical Center, New York City, 2007.

In general, regardless of when a woman begins a hormonal method, there should be some reasonable assurance that she is not pregnant. This assurance can take the form of a normal menses within the last 5 days, a negative pregnancy test, or history indicating that she has not had sexual intercourse since her last menses. Upon initiation of a hormone-based contraceptive method, most women are advised to use a back-up or secondary method until the plasma level of hormone is such that ovulation is suppressed.

Use of Back-up Methods

Evidence regarding the optimal duration of use of back-up contraception is lacking, and most guidelines are empirical at best. Years ago, it was not uncommon to recommend back-up use for a full month after initiation of COCs. Today, a week is more common. Similarly, back-up recommendations for missed pills or patches are not based on scientific studies, but instead represent common clinical practice with extrapolation from some pharmacologic studies.

The suggested back-up method also varies. Some midwives recommend condoms as the back-up, whereas others recommend condoms combined with another spermicidal method such as foam or film. Women who are comfortable with a diaphragm may choose to use that method. As usual, the choice should be based on the considerations outlined in the *Family Planning* chapter and customized to the woman's needs and desires.

Changing from One Hormonal Method to Another

Women may change from one method to another or even discontinue use of contraception for a variety of personal reasons. When a woman desires to switch from one hormonal method to another, her rationale may be based on cost, convenience, desire for increased effectiveness, or a number of other reasons. In this situation, the goal should be to maximize protection from pregnancy. A critical strategy is to avoid any gap between the methods that leaves the woman at risk. Overlapping methods can be a helpful strategy to accomplish this goal. If overlap cannot be arranged, a back-up method can provide temporary contraception. If condoms will be used as the back-up, they offer the added benefit of protection from STIs.

Table 17-7 provides information on how to switch from one hormonal method to another while maintaining maximum protection from pregnancy.[40,41] Women switching from oral contraceptives should follow the guidance in this table and not necessarily finish a package of pills.

Table 17-7 When to Start a New Hormonal Method When Switching from Another Method to Minimize Risk of Pregnancy

Current Method	New Method						
	COC or POP	**Transdermal Patch**	**Vaginal Ring**	**Progestin Injection**	**Implant**	**LNG-IUD**	**Copper IUD**
COC or POP	No gap: take first pill of new pack the day after taking any pill in old pack	1 day before stopping pill	No gap: insert ring the day after taking any pill	7 days before stopping pill	4 days before stopping pill	7 days before stopping pill	Insertion up to 5 days after stopping pills
Transdermal Patch	1 day before stopping patch		No gap: insert ring same day stopping patch	7 days before stopping patch	4 days before stopping patch	7 days before stopping patch	Insertion up to 5 days after stopping patch
Vaginal Ring	1 day before stopping ring	2 days before stopping ring		7 days before stopping ring	4 days before stopping ring	7 days before stopping ring	Insertion up to 5 days after stopping ring
Progestin Injection	Any day up to 15 weeks after injection	Any day up to 15 weeks after injection	Any day up to 15 weeks after injection		Any day up to 15 weeks after injection	Any day up to 15 weeks after injection	Insertion up to 16 weeks after last injection
Implant	7 days before implant removed	7 days before implant removed	7 days before implant removed	7 days before implant removed		Insert 7 days before implant removed	Insertion up to 5 days after implant removed
LNG-IUD	7 days before LNG-IUD removed	7 days before LNG-IUD removed	7 days before LNG-IUD removed	7 days before LNG-IUD removed	4 days before LNG-IUD removed		No gap: insert immediately after LNG-IUD removed
Copper IUD	7 days before IUD removed	7 days before IUD removed	7 days before IUD removed	7 days before IUD removed	4 days before IUD removed	No gap: insert immediately after copper IUD removed and use back-up method for 7 days	

Managing Common Side Effects Associated with Hormonal Contraceptive Methods

Assisting a woman who is experiencing the side effects of any contraceptive is a key role that a midwife can assume to help women optimally use the method. Studies regarding contraceptive use reveal that side effects are the primary reason that the majority of women discontinue their hormonal contraceptive.[9] In addition to counseling women about potential side effects, it is important to include in health education the caveats that not all women will experience these side effects, and that not all discomforts or health concerns experienced while using hormonal contraception will be related to the contraceptive method being used.

Bleeding Irregularities

Changes in menstrual bleeding may be viewed as either an advantage or a disadvantage. Lighter menses generally are well received by most women. Many women are comfortable with amenorrhea, while others are not.[42] The majority of women find unpredictable or breakthrough bleeding to be at least a nuisance. Originally women were counseled that breakthrough bleeding might occur during the first 3 months of use of a hormonal method. However, especially with use of modern low-dose pills, this kind of bleeding may continue for as long as 6 months.

Progestin-only methods are more likely than COCs to be associated with irregular bleeding, including amenorrhea. Strong evidence has not yet emerged regarding how to manage hormone-associated bleeding, although extended and combined regimens have been suggested to suppress menstrual bleeding.[43,44] Many clinicians base their practice in this area on observation or expert opinions. **Figure 17-3** provides a sample algorithm for basic management of a woman with problematic irregular bleeding.[43-45] The pharmacologic treatments included in the algorithm are based on common clinical practices and should not be accepted as evidence based. Most medications provide temporary relief at best.

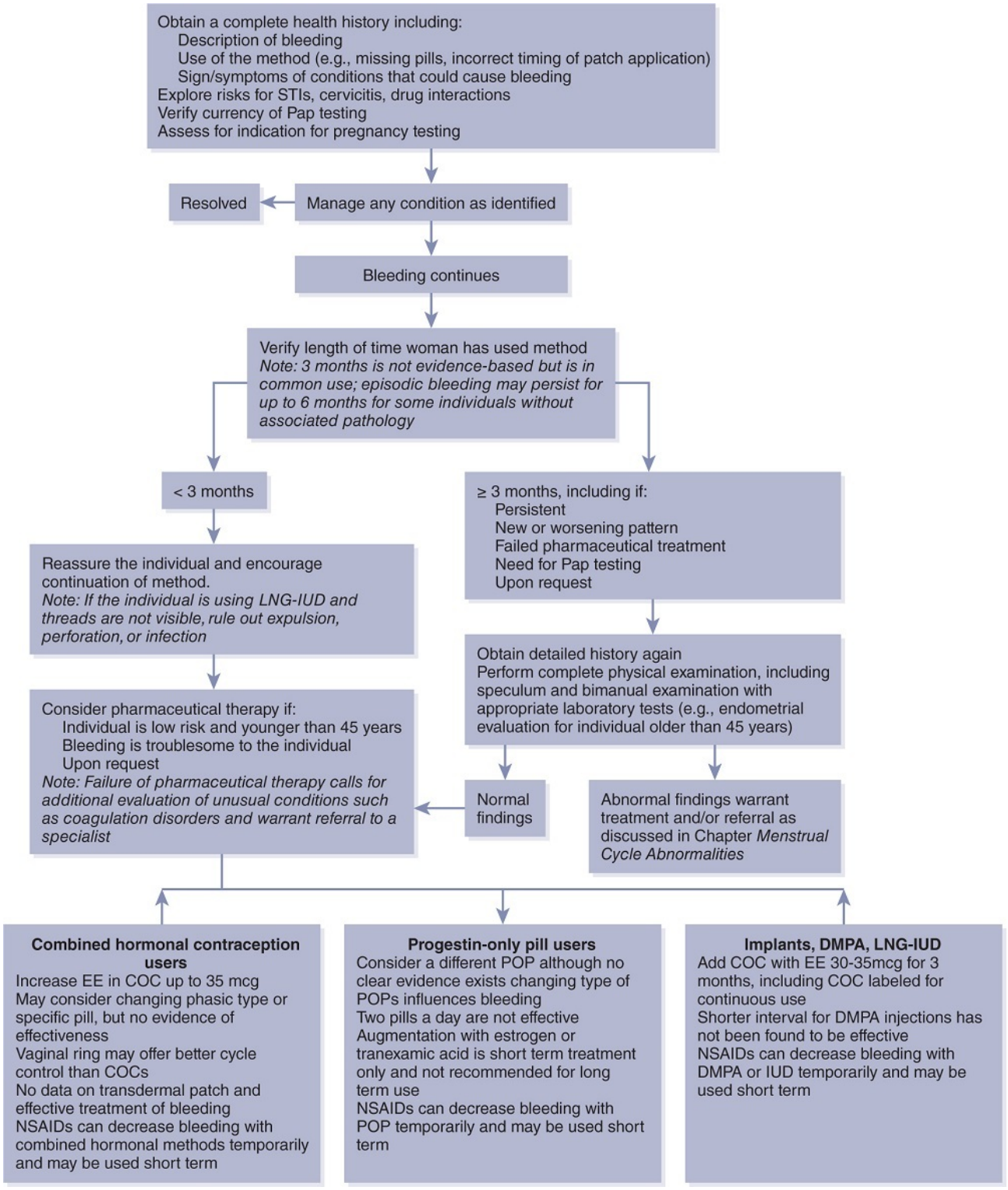


Figure 17-3 Sample algorithm for management of woman using a hormonal contraceptive method and experiencing associated troublesome bleeding.

Abbreviations: COCs, combined oral contraceptives; DMPA, depot medroxyprogesterone acetate; EE, ethinyl estradiol; IUD, intrauterine device; LNG-IUD, levonorgestrel intrauterine device; NSAIDs, nonsteroidal anti-inflammatory drugs; POP, progestin-only contraceptive; STIs, sexually transmitted infections.

[a] Medical therapy is primarily based on expert opinion and common practice. Little strong evidence exists in this area.

Based on Edelman A, Micks E, Gallo MF, Jensen JT, Grimes DA. Continuous or extended cycle vs. cyclic use of combined hormonal contraceptives for contraception. *Cochrane Database Syst Rev.* 2014;7:CD004695. doi:10.1002/14651858.CD004695.pub3[43]; Faculty of Sexual and Reproductive Healthcare Clinical Effectiveness Unit Clinical guidance: problematic bleeding with hormonal contraception–July 2015. Available at: https://www.fsrh.org/standards-and-guidance/documents/ceuguidanceproblematicbleedinghormonalcontraception/. Accessed January 21, 2018;[44] Grossman Barr N. Managing adverse effects of hormonal contraceptives. ***Am Fam Physician.*** 2010; 82(12):1499-1506.[45]

Headaches

Many women have mild occasional headaches, usually associated with tension or allergies. However, for some women, headaches can be disabling and may even herald a severe adverse effect such as a cerebral vascular accident. Some contraceptive methods are associated with increased incidence of headaches, such that the midwife is faced with the perplexing problem of identifying whether the headache is minor or signals a significant healthcare problem. If a woman presents with neurovascular symptoms such as flashing lights, loss of vision, muscle weakness, slurred speech, dizziness, or abnormal cranial nerve changes as well as a headache, these symptoms are an emergency and the woman requires immediate care to rule out a stroke. She also should discontinue the hormonal contraceptive method as quickly as possible and be counseled to use a nonhormonal method until the neurologic condition is resolved. For women who have headaches but no neurologic symptoms, and for whom no abnormalities are found upon examination, the midwife may consider alternative hormonal therapies. For example, women using COCs can have headaches during the withdrawal period (menstrual headaches) and may benefit from an extended-cycle method.[42]

Nausea

Nausea is a common side effect reported by women using any drug, but it is more likely with oral agents and patch contraceptives. There is no simple remedy. No single type of COCs or POPs is superior to another in terms of mitigating nausea. Ultimately, if the nausea and vomiting prove onerous for her, the woman may seek another contraceptive method, especially the contraceptive ring, implant or intrauterine device.[45]

If the nausea is mild but problematic, several suggestions for its management may be made, although evidence is lacking regarding these interventions' effectiveness. Among the most commonly used empirical management strategies for women experiencing nausea with oral contraceptives are taking the pill before sleep and to avoiding ingesting it on an empty stomach. Ginger often is suggested as a food with antinausea effects.

Short-Acting Reversible Contraceptives

Short-acting reversible hormonal contraceptives (SARCs) include pills, injectable agents, transdermal patches, and intravaginal rings that contain either the combination of estrogen and a progestin or only a progestin. Once initiated, the duration of action of these methods can range from a single day to as long as 13 weeks. Advantages of these methods include high rates of effectiveness, relative ease of use, and reduction in dysmenorrhea. Disadvantages of SARCs include the woman's need to take a daily pill or a weekly patch or ring, continued expense, and lack of protection against transmission of STIs.

Combined Oral Contraceptives

COCs produce a pharmacologic—rather than a physiologic—cycle. When the placebo pills are taken, the exogenous estrogen and progestin are "withdrawn" and endometrial wall shedding takes place. This bleeding is sometimes called a "pseudo-menstruation," but the more common term is "withdrawal bleeding," although most women will characterize it as a normal menses or a "period." Typically this bleeding is scant and of shorter duration when compared to a woman's menses associated with normal physiologic events.

The main variations among the formulations of the combined pill relate to the hormonal dose, the relative proportions of estrogen and progestin, and the particular progestin component used. The result is a wide variety of pills, each with a different side-effect profile. Complicating this discussion is the reality that more than 75 brand-name COCs are currently available. In years past, there had been the suggestion that women could be matched to specific pills. Other practices advocated switching certain COCs to others via a complicated process of estimating strength of progestin or dose of estrogen. For most women, however, there is little clinical difference among the formulations.

Regimens for Combined Oral Contraceptives

COCs often are identified according to whether they involve one of three regimens: monophasic, multiphasic, or extended cycle. Different dosing regimens can have various advantages or disadvantages, and one may be preferred over another by a woman.

Monophasic Regimen

The pills in the monophasic regimen come in packs of either 21 or 24 pills that are identical every day in terms of the amount and type of estrogen and progestin. The packs also include either 7 or 4 placebo pills, respectively. Some formulations have replaced the placebo with a folate supplement. One pill is taken each day for a total of 28 days, and then a new pack is begun. Although monophasic COCs have been marketed for decades, the newer regimen of 24 active pills and 4 placebo tablets shortens the duration of withdrawal bleeding and increases effectiveness.

Multiphasic Regimen

The hormone-containing pills in this pack vary in amount of estrogen and/or progestin provided during particular weeks of the cycle of pills and also include either 4 or 7 days of placebo pills. Multiphasic regimens often are referred to as biphasic when two different combinations of estrogen and progestin are provided, or triphasic when the pack has three different combinations or doses. Most multiphasic COCs vary the dose of the progestin, but a few are estrophasic in that they maintain a stable amount of progestin and vary the dose of ethinyl estradiol.

Extended Cycle

For years, women have taken COCs back to back without stopping for a placebo week. The major side effect of this pattern was breakthrough bleeding. Based on women's desire for such convenience, new regimens were introduced. An extended cycle includes hormone and placebo pills, but the hormone pills are taken daily for three consecutive months instead of monthly. For example, in one type of extended-cycle COC, known as Seasonale, the pack includes 84 active hormone pills and 7 placebos. This extended regimen results in menstrual suppression as evidenced by a decreased frequency of withdrawal bleeding and potential amenorrhea. Some of the extended-cycle regimens use pills that contain a reduced dose of ethinyl estradiol instead of placebos, in an attempt to decrease dysmenorrhea and other menstrual-related symptoms.

Special Considerations for Use of Combined Oral Contraceptives

Adolescents can safely use COCs but are more likely than adults to discontinue use if they experience side effects such as nausea; adolescents may also find taking a daily pill more challenging than do older women.[46] Counseling about common side effects, strategies to enhance regular use of the agents such as taking the pill before sleep, and emergency contraception generally are included in health education for all women taking COCs but may be of particular value to young women.

Care of the Woman Requesting Combined Oral Contraceptives

For women requesting to use COCs, the steps in initiating these contraceptives are summarized in **Table 17-6**. Physical examination should include blood pressure and weight, but a pelvic examination is not required. When selecting the specific COC, the lowest dose of estrogen possible should be chosen to minimize risks and side effects. Prescribing four to six cycles for a new user with a return visit to assess the woman's concerns with the method is recommended but may not be needed for all users.

Of all the SARCs, COCs have the largest number of contraindications or Category 3 or 4 considerations in the U.S. MEC for Contraceptive Use.[13] Most of these conditions are significant disorders, especially those associated with cardiovascular events such as hypertension, stroke, headaches with aura, and myocardial infarction. Thrombotic diseases, severe liver disease, breast cancer, lupus, diabetes with neuropathies, and vascular disease also are commonly included in the Category 3 or 4 considerations. The U.S. MEC for Contraceptive Use should be reviewed for any woman who wants to consider COCs as the most efficient way to identify potential contraindications.[13]

Figure 17-4 provides a guide that can be used to counsel women about missed pills. The majority of women, even when using an extended-cycle regimen, will resume normal ovulatory function within 90 days or 3 months of taking the last pill. For years, women have been advised to postpone pregnancy until 3 months after COCs are discontinued to ensure credible dating of a pregnancy. However, there is no evidence that earlier conception is associated with untoward perinatal outcomes.

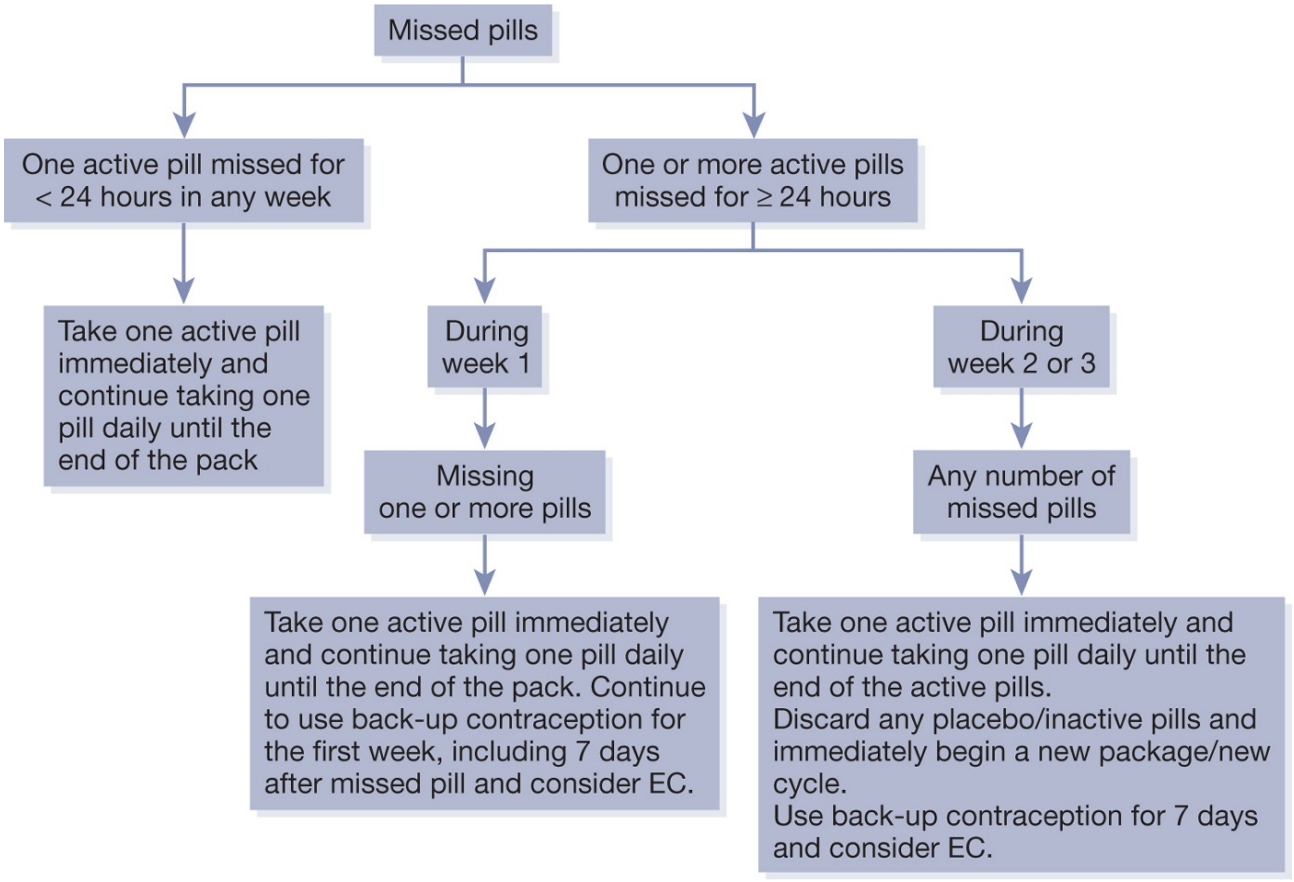


Figure 17-4 Algorithm for counseling women who have missed pills (combined oral contraceptives or progestin-only pills).

Abbreviation: EC, emergency contraception.

Table 17-8 lists content that should be specifically emphasized when counseling women using COCs, in addition to the health education reviewed for any hormonal contraception (Table 17-6).

Table 17-8	**Educational Content to Be Emphasized When Counseling Women Using Combined Oral Contraceptives**[a]
Pills should be taken daily, preferably at approximately the same time. Conditions that should be reported are identified by the mnemonic ACHES: **A**bdominal pain **C**hest pain **H**eadache (severe) **E**ye problems or loss of vision **S**evere leg pain or swelling in a calf or thigh	

[a] In addition to the information in Table 17-6.

Progestin-Only Pills

Progestin-only pills (POPs) contain a low dose of a single progestin and, therefore, can be used by women who want to use contraceptive pills but for whom estrogen is contraindicated. Originally FDA approved in the early 1970s, these agents tend to occupy a niche market for women who are breastfeeding. Although some individuals have called these agents "mini-pills," use of that term can be confusing for women and providers alike because of the number of low-dose COCs available. Therefore, "progestin-only pills" or "POP" is the preferred term. Currently several POPs are available.

Mechanism of Action of Progestin-Only Pills

Because the progestin dose is low, the risk of pregnancy is higher for the average woman using POPs as compared to COCs. To minimize risk of pregnancy, the daily dose must be taken at the same time each day to maintain a steady-state plasma level of the progestin. If more than 24 hours elapses between doses, the plasma level falls and escape ovulation can occur, allowing possible pregnancy if sexual intercourse takes place during that interval.

Special Considerations for Use of Progestin-Only Pills

Although women who are breastfeeding often use POPs, there is conflicting information regarding the amount of progestin transmitted to breast milk. POPs have not been associated with an increased risk of VTE.

Care of the Woman Requesting Progestin-Only Pills

While the management plan for women choosing to initiate progestin-only pills is the same as that for women using COCs, two points should be emphasized. The regularity of the daily dose is unforgiving with POPs; that is, women must take the pills at 24-hour intervals with no more than 3 hours of variation. Second, irregular spotting and bleeding are expected, not simply possible.

Table 17-9 lists topics to be emphasized for the woman regarding progestin-only pill use in addition to the health education required for women using any type of hormonal contraception (Table 17-6). Prescribing 4 months of POPs with a follow-up appointment with the woman in 3 months allows assessment of method satisfaction and review of her experience with side effects, including bleeding irregularities, and her willingness to cope with them. Similar to using COCs, the majority of women using POPs resume ovulatory function within 90 days or 3 months after discontinuing this contraceptive method.

Table 17-9	**Educational Content to Be Emphasized When Counseling Women Using Progestin-Only Contraceptive Pills**[a]
The pill should be taken within 3 hours of the designated time every day.	
If menses continue to be regular and predictable, there should be a suspicion that the woman is ovulating and use of another method or midcycle back-up should be considered.	

Conditions that should be reported are identified by the mnemonic ACHES:

Abdominal pain
Chest pain
Headache (severe)
Eye problems or loss of vision
Severe leg pain or swelling in a calf or thigh

[a] In addition to the information in Table 17-6.

While it is not unusual for women using POPs to have irregular bleeding and amenorrhea, if more than 45 days elapses without any vaginal withdrawal bleeding, the woman should be evaluated for possible pregnancy. Anytime a woman uses POPs and experiences severe abdominal pain, she will need to be evaluated emergently for possible ovarian cyst, ectopic pregnancy, or pelvic inflammatory disease.

Transdermal Contraception (The Patch)

Currently, only one transdermal delivery system for contraception, Xulane, is available in the United States. Ortho Evra was FDA approved in 2002 but withdrawn from U.S. market by the manufacturer in 2015, although it remains available in Canada. Xulane was introduced as a replacement for Ortho Evra; it is the same patch but marketed by a different manufacturer. Xulane is a medicated, adhesive patch with a surface contact area of 14 cm^2. The patch is composed of a thin, lightweight polyester material and constructed in three layers. The outer surface of the patch, called a backing layer, consists of a flexible, beige polyester film and serves as a protective covering. The patch's middle layer contains both the adhesive component and the active drug compounds. A third layer of clear polyethylene film protects the adhesive layer during storage and is removed prior to use.

The contraceptive patch is a combined hormonal method of contraception containing 6 mg norelgestromin and 0.75 mg ethinyl estradiol. Norelgestromin is the primary active metabolite of norgestimate, a progestin commonly used in formulations of some oral contraceptives.

Mechanism of Action of the Contraceptive Patch

The primary mechanism of action for the contraceptive patch is similar to that for the COCs. The transdermal absorption of exogenous estrogen and progestin inhibits ovulation by suppressing release of gonadotropins within the hypothalamic–pituitary–ovarian axis. Alterations in cervical mucus and endometrial lining are secondary mechanisms of contraceptive action. The active drug is absorbed through the skin, thereby avoiding a first-pass effect through the liver. Therefore, this route can decrease the total dose of exogenous hormones delivered when compared to oral contraceptive methods.

The contraceptive patch is self-applied by the woman. Each patch is impregnated with hormones that are adequate for approximately 1 week. To mimic the normal 28-day menstrual cycle, a single contraceptive patch is applied for 7 days and then immediately replaced with a new patch. Three patches are used in a month, resulting in 21 consecutive days of hormone exposure. The fourth week is a patch-free week, and a withdrawal bleed is expected following that interval. The contraceptive cycle is then restarted with the application of a new patch immediately following the 7-day patch-free week. Situations in which the transdermal delivery of the active drugs is disrupted (e.g., partial detachment) can result in decreased effectiveness. However, the adhesive component of the contraceptive patch is not generally affected by heat, humidity, or exercise.

Four application sites have been approved for use with Xulane and have been studied for their therapeutic effectiveness: the buttocks, the upper outer arm, the abdomen, and the upper torso. The area around the breasts is to be excluded, not because of known problems, but because of general concerns about breast tissue being exposed to hormones. Application of the patch to each of these sites has demonstrated therapeutic equivalence by releasing 150 mcg of norelgestromin and 20 mcg of ethinyl estradiol into the bloodstream daily. To avoid problems with adherence/effectiveness, patches should be applied to clean, dry skin and women should be counseled to avoid areas that have been recently exposed to perfumes, gels, lotions,

shampoos, and other topical products unless they clean and dry the skin first.

Special Considerations for Use of the Contraceptive Patch

Contraindications to the use of the contraceptive patch are the same as contraindications for any combined hormonal contraceptive. Side effects include headaches and breast discomfort. There also have been reports of application-site reactions, a non-hormonal-related side effect.

Unlike oral contraceptives, the patch does not require daily maintenance. When dispensed from a pharmacy as a single-month supply, the contraceptive patch is packaged as a set of three individual patches. Even though the patch is marketed for 7 days of use, studies have found that after the second week, a single patch will continue to deliver a sufficient amount of hormones for an additional 2 days, providing some forgiveness at that time should the woman be delayed in making the patch change after week 2 or 3.[14]

The contraceptive patch has received a significant amount of media attention regarding its association with VTE. A Cochrane review estimated that the risk of VTE with patch use is 53 cases per 100,000 women.[47] Other studies have yielded conflicting evidence related to VTE risk in women with contraceptive patch use. The general consensus is that the risk of VTE with the patch is higher than the risk associated with levonorgestrel-containing COCs, but still less than the risk of VTE during pregnancy.

Care of the Woman Requesting Contraceptive Patches

When transdermal patches are requested as the woman's contraceptive method, the midwife should screen the woman for previous skin allergies such as reactions to bandages or other medicated patches. The woman's current weight should also be considered. If her weight exceeds 90 kg (198 pounds), the woman should be advised of the potentially decreased effectiveness of the patch: The package label carries a precaution related to this weight threshold.

It is reasonable to initially prescribe 4 months of contraceptive patches and schedule a follow-up appointment with the woman in 3 months so that if unanticipated problems with detachment occur, the women will have an extra patch. The follow-up visit should include assessment of the woman's satisfaction with the method and experience of any side effects or problems with method use. **Table 17-10** discusses health education specifically related to use of a transdermal contraceptive patch.

Table 17-10	Educational Content to Be Emphasized When Counseling Women Using the Contraceptive Patch[a]
When removing the patch from packaging, the woman should not touch the medicated side. Instead, she should remove only half of the release liner, apply that half to the skin, and then remove the liner for the remaining half, applying with gentle pressure to ensure adhesion.	
Only one patch at a time should be used. Use of multiple patches is more likely to increase side/adverse effects than effectiveness.	
Verify attachment daily. If there is indication that the patch may be detaching, gently press on the patch for approximately 10 seconds. If that effort is unsuccessful, remove the patch and replace it with a new one.	

Patches should not be reused or relocated.

Patches should not be secured by other tape or wrapping.

If the patch becomes nonadherent for < 1 day, remove and apply a new patch. Do not attempt to reapply a patch that has adhered to clothing, as this may decrease effectiveness.

If the patch is off for > 1 day during the 3 weeks, she should apply another patch and start a new 4-week cycle, and use a back-up method for the first week.

Used patches can be discarded in the trash, away from pets and children as they may contain residual hormones. They should never be flushed down a toilet.

[a] In addition to the information in Table 17-6.

The midwife may provide two prescriptions: one for ongoing monthly supply and a second for a single replacement patch in case one is detached. If the woman forgets to apply her second or third patch of the cycle and no more than 1 to 2 days has elapsed since the day she would normally apply the patch (9 days from the application of her current patch), she should apply a new patch as soon as possible (**Figure 17-5**). The original patch change schedule remains unaltered. If more than 9 days has elapsed since the application of the current patch, she should understand that she is not protected from pregnancy and that she should start a new 21-day cycle by applying a new patch and using a back-up contraceptive method for the first week.[14]

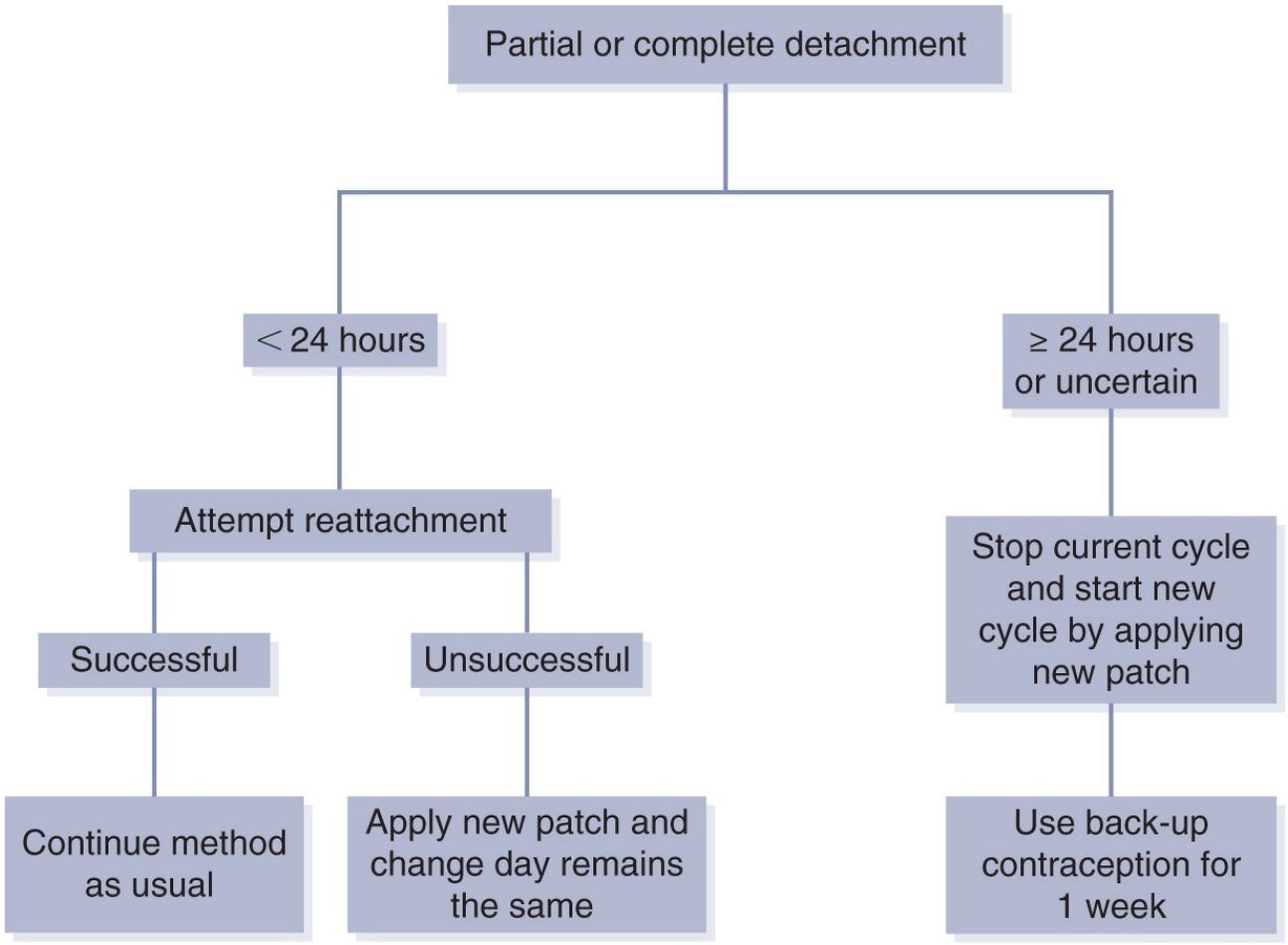


Figure 17-5 Algorithm for management of women with a partial or completely detached transdermal contraceptive patch.

If the woman forgets to take off the patch at the beginning of the patch-free week, she should remove it as soon as she remembers. If more than 9 days has elapsed since placement of the third patch, she should replace the patch according to the original schedule. Depending on how far into the patch-free week she removes the third patch, she may or may not experience a withdrawal bleed, although contraceptive protection is maintained.

If the woman forgets to restart a new patch following the patch-free week, she needs to understand that she is not protected from pregnancy and that she should put on a new patch and use a secondary method for pregnancy prevention for the first week of this new cycle.

Intravaginal Contraceptive Ring

Currently, only one intravaginal delivery system for contraception is available in the United States. NuvaRing, manufactured by Merck Pharmaceuticals, was introduced in the U.S. market in 2001. It is a flexible, vinyl ring measuring approximately 4 mm thick and 54 mm in diameter. The ring is composed of ethylene vinyl acetate, which encases the active components and controls the daily release of the drugs. This combined hormonal method of contraception contains both estrogen and progestin components. The active drugs in the contraceptive ring consist of 11.7 mg etonogestrel and 2.7 mg ethinyl estradiol.

Mechanism of Action of Intravaginal Contraceptive Ring

The primary mechanism of action for the contraceptive ring is similar to that of combined oral contraceptives. The vaginal mucosa absorbs the exogenous estrogen and progestin compounds, inhibiting ovulation via suppression of gonadotropins within the hypothalamic–pituitary–ovarian axis. Alterations in cervical mucus and endometrial lining are secondary mechanisms of contraceptive action. Because the hormones are released directly into the vagina, a lower daily dose of hormones is required in comparison to COCs.

The contraceptive ring is self-administered by the woman, who inserts a ring once per month. Each ring is impregnated with sufficient hormones to last for approximately 21 days. To mimic the normal 28-day menstrual cycle, a single contraceptive ring is inserted and left in place for 21 days. It is then removed for 7 days, thereby inducing a withdrawal bleed. The contraceptive cycle restarts with the insertion of a new ring immediately following the 7-day ring-free week.

The vaginal ring comes in only one size, does not require fitting, and is not position dependent. That is, as long as the ring is in contact with vaginal mucosa, the hormones will be absorbed. The contraceptive ring releases 0.015 mcg of ethinyl estradiol and 0.120 mcg of etonogestrel into the bloodstream daily. The contraceptive ring is intended to be left in the vagina for a continuous period of 21 days; however, if it is out of the vagina for less than 3 hours, contraceptive effectiveness has not been found to be reduced.[10,14]

Special Considerations for Use of the Intravaginal Contraceptive Ring

The most prevalent side effects with the vaginal ring include headache, dysmenorrhea, and breast discomfort. Additionally, a method-specific, nonhormonal side effect is vaginal irritation. When dispensed from a pharmacy, the contraceptive ring comes individually packaged in a resealable foil pouch. NuvaRing is available to be dispensed in boxes of one or three foil pouches. Prior to dispensing, NuvaRing is stored refrigerated, but it can be stored for up to 4 months at room temperature afterward. Temperature extremes should be avoided when storing the contraceptive ring, as there is potential for decreased effectiveness in those situations.

The contraceptive ring is convenient for many women because it does not require daily or weekly maintenance, and its insertion and removal are not difficult. The ring usually is not

perceived by the woman and only occasionally felt by her partner during sexual intercourse. If the woman desires, the contraceptive ring can be removed from the vagina for intercourse; it has been shown to maintain its effectiveness as long as it is replaced within 3 hours. The contraceptive ring should be rinsed only with cool water. Soap, hot water, or other cleansers should not be used on the ring, as they can affect this method's effectiveness.

Contraceptive rings may be of value for adolescents. Not only does this method offer more privacy than oral contraceptives or patches, but it also provides the same benefits as other combined hormonal methods—namely, lighter menses, less dysmenorrhea, no weight gain, and no effect on bone mineral density.

Care of the Woman Requesting an Intravaginal Contraceptive Ring

The management plan for women using the contraceptive ring is similar to that of other combined hormonal contraceptive methods. The midwife should screen for contraindications to combined hormonal contraception.[13] A prescription for emergency contraception should be considered, and women should be encouraged to have spermicide and condoms available in the event that the ring is expelled or an error occurs in removing or starting a new ring on schedule.

It is reasonable for the midwife to initially prescribe 4 months of rings and schedule a follow-up appointment with the woman in 3 months. The follow-up visit is the same as the return visit for any combined hormonal contraceptive and should include assessment of the woman's satisfaction with the method and her experience of any side effects or problems with method use. **Table 17-11** outlines health education points related to the use of the contraceptive ring.

Table 17-11 Educational Content to Be Emphasized When Counseling Women Using the Intravaginal Contraceptive Ring[a]

Remove the ring from its packaging and insert into the vagina by compressing the sides.
The ring must be completely inserted into the vagina.
Insert only one ring at any time. Use of multiple rings is likely to increase side/adverse effects but not effectiveness.
Rings usually are best removed by the woman slipping her index finger under the rim of the ring and guiding it down and out of the vagina.
Because rings should be removed every 21 days, a woman may benefit from using a calendar (hard copy or electronic) as a reminder.
A used ring can be replaced inside its foil pouch and discarded in the trash, away from pets and children. Rings should never be flushed down a toilet.

[a] In addition to the information in Table 17-6.

Spontaneous expulsion occurs in fewer than 3% of women using the contraceptive ring. If the ring is outside of the woman's body for less than 3 hours, then it should simply be rinsed with tepid water and reinserted. If it has been out of her vagina for more than 3 hours, then the woman may not be protected from pregnancy and needs to use a back-up method for 7

consecutive days in addition to reinserting the ring.[14]

If the woman fails to remove the ring and no more than 28 days has passed since she first inserted this ring, she should remove it and insert a new ring. She may or may not experience a withdrawal bleed, but contraceptive protection is maintained. Some women continuously use rings every 21 days, but there is no FDA indication for extended use at this time.

Like the patch, the ring may retain some residual active agent and should be carefully discarded away from children and pets. If more than 28 days has passed since the ring's initial insertion, the woman may not be protected from pregnancy. She should perform a pregnancy test. If the test is negative, she should insert a new ring and use a back-up method for 7 days.

Response to the contraceptive ring has been positive from both women and their partners. Clinical trials are being conducted in other countries for a progestin-only ring, especially to be targeted for lactating women.[48]

Injectable Depot Medroxyprogesterone Acetate Contraceptives

Depot medroxyprogesterone acetate (DMPA) is an injectable hormonal method. Injectable contraception is highly effective, is reversible, does not require partner participation, avoids daily need for intervention, and is not coital dependent.

Mechanism of Action of Depot Medroxyprogesterone Acetate

DMPA is a derivative of progesterone. Its primary mechanism of action is inhibition of ovulation by prevention of follicular maturation. Secondary mechanisms of action include thickening of cervical mucus, which serves as a barrier to sperm, and induction of endometrial atrophy, which reduces the likelihood of implantation. Like other steroidal hormones, DMPA is metabolized by the liver and excreted in the urine.[10]

Two formulations of DMPA are available, both of which require a prescription. The first is DMPA-IM 150 mg/mL (Depot-Provera), which is contained in a 1-mL vial or prefilled syringe and administered every 13 weeks intramuscularly. DMPA is detected in the serum levels within 30 minutes after an injection of 150 mg of this formulation. Serum concentrations vary, but gradually plateau for approximately 3 months, after which the serum concentration gradually declines. The second formulation is DMPA-SC 104 (Depo-subQ Provera 104), an agent administered subcutaneously every 13 weeks that has been found to be equally effective in inhibiting ovulation. The Depo-subQ Provera 104-mg dose and serum levels are approximately 30% lower than those found with the standard DMPA-IM 150-mg dose. This formulation has been shown to have equal, if not improved, tolerability, as side effects of DMPA are dose dependent.

Special Considerations for Use of Depot Medroxyprogesterone Acetate

Allergic reactions are possible but rare, following administration of DMPA-IM 150 and DMPA-SC 104 and a decrease in glucose tolerance has been observed in some women on DMPA. The major and common side effects of DMPA are menstrual changes, irregular bleeding, weight gain, headaches, and delayed return of fertility. The overall incidence of irregular bleeding experienced by women on DMPA can be as high as 70% in the first year of use.[49] Variations usually begin as episodes of unpredictable irregular bleeding and spotting that may last as long as 7 days or more or may be heavy during the first few months. These episodes gradually become less frequent and of shorter duration until the woman has amenorrhea. Approximately 10% to 30% of women experience amenorrhea after one injection, and 40% to 50% do so by the fourth injection. The rates of irregular bleeding and amenorrhea are similar for the subcutaneous and intramuscular formulations.

Weight gain often is noted as a concern among women using DMPA. However, studies of weight gain in users of DMPA have had variable results. The largest systematic review failed to find a consistent association of DMPA with significant increase in weight among users.[50] Nevertheless, it has been reported in some observational trials that women who are obese gain

additional weight, suggesting special attention should be given to counseling for these women.

The use of progestins may cause fluid retention. Certain conditions may, in turn, be exacerbated by this fluid retention, such as headache. Headaches have been reported among DMPA users. Women who develop migraines with aura should stop using DMPA (U.S. MEC for Contraceptive Use Category 3) because aura is a specific risk for stroke. However, studies examining use of natural progesterone or progestin use have found these agents to be associated with a decrease in frequency of migraines.[51] Therefore, a history of headaches is not a contraindication to initiation of DMPA,[13] but women should be counseled to report the development of headaches to their healthcare provider. If a woman develops severe headaches while using DMPA, she should have a thorough evaluation to diagnose the type of headache she experiences.

Long-term DMPA users may develop temporary, but usually reversible, decreased bone density, with declines ranging from 5% to 7% in the hip and spine.[52-55] DMPA suppresses gonadotropin secretion, which in turn suppresses ovarian estradiol production. In this hypoestrogenic state, bone resorption outpaces bone formation, resulting in a decline in bone mineral density. Based on studies of adolescents, bone loss is greatest during the first 2 years of use, but then slows dramatically.[54] Bone density has been shown to return to baseline following discontinuation of DMPA in women of all ages, with this effect being seen as early as 24 weeks after stopping therapy.[55] As the effects of DMPA on bone mineral density are reversible, the lifetime risk of fracture is small.[53] This finding is similar to that with lactational hypoestrogenism, which also has not been associated with an increased risk of fracture. Even so, women with conditions that place them at high risk for osteoporosis and fracture, such as chronic corticosteroid use, disorders of bone metabolism, a strong family history of osteoporosis, or anorexia nervosa, may not be well suited for long-term DMPA use. Likewise, women who have other risk factors that may contribute to lower bone mineral density, such as low calcium intake, alcohol abuse, and lower body mass index, should be evaluated carefully to assess suitability of DMPA use. Inadequate vitamin D levels may also contribute to more substantial bone mineral density loss, particularly among adolescents.[52,54]

Concerns regarding bone loss caused the FDA to mandate a black box warning be placed on the label for DMPA. This warning includes the recommendation that DMPA be limited to a 2-year period of use. However, the labeling also notes that when no other method would be indicated, DMPA can be used. This exception often is employed for adolescents and others for whom other methods would be likely to be less effective.[52,56]

Studies have been inconsistent regarding the effects of DMPA on mood changes. Administration of DMPA in the immediate postpartum period does not appear to predispose women to postpartum depression. If depression occurs or worsens in a woman who is using DMPA as her contraceptive method, her symptoms may persist for the 3-month duration of the drug's effects.

DMPA may cause changes within the vaginal environment and cervical ectopy that can increase susceptibility to STIs, although evidence of such changes is inconclusive. Other potential side effects include nervousness, decreased libido, breast discomfort, dizziness, hair loss, and bloating.[14] Among those women using the subcutaneous formulation, some report an

indentation or induration at the injection site (granuloma or atrophy) or other minor skin reactions.

Return to fertility for women using DMPA tends to take longer than for women using other hormonal methods. Ovulation suppression following DMPA discontinuation is not related to the duration of use, but rather to the weight of the woman. Women with lower body weights may conceive sooner after discontinuation of DMPA than women with higher body weights. If inadvertently used during pregnancy, DMPA is not associated with increased risk of birth defects.

Detectable amounts of the drug have been found in the breast milk of women receiving DMPA; however, no adverse changes in milk composition, quality, or amount have been identified. Likewise, neonates and infants receiving breast milk containing medroxyprogesterone appear to have no adverse developmental and behavioral effects through puberty. Nevertheless, based on theoretical concern for neonatal exposure to exogenous steroids, many providers reserve use of progestin-only contraceptive methods for new mothers until after the first 3–6 weeks postpartum.

Care of the Woman Requesting Depot Medroxyprogesterone Acetate

DMPA can be administered at any time in a menstrual cycle if the woman can be reasonably certain that she is not pregnant. However, if it is given later than the seventh day in the menstrual cycle, the woman should use back-up contraception for 7 days and receive a follow-up pregnancy test several weeks later to confirm the absence of an early pregnancy prior to a second dose of DMPA. If DMPA is initiated within the first 5 to 7 days of the menstrual cycle, a pregnancy test is unnecessary. A pregnancy test is warranted if DMPA is initiated outside of this period. Women can safely continue to use DMPA until menopause.

Fewer absolute (U.S. MEC for Contraceptive Use Category 4) contraindications exist for DMPA compared to COCs, but the relative contraindications (U.S. MEC Criteria for Contraceptive Use Category 3) include several major conditions as well as unexplained vaginal bleeding. The complete list of contraindications is available through the U.S. MEC for Contraceptive Use.[13]

DMPA is injected either subcutaneously or intramuscularly, depending on the formulation. Massaging the injection site can lower the effectiveness of the DMPA. Ovulation does not occur for at least 14 weeks post injection. If the woman is more than 2 weeks late for her subsequent dose, a pregnancy test is recommended prior to administration to assure she is not pregnant. Various methods of appointment reminders are available.

Starting at 13 weeks post injection, a secondary contraceptive method is recommended until the next dose is given. Conversely, subsequent injections may be administered up to 10 weeks after the previous DMPA injection without adverse effects. **Table 17-12** provides important information for women regarding DMPA injections.

Table 17-12 Educational Content to Be Emphasized When Counseling Women Using Depot Medroxyprogesterone Acetate[a,b]

Women should eat a diet high in calcium and vitamin D. If not, a woman should consider taking a daily multivitamin

supplement.

Although such outcomes are rare, a woman should be aware of and report any signs or symptoms of infection at the injection site, such as a fever, or changes at the insertion site, including drainage, redness, and warmth.

Other conditions that should be reported to a healthcare provider:

- Significant headaches
- Menorrhagia
- Depression
- Severe lower abnormal pain or any other signs or symptoms that may suggest pregnancy

[a] In addition to the information in Table 17-6.

[b] Education counseling is primarily based on expert opinion and common practice. Little strong evidence exists in this area.

Long-Acting Reversible Contraceptives

Long-acting reversible contraceptives tend to be more effective than their short-acting counterparts because they are not user dependent. LARCs include implants and IUDs.[10,13] Once inserted, these methods convey contraceptive protection for years. Although these methods tend to have a higher initial expense, their long-term use tends to make them cost-effective.

Subdermal Implants

Implantable contraceptives consist of one or more matchstick-sized rods or tubes containing progestin. These implants are inserted beneath the top layers of skin, on the inner aspect of the upper arm. Depending on the model of implant, subdermal devices can provide contraception for up to 7 years.

The first subdermal implant available in the United States was Norplant. It consisted of six plastic tubes containing levonorgestrel, a progestin. In 2002, the manufacturer removed Norplant from the U.S. market, citing limited availability of product components. Although Norplant is not currently available in the United States, it continues to be used internationally and a woman may have it inserted outside the country and desire to retain it for 7 years.

Although more implants may be available in the future, the only subdermal implant currently available in the United States is an etonogestrel-containing, single-rod device. Nexplanon entered the U.S. market in 2012, replacing its predecessor Implanon. Implanon and Nexplanon are biologically equivalent to each other.

Mechanism of Action of Subdermal Implants

Nexplanon is a polyethylene vinyl rod, 4 cm in length and 2 mm in diameter. The device is impregnated with 68 mg of etonogestrel, a progestin analogue, as well as 12 mg of barium sulfate. This latter substance makes the device detectable by X ray. Nexplanon works by continuously releasing etonogestrel to suppress ovulation. A single Nexplanon implant provides contraception for up to 3 years.

Special Considerations for Use of Subdermal Contraceptive Implants

As with most long-acting contraceptive devices, the initial costs of subdermal implants are significantly higher when compared to the costs of SARCs. However, when averaged over 3 years of use, the total costs of subdermal implants usually make these options less expensive than COCs, POPs, or DMPA. At the time of implant removal, a new device can be reinserted in the same site, thereby providing continuing contraception.

Care of the Woman Requesting a Subdermal Contraceptive Implant

Based on an agreement between the manufacturer of Nexplanon and the FDA, the implants are available for dispensing only to providers who have completed a 3-hour training session focusing on identifying women who are candidates for the implant as well as health counseling, insertion, and removal. Individuals who have taken Implanon training previously can update their training online. Therefore, this chapter will not address specifics on insertion or removal.

A few contraindications exist for contraceptive implants. All contraindications can be found in the U.S. MEC for Contraceptive Use.[13] No specific physical or laboratory assessment is recommended before insertion, with the exception of being reasonably certain that the woman is not pregnant. Monitoring for detrimental side effects, menstrual pattern, blood pressure, and user satisfaction can be done at a follow-up visit approximately 3 months following insertion.

Limited data are available regarding the use of the implants by breastfeeding women, but small amounts of etonogestrel have been observed in breast milk. **Table 17-13** lists health education topics for women using the subdermal implant.

Table 17-13	Educational Content to Be Emphasized When Counseling Women Using Subdermal Contraceptive Implants[a]
Although rare, a woman should be aware of and report the following: Signs or symptoms of infection at the insertion site, such as a fever Changes at the insertion site, including drainage, redness, and warmth	

[a] In addition to the information in Table 17-6.

When a woman wishes to discontinue use of the method, the removal of the implant is usually performed easily during an office visit. Often this is scheduled at the 3-year mark, but some women may desire to continue with the method. In that case, a new implant can be placed into the small incision made for removal.

In spite of adequate education, a midwife may occasionally find it difficult to remove an implant. Removal, however, is never an emergency. The first action should be to verify placement by ultrasound imaging to confirm that the implant is still present as well as to determine its location. On rare occasions, the device may inadvertently be placed very deep (< 2 cm). Should such a situation occur, midwives are encouraged to consult with or refer women to a specialist, especially those at one of the “Family Planning Fellowship” training sites around the country. There the contraceptive specialist will collaborate with interventional radiologists to remove the implant, and other surgeons are available as needed.

Hormonal Intrauterine Devices

Two types of IUDs exist: nonhormonal and hormonal. The chapter *Nonhormonal Contraception* introduces the use of the intrauterine contraceptive device (IUCD). These devices share multiple commonalities and that general information can be found in the aforementioned chapter. For example, most adverse effects, timing for insertion, and removal are the same and found in the Intrauterine Device Insertion and Removal appendix in the Nonhormonal Contraception chapter. This chapter focuses only on specifics of the levonorgestrel IUD, the hormonal device.

Mechanism of Action of Levonorgestrel Intrauterine Device

The levonorgestrel IUD (LNG-IUD) is a T-shaped, flexible polyethylene device widely marketed in the United States. The body of the device includes a reservoir that contains levonorgestrel, a progestin. The LNG-IUD provides for a continuous release of levonorgestrel, which is absorbed locally by the endometrium and results in increased viscosity of cervical mucus, remodeling of the endometrial lining, and impaired tubal motility. As a result of these localized effects, sperm motility is impaired, thereby inhibiting fertilization of the ovum. A possible secondary mechanism of action is suppression of ovulation.

The LNG is a LARC contraceptive method, which is cost-effective when used over a period of years. Women who have an aversion to checking for the device strings after each menstrual period may not desire this method. A few women are intolerant of IUD-associated side effects such as bleeding changes, and others simply do not like the idea of having a foreign object inside their body.

In 2000, the FDA approved the first LNG-IUD, which was marketed with the brand name Mirena. The Mirena IUD releases an average of 20 micrograms of levonorgestrel per day and provides contraception for up to 5 years. This device measures 33 mm by 32 mm wide. Nine years after the initial approval, the FDA approved an additional indication for this device—namely, its use as a therapeutic agent for women with menorrhagia. Subsequent studies have found that the LNG-IUD is more effective than oral therapy and, although not as effective in reducing bleeding as a hysterectomy, more cost-effective than the surgery.[57] When compared to the copper IUD, discussed in the *Nonhormonal Contraception* chapter, an IUD with levonorgestrel is associated with decreased bleeding, decreased dysmenorrhea, and increased incidence amenorrhea. The commonly accepted contraindications for this type of IUD include uterine anomalies, acute pelvic inflammatory disease, pregnancy, breast cancer, and unexplained bleeding.

Today, four different LNG-IUDs are available in the United States (**Table 17-14**). These devices contain different amounts of levonorgestrel and have different FDA-approved indications in terms of years of use. No specific guidelines exist regarding how to match a woman to a particular IUD. Mirena and Liletta, however, are approximately 2–4 mm larger than the others and might be more easily tolerated by a parous woman. These two LNG-IUDs release higher levels of daily levonorgestrel, and may be associated with more amenorrhea, which may or may not be desired.[58] Because of the dosing, they may be indicated for women

with heavy bleeding. Kyleena and Mirena provide the longest duration of action. As with all methods, cost, availability, and personal desires of women must be considered when selecting an LNG-IUD.

Table 17-14 Intrauterine Devices Containing Levonorgestrel

Brand Name	Total Dose of LNG in Device (mg)	Initial Release of LNG (mcg/day)	Size	Years of FDA Approval[a]
Liletta	52	18.6	32 mm horizontal and vertical	4
Kyleena	19.5	17.5	28 mm horizontal and 30 mm vertical	5
Mirena	52	20	32 mm horizontal and vertical	5
Skyla	13.5	14	28 mm horizontal and 30 mm vertical	3

Abbreviations: FDA, U.S. Food and Drug Administration; LNG, levonorgestrel.

[a] FDA approval may change, given that ongoing studies tend to suggest longer-term use is effective.

Special Considerations for Use of Levonorgestrel Intrauterine Device

The IUD insertion and removal procedures carry some risks, even though they are only minimally invasive. Bleeding, infection, perforation of the uterus, and pain can occur, although serious complications are rare. While IUDs do not cause ectopic pregnancy, if fertilization occurs with an IUD in place, there is a greater risk of ectopic implantation. It is important to underscore, however, that the overall risk of ectopic pregnancy for a woman with any IUD is lower than the risk of ectopic pregnancy for a woman not using contraception.

The Intrauterine Device Insertion and Removal appendix in the *Nonhormonal Contraception* chapter provides detailed information about insertion and removal of both nonhormonal (copper) IUDs as well as the four types of LNG-IUDs. Information about pre-insertion medication, STI screening, timing of insertion, and immediate adverse effects also are found in the appendix. Management of a woman with a pregnancy and an IUD in situ, expulsion of the device, and associated ectopic pregnancies are the same for copper and LNG-IUDs and are discussed in detail in the Nonhormonal Contraception chapter.

Care of the Woman Requesting a Levonorgestrel Intrauterine Device

Most women, including adolescents and nulliparas women, are good candidates for intrauterine contraception.[13] Category 3 and 4 contraindications are outlined in the U.S. MEC for Contraceptive Use, and include current pelvic inflammatory disease, breast cancer, endometrial cancer, severe cirrhosis, suspicion of intrauterine disease including chlamydia, gonorrhea, and tuberculosis. Women should not have an IUD inserted immediately postpartum or post abortion if sepsis has occurred.

Table 17-15 lists content to be reviewed when counseling women using an LNG-IUD.

Table 17-15 Educational Content to Be Emphasized When Counseling Women Using an LNG-IUD[a]

Women should verify that the device is in situ by feeling for strings at least monthly.

Use of the mnemonic PAINS can provide a reminder for women regarding conditions that should be reported to the midwife:

Period—late or abnormal bleeding (although this is relatively common for these women)

Abdominal pain or dyspareunia

Infection, especially associated with exposure to STI or an abnormal vaginal discharge

Not feeling well, especially if woman experiences fever, chills, or generalized malaise

Strings missing or inability to feel the plastic thread of the device

[a] In addition to the information in Table 17-6.

Conclusion

Over the last half-century, hormonal contraceptives have expanded from a drug category that included only combined pills to a repertoire of effective methods such as progestin-only pills, transdermal patches, intrauterine devices, vaginal rings, and implants. Hormonal contraception, like nonhormonal methods, is not a “one size fits all” situation: Women should choose the option that represents the best fit for their unique personal circumstances. A woman’s decision should consider her desires, her own eligibility, the conceptive method’s effectiveness, noncontraceptive benefits, and the woman’s ability to use the method correctly and consistently.

Resources

Organization	Description	Webpage
Centers for Disease Control and Prevention (CDC)	U.S. Medical Eligibility Criteria (U.S. MEC) for Contraceptive Use	http://www.cdc.gov/mmwr/pdf/rr/rr59e0528.pdf
	Selected Practice Recommendations (SPR) for Contraceptive Use	https://www.cdc.gov/mmwr/volumes/65/rr/rr6504a1.htm eBook: https://www.cdc.gov/reproductivehealth/contraception/ebook.html
Monthly Prescribing Reference (MPR)	Monographs, charts, and app that list all current oral contraceptives by brand name and their components	http://www.empr.com/download-empr/section/753/

References

1. Christin-Maitre S. History of oral contraceptive drugs and their use worldwide. *Best Pract Res Clin Endocrinol Metab*. 2013;27(1):3-12.
2. Beasley WBR. After office hours, coping with family planning in a rural area. *Obstet Gynecol*. 1973;41:155-159.
3. Kelly PJ, Cheng AL, Carlson K, Witt J. Advanced practice registered nurses and long-acting reversible contraception. *J Midwifery Womens Health*. 2017;62(2):190-195.
4. Potter L, Oakley D, deLeon-Wong E, Canamar R. Measuring compliance among oral contraceptive users. *Fam Plann Perspect*. 1996:28:154-158.
5. Likis FE. Contraceptive applications of estrogen. *J Midwifery Womens Health*. 2002;47(3):139-156.
6. Brucker MC, Likis FE. Steroid hormones. In: Brucker MC, King TL. *Pharmacology for Women's Health*. Burlington, MA: Jones & Bartlett Learning; 2017:355-375.
7. Benagiano G, Gabelnick H, Brosens I. Long-acting hormonal contraception. *Womens Health (Lond)*. 2015;11(6):749-757.
8. de Bastos M, Stegeman BH, Rosendaal FR, et al. Combined oral contraceptives: venous thrombosis. *Cochrane Database Syst Rev*. 2014;*3:CD010813. doi:10.1002/14651858.CD010813.pub2*.
9. Trussell J, Guthrie KA. Choosing a contraceptive: efficacy, safety and personal considerations. In: Hatcher RA, Trussell J, Nelson AL, Cates W, Kowal D, Policar MS, eds. *Contraceptive Technology*. 20th ed. New York, NY: Ardent Media; 2011:45-75.
10. Speroff L, Darney P. *A Clinical Guide for Contraception*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.
11. American College of Obstetricians and Gynecologists. Practice Bulletin No. 110: noncontraceptive uses of hormonal contraceptives. Obstet Gynecol. 2010;115:206-218. [Reaffirmed 2016].
12. Hilliard PA. Menstrual suppression: current perspective. *Int J Womens Health*. 2014;6:631-637.
13. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(3):1-103. Available at: https://www.cdc.gov/reproductivehealth/contraception/pdf/summary-chart-us-medical-eligibility-criteria_508tagged.pdf. Accessed August 27, 2017.
14. Murphy PA, Elmore CE. Contraception and reproductive health. In: Brucker MC, King TL. *Pharmacology for Women's Health*. Burlington, MA: Jones & Bartlett Learning; 2017:863-900.
15. American College of Obstetricians and Gynecologists. Committee Opinion No. 539: adolescents and long-acting reversible contraception: implants and intrauterine devices. *Obstet Gynecol*. 2012;120:983-988.
16. American College of Obstetricians and Gynecologists. Committee Opinion No. 710: counseling adolescents about contraception. *Obstet Gynecol*. 2017;130:e74-e80.
17. American College of Obstetricians and Gynecologists. Committee Opinion No. 670: immediate postpartum long-acting reversible contraception. *Obstet Gynecol*. 2016;128:e32-e37.
18. Sothornwit J, Werawatakul Y, Kaewrudee S, Lumbiganon P, Laopaiboon M. Immediate versus delayed postpartum insertion of contraceptive implant for contraception. *Cochrane Database Syst Rev*. 2017;4:CD011913. doi:10.1002/14651858.CD011913.pub2.
19. Lopez LM, Bernholc A, Hubacher D, Stuart G, Van Vliet HAAM. Immediate postpartum insertion of intrauterine device for contraception. *Cochrane Database Syst Rev*. 2015;6:CD003036. doi:10.1002/14651858.CD003036.pub3.
20. Patil E, Darney B, Orme-Evans K, et al. Aspiration abortion with immediate intrauterine device insertion: comparing outcomes of advanced practice clinicians and physicians. *J Midwifery Womens Health*. 2016;61(3):325-330.
21. Moniz MH, Roosevelt L, Crissman HP, et al. Immediate postpartum contraception: a survey needs assessment of a national sample of midwives. *J Midwifery Womens Health*. September 7, 2017. [Epub ahead of print]. doi:10.1111/jmwh.12653.
22. Lopez LM, Bernholc A, Chen M, et al. Hormonal contraceptives for contraception in overweight or obese women. *Cochrane Database Syst Rev*. 2016;8:CD008452. doi:10.1002/14651858.CD008452.pub4.
23. Lopez LM, Ramesh S, Chen M, et al. Progestin-only contraceptives: effects on weight. *Cochrane Database Syst Rev*. 2016;8:CD008815. doi:10.1002/14651858.CD008815.pub4.
24. Audet MC, Moreau M, Koltun WD, et al. Evaluation of contraceptive efficacy and cycle control of a transdermal contraceptive patch vs an oral contraceptive: a randomized controlled trial. *JAMA*. 2001;285(18):2347-2354.
25. Bonny A, Secic M, Cromer B. Early weight gain related to later weight gain in adolescents on depot medroxyprogesterone acetate. *Obstet Gynecol*. 2011;117(4):793-797.
26. Xu H, Wade JA, Peipert JF, Zhao Q, Madden T, Secura GM. Contraceptive failure rates of etonogestrel subdermal implants in overweight and obese women. *Obstet Gynecol*. 2012;120(1):21-26.
27. Centers for Disease Control and Prevention. Diagnoses of HIV infection in the United States and dependent areas, 2015. *HIV Surveill Rep*. 2015;27. Available at: https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-report-2015-vol-27.pdf. Accessed January 6, 2017.
28. American College of Obstetricians and Gynecologists. Practice Bulletin No. 167: gynecologic care for women and

adolescents with human immunodeficiency virus. *Obstet Gynecol.* 2016;128:e89-e110.
29. Centers for Disease Control and Prevention. Update to CDC's *U.S. Medical Eligibility Criteria for Contraceptive Use, 2016*: revised recommendations for the use of hormonal contraception among women at high risk for HIV infection. *MMWR*. 2017;66(37);990-994.
30. Fouquier KF, Camune BD. Meeting the reproductive needs of female adolescents with neurodevelopmental disabilities. *J Obstet Gynecol Neonatal Nurs*. 2015;44(4):553-563.
31. Abells D, Kirkham YA, Ornstein MP. Review of gynecologic and reproductive care for women with developmental disabilities. *Curr Opin Obstet Gynecol*. 2016;28(5):350-358.
32. American College of Obstetricians and Gynecologists. Committee Opinion No. 668: menstrual manipulation for adolescents with physical and developmental disabilities. *Obstet Gynecol.* 2016;128(2):e20-e25.
33. U.S. Veterans Administration. *Women Veterans Report: The Past, Present, and Future of Women Veterans.* Washington, DC: National Center for Veterans Analysis and Statistics, Department of Veterans Affairs; 2017. Available at: https://www.va.gov/vetdata/docs/SpecialReports/Women_Veterans_2015_Final.pdf. Accessed September 29, 2017.
34. U.S. Bureau of Justice. Fact sheet on women offenders—2016. Available at: https://www.csosa.gov/newsmedia/factsheets/statistics-on-women-offenders-2016.pdf. Accessed September 29, 2017.
35. American College of Obstetricians and Gynecologists. Committee Opinion No. 535: reproductive health care for incarcerated women and adolescent females. *Obstet Gynecol*. 2012;120(2 pt 1):425-429.
36. ESHRE Capri Workshop Group. Female contraception over 40. *Hum Reprod Update*. 2009;15(6):599-612.
37. Westhoff C, Kerns J, Morroni C, Cushman LF, Tiezzi L, Murphy PA. Quick Start: novel oral contraceptive initiation method. *Contraception*. 2002;66(3):141-145.
38. Lopez LM, Newmann SJ, Grimes DA, Nanda K, Schulz KF. Immediate start of hormonal contraceptives for contraception. *Cochrane Database Syst Rev.* 2012;12:CD006260. doi:10.1002/14651858.CD006260.pub3.
39. The Center for Reproductive Health Education in Family Medicine. Quick Start Algorithm. 2017. www.rhedi.org.
40. Lesnewski R, Prine L, Ginzburg R. Preventing gaps when switching contraceptives. *Am Fam Physician*. 2011;83(5):567-570.
41. Reproductive Health Access Project. How to switch birth control methods. June 2015. Available at: http://www.reproductiveaccess.org/wp-content/uploads/2014/12/switching_bc.pdf. Accessed January 6, 2018.
42. Jacobson JC, Likis FE, Murphy PA. Extended and continuous combined contraceptive regimens for menstrual suppression. *J Midwifery Womens Health*. 2012;57(6):585-592.
43. Edelman A, Micks E, Gallo MF, Jensen JT, Grimes DA. Continuous or extended cycle vs. cyclic use of combined hormonal contraceptives for contraception. *Cochrane Database Syst Rev.* 2014;7:CD004695. doi:10.1002/14651858.CD004695.pub3.
44. Faculty of Sexual and Reproductive Healthcare Clinical Effectiveness Unit. Clinical guidance: problematic bleeding with hormonal contraception–July 2015. Available at: https://www.fsrh.org/standards-and-guidance/documents/ceuguidanceproblematicbleedinghormonalcontraception/. Accessed January 21, 2018.
45. Grossman Barr N. Managing adverse effects of hormonal contraceptives. *Am Fam Physician*. 2010;82(12):1499-1506.
46. Westhoff C, Heartwell S, Edwards S, Zieman M, Stuart G, Cwiak C. Oral contraceptive discontinuation: do side effects matter? *Am J Obstet Gynecol.* 2007;196:412.e1-412.e6.
47. Lopez LM, Grimes DA, Gallo MF, Stockton LL, Schulz KF. Skin patch and vaginal ring versus combined oral contraceptives for contraception. *Cochrane Database Syst Rev.* 2013;4:CD003552. doi:10.1002/14651858.CD003552.pub4.
48. RamaRao S, Clark H, Merkatz R, Sussman H, Sitruk-Ware R. Progesterone vaginal ring: introducing a contraceptive to meet the needs of breastfeeding women. *Contraception*. 2013;88:591-598.
49. Haider S, Darney P. Injectable contraception. *Clin Obstet Gynecol.* 2007;50:898-906.
50. Lopez LM, Edelman A, Chen M, et al. Progestin-only contraceptives: effects on weight. Cochrane Database Syst Rev. *2013;4:CD008815. doi:10.1002/14651858.CD008815.pub2*.
51. Martin VT, Behbehani M. Ovarian hormones and migraine headache: understanding mechanisms and pathogenesis—part 2. Headache. 2006;46:365-369.
52. American College of Obstetricians and Gynecologists. Committee Opinion No. 602: depot medroxyprogesterone acetate and bone effects. *Obstet Gynecol.* 2014;123:1398-1402.
53. World Health Organization. *Hormonal Contraception and Bone Health: Provider Brief*. Geneva, Switzerland: World Health Organization, Department of Reproductive Health and Research; 2007.
54. Scholes D, Lacroix A, Ichikawa L, Barlow W, Ott S. Change in bone mineral density among adolescent women using and discontinuing depot medroxyprogesterone acetate contraception. *Arch Pediatr Adolesc Med*. 2005;159:139-144.
55. Harel Z, Johnson C, Gold M. Recovery of bone mineral density in adolescents following the use of depot medroxyprogesterone acetate contraceptive injections. *Contraception*. 2010;81(4):281-291.
56. Kaunitz AM. Long-acting hormonal contraceptives—indispensable in preventing teen pregnancy. J Adolesc Health. 2007;40:1-3.

57. Lethaby A, Hussain M, Rishworth JR, Rees MC. Progesterone or progestogen-releasing intrauterine systems for heavy menstrual bleeding. *Cochrane Database Syst Rev.* 2015;4:CD002126. doi:10.1002/14651858.CD002126.pub3.
58. Nelson AL. LNG-IUS 12: a 19.5 levonorgestrel-releasing intrauterine system for prevention of pregnancy for up to five years. *Expert Opin Drug Deliv.* 2017;14(9):1131-1140.

CHAPTER

18

Midlife, Menopause, and Beyond

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Introduction

Midlife encompasses approximately one-third of a modern woman's life. Most individuals entering midlife are healthy. A person's midlife and older years are characterized by multidimensionality and complexity, including both negative and positive aspects of the normal life-course transition process toward aging.[1] The term *midlife* and its synonym *middle age* lack a standard definition. In general, midlife is characterized as the years from the 40s to late 50s. Midlife encompasses a time of changing hormones, relationships, and roles. The only universal "change" for women is the final cessation of menstruation; other aspects of the midlife and beyond are highly individualized.

The influence of scientific, technological, and healthcare sources for the modern midlife and older individuals, however, can result in women being more vulnerable to messages that "medicalize" aging. Many can benefit from a knowledgeable guide who can assist them *as individuals*, navigating the opportunities and challenges of the menopausal transition and beyond.[2] Partnering with individuals to provide information about the changes surrounding aging, nonpharmacologic and pharmacologic options to manage bothersome symptoms if needed,[3] and lifestyle modifications to optimize health during the many years after menopause are within the scope of midwifery practice today.

Physiology of Midlife, Menopause, and Aging

Menopause is the permanent cessation of menstruation that occurs after the ovaries cease to produce follicles. The median age of menopause in North America is 51 years. During this period of life, numerous physiologic changes occur, including in the ovaries, endocrine milieu, and cardiovascular and skeletal systems, as well as emotional changes. The ovaries and the endocrine system change over the course of a woman's lifetime. During fetal life, the ovaries contain approximately 1 to 2 million follicles—the maximum number of follicles a woman will ever have.[4] The loss of follicles eventually leading to menopause occurs primarily due to atresia, not ovulation, because most women will ovulate fewer than 500 times during their reproductive years.[5]

As a woman ages, the rate of follicular loss increases. In turn, as the number of follicles diminishes, the ovaries become more resistant to the action of follicle-stimulating hormone (FSH).[4] The ovaries also decrease production of estrogen, androgen, and progesterone. Loss of the negative feedback from ovarian estrogen production means that gonadotropin production is no longer inhibited. Subsequently, levels of FSH and luteinizing hormone (LH) rise markedly, to the point that the FSH level exceeds the LH level. Secretion of the ovarian glycoprotein inhibin, which selectively inhibits FSH, also decreases. The decreased release of inhibin eventually results in sustained elevation of FSH. Elevated FSH levels stabilize approximately 24 months after the final menstrual period.[4] Although levels of these hormones generally trend upward over time throughout this process, they can fluctuate. These unexpected, erratic changes provide the rationale for not routinely testing hormone levels as a method to diagnose menopause.

After menopause, the ovary changes considerably both in physical appearance and in function. In spite of the absence of functional follicles, both the remaining corticostromal cells and the hilar cells are steroidogenic; as such, they contribute to androgen production and continue to provide significant amounts of androstenedione and testosterone for several years. These hormones influence a woman's muscle strength and sexual drive.[6]

A postmenopausal woman's ovaries become small and are normally not palpable on bimanual examination. Thus, if a postmenopausal woman's ovary is palpated, an ovarian neoplasm must be ruled out.[4]

During the postmenopausal period, the cervix gradually decreases in size and produces less mucus; this change can contribute to vaginal dryness and dyspareunia. Progressive atrophy of the cervical epithelium creates an increasingly sparse capillary bed and makes the surface of the cervix appear smooth, shiny, and pale.[4]

After ovarian follicular activity ceases, hormonal stimulation of the endometrium and myometrium also dissipates. Endometrial tissue, regardless of its location, becomes atrophic and inactive in postmenopausal women. Thus, not only does the uterine lining become atrophic, but small to moderate-size fibroids, which are highly sensitive to estrogen stimulation, usually shrink after menopause, as do the cyst-like invasions of the myometrium characteristic of adenomyosis.[7]

The three main human estrogens are estrone (E_1), estradiol (E_2), and estriol (E_3).

Postmenopausally, estrone accounts for the majority of circulating estrogen; it is derived principally from the metabolism of estradiol and from the conversion of androstenedione in adipose tissue. Conversely, estradiol—the most potent of the three estrogens—accounts for 95% of the circulatory estrogen in premenopausal women. The dominant follicle and the corpus luteum both excrete large amounts of estradiol in reproductive-age women.[4] Estriol, a weak estrogen, is secreted from the placenta but can also be metabolized in small amounts from estrone.[6] Postmenopausal women have serum estradiol levels lower than 37 pg/mL (picograms/milliliter) and mean estrone levels between 6 and 63 pg/mL. In contrast, premenopausal women have estradiol levels in the range of 10–100 pg/mL in the early follicular phase, 200–800 pg/mL at midcycle, and 200–340 pg/mL during the luteal phase, as well as premenopausal estrone levels in the range of 30–180 pg/mL. Although 95% of postmenopausal androstenedione production occurs in the adrenal gland and 5% occurs in the ovaries, the ovarian stroma continues to produce androstenedione and testosterone under the influence of LH. These hormones, along with the androstenedione produced by the adrenal glands, are converted to estrone in peripheral adipose tissue. Thus, increased conversion to estrone is associated with a higher body mass index (BMI) or increasing weight in postmenopausal women.[4] Some estradiol continues to be produced, but the amount is significantly less than that produced in reproductive-age women.

Menopause

Although menopause is a universal experience for women as they age, menopause may be influenced by diet, environment, cultural beliefs, and genetics. Women may have few, if any, bothersome symptoms when menopause occurs naturally. By comparison, when menopause is induced by surgery, chemotherapy, or radiation therapy, symptoms are more likely to be troublesome.[4]

The experience of menopausal symptoms is culturally determined to a great degree. Generalizing from the experiences of one group of women to other groups can lead to misunderstandings and suboptimal care.[8] In many cultures, menopause is a welcome change that heralds a period of wisdom, renewal, and energy. Midwives can provide women with balanced information about menopause in modern society that can help foster positive attitudes toward this period of life.[9]

Terms and Definitions for Menopause

As noted in **Table 18-1**, the definition of menopause is based on a retrospective diagnosis that is accurate only 12 months after a woman's last menstrual cycle. **Table 18-1** includes other terms frequently used to describe aspects during this period of a woman's life.[4,6,10-14] A number of the terms that relate to menopause are imprecise, but continue to be used colloquially.

Table 18-1 Definitions Related to Menopause

Term	Definition
Climacteric	Process of age-related change from a reproductive state to a nonreproductive state. Sometimes used interchangeably with *perimenopause*. In some regions, this term is considered too vague for use and has fallen out of favor.
Early menopause	Natural or induced menopause that occurs long before the average age of spontaneous menopause. This imprecise umbrella term encompasses premature menopause. The most frequently used cut-off age for early menopause is 40 years, but this is not standardized.
Estrogen therapy (ET)	Treatment of women for menopausal symptoms such as vasomotor conditions or vulvovaginal atrophy.
Estrogen plus progestin therapy (EPT)	Similar to *estrogen therapy* but with added progestin to decrease the risk of endometrial hyperplasia or cancer.
Final menstrual period (FMP)	The last menstrual period of a woman's life; it is recognized after 12 months of amenorrhea has passed. The average age at FMP in the Western world is 51 years (range: 40–58 years).
Hormone therapy (HT)	Primarily use of estrogen with or without an added progestin. Also known as *menopausal hormone therapy (MHT).* The older terms "hormone replacement therapy" (HRT) and "estrogen replacement therapy" (ERT) have fallen out of favor because "replacement" implies that the pharmacologic agent provides premenopausal hormonal levels. In reality, HT provides only a small fraction of the hormone levels produced by premenopausal ovaries. Moreover, "replacement" connotes an unnatural deficiency state when the changed endogenous hormone levels that a menopausal woman experiences are actually natural and expected.
Induced menopause	Cessation of menstruation due to surgical removal of a woman's ovaries or ablation of ovarian function from chemotherapy or pelvic radiation therapy. Also known as *surgical menopause*.
Menopause	A one-time event that marks permanent cessation of ovulation and menstruation. It is diagnosed when a woman has had 12 months of amenorrhea with no other identified cause such as pregnancy.
Menopause transition	A time of menstrual and endocrine changes, beginning with variation in cycle length and ending with the FMP. Average age at onset is 46 years (range: 39–51 years). Average duration of transition is 5 years (range: 2–8 years).
Perimenopause	Symptomatic years of menopausal transition, encompassing the time from early menopausal transition to 12 months after the FMP. Experts disagree about whether this term should be reserved for use with the public or if it is suitable for use in scientific literature. It is similar to the time of menopause transition. This term is often used synonymously with *climacteric.*
Postmenopause	The period of time after a woman's final menstrual period.
Premature menopause	Any menopause that occurs in women younger than 2 standard deviations below the mean estimated age for the reference population. The age frequently used as a benchmark is 40 years.
Primary ovarian insufficiency	Ovarian failure leading to amenorrhea in women younger than 40 years. Sometimes used synonymously with *premature ovarian failure*. Some authorities prefer premature ovarian

	insufficiency rather than premature ovarian *failure*, because cessation of ovarian function is not always permanent.
Progesterone	A steroid hormone secreted by the corpus luteum and by the placenta. This term is most commonly used to describe women's naturally occurring hormone.
Progestin	A natural or synthetic substance that mimics the actions of progesterone. This term is most commonly used to describe the therapies women use for contraception and menopausal hormone therapy.

Based on North American Menopause Society. *Menopause Practice: A Clinician's Guide.* 5th ed. Mayfield Heights, OH: North American Menopause Society; 2017[4]; Fritz MA, Speroff L. Menopause and perimenopausal transition. In: *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:673-748[6]; Hale GE, Robertson DM, Burger HG. The perimenopausal woman: endocrinology and management. *J Steroid Biochem Molec Biol.* 2014;142:121-131[10]; Harlow SD, Gass M, Hall JE, et al. Executive summary of the Stages of Reproductive Aging Workshop + 10: addressing the unfinished agenda of staging reproductive aging. *Fertil Steril.* 2012;97:843-851[11]; Singer D. "It's not supposed to be this way": psychological aspects of a premature menopause. *Counsel Psychother Res*. 2012;12(2):100-108[12]; Pickar JH, Archer DF, Kagan R, Pinkerton JV, Taylor HS. Safety and benefit considerations for menopausal hormone therapy. *Expert Opin Drug Saf.* 2017;16(8):941-954[13]; Fritz MA, Speroff L. Postmenopausal hormone therapy. In: *Clinical Gynecologic Endocrinology and Infertility.* 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:749-858.[14]

Stages of Reproductive Aging

Because chronological age is not an accurate predictor of a woman's path to menopause, leading menopause researchers developed an alternative system for assessing women's stages of life at the Stages of Reproductive Aging Workshop (STRAW). Investigators published the initial system in 2001, research continued during the next 10 years, and the updated STRAW + 10 system was published in 2012. This system has become the gold standard for describing reproductive aging through menopause.[11] **Figure 18-1** provides a graphic representation of the STRAW + 10 stages. Use of this staging system can aid clinicians and women in understanding the expected physiologic changes that occur during the menopausal transition. The stages progress from a woman's reproductive years toward her final menstrual period and then beyond.

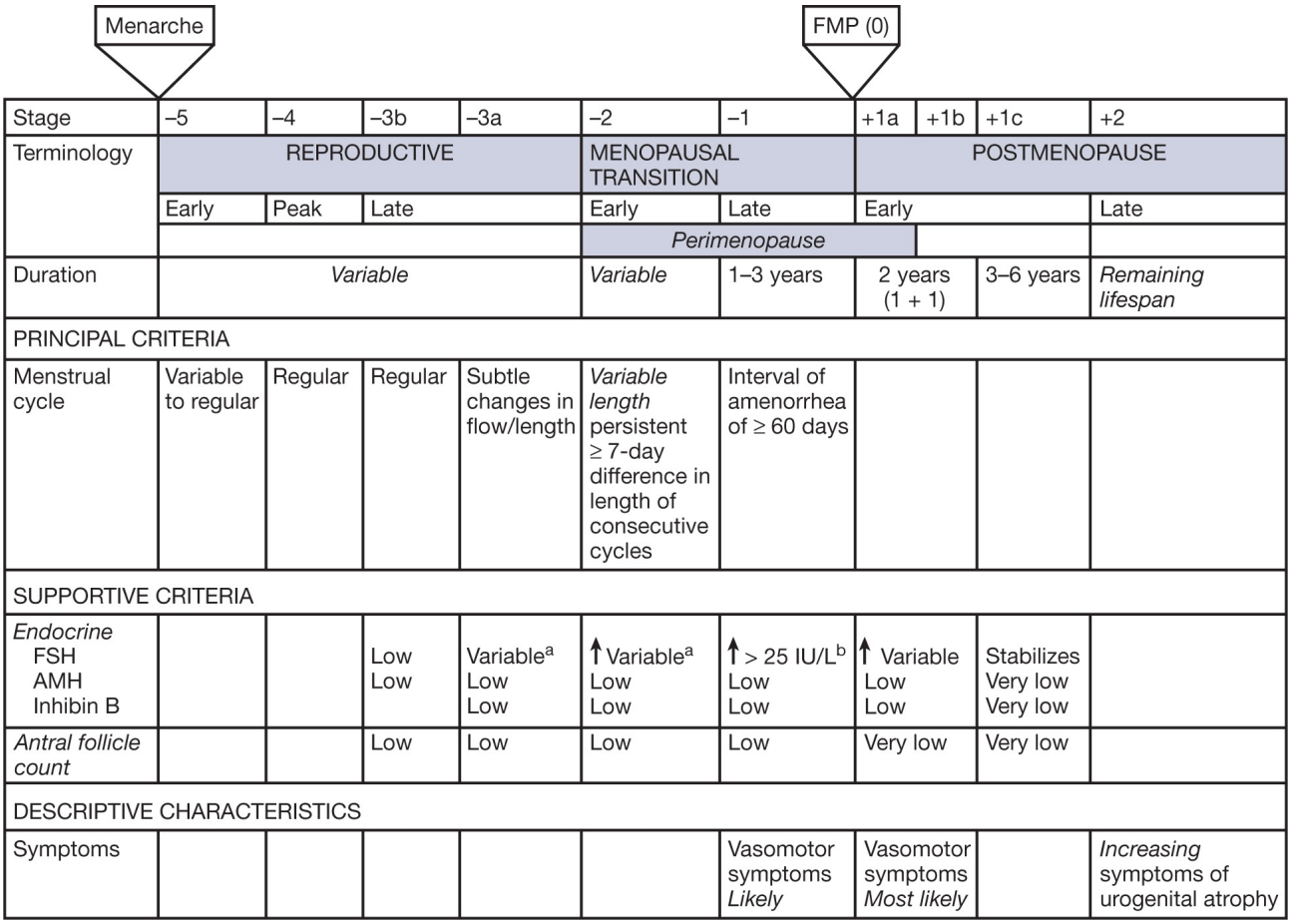


Figure 18-1 The Stages of Reproductive Aging Workshop + 10 staging system.

Abbreviations: ↑ , elevated; AMH, anti-Müllerian hormone; FMP, final menstrual period; FSH, follicle stimulating hormone.

[a] Blood draw on cycle days 2–5.

[b] Approximate expected level based on assays using current international pituitary standard.

Reproduced with permission from Harlow SD, Gass M, Hall JE, et al. Executive summary of the Stages of Reproductive Aging Workshop + 10: addressing the unfinished agenda of staging reproductive aging. *Fertil Steril*. 2012;97:843-851.[11] Used with permission of Elsevier, Ltd.

Late Reproductive Stage (Stage –3b)

During the late reproductive stage, women begin to notice changes in the amount of menstrual flow and in cycle length, and they often have shorter cycles. FSH levels usually increase, but can vary from one cycle to the next. Importantly, even irregular or long cycles can still be ovulatory. In as many as 25% of these cycles, women may ovulate[7]; thus, women may conceive a pregnancy during this phase.

Early Menopausal Transition (Stage –2)

As women move closer to their final menstrual period, menstrual cycle length becomes even more variable. In this stage, there is often a difference of 7 days or more in the length of consecutive cycles, and these variations persist for at least 10 cycles after the first variable-length cycle. The FSH level remains elevated but, once again, varies. Estradiol levels remain in the normal range but may even be slightly elevated until approximately 1 year before the cessation of follicular development. This finding is contrary to earlier research that indicated estradiol levels gradually waned in the years before menopause.[6]

Late Menopausal Transition (Stage –1)

During the late menopausal transition stage, women generally are amenorrheic for spans of 60 days or longer, have wide deviations in cycle length, exhibit marked fluctuations in hormone levels, and are increasingly anovulatory. In this stage, which usually lasts 1 to 3 years, women frequently experience vasomotor symptoms. The conclusion of the late menopausal transition phase is the final menstrual period (FMP).

Early Postmenopause (Stages +1a, +1b, and +1c)

The FSH level continues to increase and estradiol levels continue to decrease until approximately 2 years after the FMP. Early postmenopause is subdivided into three distinct stages:

- *Stage +1a* usually lasts 1 year, and its end marks the 12-month period required to make the retrospective diagnosis of spontaneous menopause.
- *Stage +1b* lasts approximately 1 year and is characterized by rapid changes in FSH and estradiol levels. Symptoms such as hot flashes are most likely to occur in this stage.
- *Stage +1c* lasts approximately 3 to 6 years, during which women experience a period of stabilization with high FSH and low estradiol levels.

Late Postmenopause (Stage +2)

The late postmenopause stage encompasses a woman's remaining lifespan; with today's life expectancy, this time period may account for one-third of her life or more. Hormone levels stabilize during this stage, and symptoms associated with low estrogen levels, such as vulvovaginal atrophy, can become increasingly apparent.

Smoking, lower body weight, nulliparity, never-use of oral contraceptives, and lower socioeconomic status are factors that have been found to be associated with spontaneous menopause occurring earlier than the average age of 51 years.[15] Some experts consider higher body weight to be associated with later age at menopause,[16] but others argue that this association is not clear.[17] Family history can also affect a woman's age at her final menstrual period.[15]

Diagnosing Menopause

No single laboratory test can predict or confirm menopause. Instead, the diagnosis of menopause is based on a woman's menstrual and medical history and on her reported symptoms.[4] Thus, a diagnosis of menopause is generally assured if a woman has been amenorrheic for 12 months and if there is no reason to suspect an underlying alternative cause of the symptoms.

Hormone testing to predict menopause or help manage symptoms is not usually of clinical value. **Table 18-2** lists potential indications for obtaining hormone levels and problems associated with hormone testing.[4,6] As with any test, hormone testing should be ordered only if the results can affect treatment options offered to the woman.

Table 18-2 Hormone Testing During Perimenopause and Menopause

Hormone	Possible Reasons to Obtain Test	Problems Associated with Test
Estrogen levels	To assess absorption of estrogen in women with persistent vasomotor symptoms in spite of hormone therapy	Estradiol and estrone levels are erratic; isolated levels are unlikely to give meaningful information
Progesterone	To document ovulation in perimenopausal women who are trying to conceive	Levels likely to be low in anovulatory cycles; isolated levels are unlikely to give meaningful information
Follicle-stimulating hormone (FSH)	Multiple FSH levels demonstrating sustained elevation > 30 mIU/mL, or months of amenorrhea and FSH > 40 mIU/mL, can provide a reasonably sound diagnosis of menopause; sometimes useful for a woman with an unclear or unusual clinical presentation	Highly variable during perimenopause: even elevated FSH levels (> 30 mIU/mL) can return to the premenopausal range days, weeks, or months later
Luteinizing hormone (LH)	If a woman taking hormonal contraceptives needs laboratory confirmation of menopause, a serum FSH:LH ratio greater than 1 on the seventh pill-free day can provide a reasonably sound diagnosis of menopause	LH elevation occurs late in perimenopause—much later than FSH elevation; minimal or no utility in confirming perimenopause or menopause
Prolactin	To rule out a pituitary cause for oligomenorrhea or amenorrhea, particularly in the presence of galactorrhea	Prolactin levels do not vary with aging or menopause
Thyroid-stimulating hormone (TSH)	To rule out thyroid dysfunction as a cause for symptoms otherwise attributed to perimenopause and menopause (e.g., abnormal uterine bleeding, hot flashes, sleep difficulties, fatigue, weight gain)	Thyroid levels do not vary with aging or menopause
Testosterone	To rule out testosterone excess state from testosterone treatment or endogenous excess	Levels change little from 4 years before to 2 years after menopause; little value in diagnosing testosterone insufficiency because testosterone levels are only one part of total androgen status

Based on North American Menopause Society. *Menopause Practice: A Clinician's Guide*. 5th ed. Mayfield Heights, OH: North American Menopause Society; 2017[4]; Fritz MA, Speroff L. Menopause and perimenopausal transition. In: *Clinical Gynecologic Endocrinology and Infertility.* 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:673-748.[6]

Hallmark Symptoms of the Perimenopausal/Menopausal Period

The severity of symptoms during the perimenopausal and menopausal periods varies dramatically from woman to woman. As such, the management of these symptoms is individualized based on symptom severity and the woman's choice of treatment options.

Bleeding Pattern Changes

Approximately 90% of women experience 4 to 8 years of menstrual changes before their final menstrual period.[4] Exactly what those changes entail for an individual woman is unpredictable and can differ from menses to menses. The most common pattern is a gradual decrease in both the amount and duration of the menstrual flow, leading to vaginal spotting and then to cessation of bleeding. However, some women experience more frequent or heavier periods during the perimenopause period and occasionally the heavy bleeding is troublesome and occasionally pathologic. This type of abnormal uterine bleeding (AUB) is discussed in detail in the *Menstrual Cycle Abnormalities* chapter. **Table 18-3** compares normal and potentially abnormal bleeding patterns during the perimenopausal period.[4]

Table 18-3 Normal Versus Potentially Abnormal Bleeding Patterns During Perimenopause

Common Perimenopausal Menstrual Changes	Potentially Abnormal Perimenopausal Bleeding Patterns[a]
Shorter intervals between periods (but at least 21 days)	Very heavy bleeding, especially with clots
Longer intervals between periods	Long bleeding duration (> 7 days or ≥ 2 days longer than usual)
Lighter bleeding	Very short cycles (< 21 days from beginning of one period to beginning of next period)
Heavier bleeding than normal (difficult to quantify; consider evaluation if significant change)	Irregular bleeding: spotting or bleeding between periods
Shorter duration of bleeding	Bleeding after sex
Longer duration of bleeding (up to 1–2 days longer than usual)	
Skipped periods	
No changes	

[a] Any bleeding that occurs after menopause (i.e., after 12 months of amenorrhea) should merit investigation of a pathologic cause.

Based on North American Menopause Society. *Menopause Practice: A Clinician's Guide.* 5th ed. Mayfield Heights, OH: North American Menopause Society; 2017.[4]

Genitourinary Syndrome

At least half of postmenopausal women experience some external genital, urinary, and sexual changes reflective of the hypoestrogenic state that occurs during menopause (**Table 18-4**).[17,18] The term genitorurinary syndrome of menopause replaces older terms used such as

vulvovaginal atrophic changes and atrophic vaginitis.[17,18] The vagina, vestibule, and bladder trigone have a high concentrations of estrogen receptors. When estrogen levels diminish, physiologic and anatomic changes occur in these organs.[17,18] The vaginal and cervical epithelium gradually becomes thin, dry, and pale in a diminished-estrogen environment. This tissue sometimes becomes inflamed, and a visible capillary bed may appear as a diffusely red appearance or small petechial hemorrhages. During the perimenopausal/postmenopausal period, the vagina transitions from the acidic environment dominated by lactobacilli to an alkaline environment with more diverse flora that can include pathogenic organisms.[4] In addition, rugae disappear and the vaginal secretions diminish.[4]

Table 18-4 Signs and Symptoms Associated with Genitourinary Syndrome of Menopause

	External genitalia	Sexual	Urologic
Physiologic Changes	General: thinning and graying of pubic hair Labia: atrophy and labia minor absorbed, loss of hymeneal remnants, introitus retracts Vagina: reduced secretions, decreased elasticity in vagina, loss of rugae, pallor, petechiae or erythema, friability	Decreased arousal, orgasm, desire lubrication	Urethral eversion Prominence of urethral meatus
Signs and Symptoms	Alkaline pH Genital dryness Postcoital bleeding Vaginal burning or itching	Bleeding or spotting with intercourse Pain with penetration	Dysuria, frequency or urgency Nocturia Postvoid dribbling
Associated Conditions	Pelvic organ prolapse Vaginal or introital stenosis	Dyspareunia Dysorgasmia	Meatal stenosis Recurrent urinary tract infections Stress or urinary incontinence Urethral polyp Urethral prolapse

Based on Portman D. Genitourinary syndrome of menopause: new terminology for vulvovaginal atrophy from the International Society for the Study of Women's Sexual Health and the North American Menopause Society. *Menopause*. 2014;21(10):1063 -1068[18]; Gandhi J, Chen A, Dagur G, et al. Genitourinary syndrome of menopause: an overview of clinical manifestations, pathophysiology, etiology, evaluation, and management. *Am J Obstet Gynecol*. 2016;215(6):704-711.[19]

Arousal time slows with aging for both women and men, allowing more time for foreplay during sexual activity is advisable. Sexual activity itself improves blood supply to the pelvis and the vaginal tissues. Consequently, sexually active older women often have less atrophy than older women who are not sexually active.[4] Women troubled by genitourinary syndrome symptoms occasionally report dryness or itching of the vulva and vagina and pain upon vaginal penetration. In such women, the midwife may find micro-abrasions and few or absent vaginal

rugae when a pelvic examination is performed. Urinary changes include prominence of urethral meatus, frequency, dysuria, nocturia, and possibly incontinence.

Vasomotor Symptoms

Vasomotor symptoms are extremely common in the perimenopausal period, although the exact prevalence is difficult to determine. Studies have provided different statistics on this phenomenon, with estimates of the percentage of women reporting hot flashes ranging from 8% to 80%.[20] Regardless of the precise rate of occurrence, hot flashes—like changes in the pattern of bleeding—are so common that they are considered a hallmark of perimenopause. Ethnicity, diet, climate, lifestyle factors, and women's attitudes about aging may all influence an individual's experience of vasomotor changes. In North America, African American women report hot flashes more frequently than Hispanic, white, or Asian women.[21] Women who have undergone surgically induced menopause have a higher incidence and more severe hot flashes, at least for the first year, compared to women having naturally occurring menopause.[4] Women who are younger at the time of menopause tend to have more frequent vasomotor symptoms.[11]

The physiology of vasomotor symptoms remains poorly understood,[6] and the terms *vasomotor symptoms*, *hot flashes*, *hot flushes*, and *night sweats* all refer to the same experience. This phenomenon is characterized by recurrent, transient periods of flushing, sweating, and a sensation of heat, often accompanied by palpitations and a feeling of anxiety, and sometimes followed by chills.[4] These flashes can occur during a woman's sleep, causing her to wake and describe the event as a night sweat.

A single hot flash usually lasts 1 to 5 minutes, during which time a woman experiences a sudden wave of heat that soon spreads over her body and particularly over her face and upper torso. Elevations in skin temperature, heart rate, skin blood flow, and metabolic rate follow quickly. As the hot flash ends, skin temperature begins to gradually return to normal and the woman sometimes feels chilled, often due to heat loss from sweating and continued peripheral vasodilation.[21]

Although a woman's precise risk of experiencing vasomotor symptoms cannot be predicted, some general risk factors have been identified. Numerous studies have associated cigarette smoking with increased risk of hot flashes. Women who have discontinued smoking for 5 years or more report less severity and frequency of hot flashes, compared to current smokers.[22] Previous research indicated that women with a low BMI and minimal adipose tissue experience more hot flashes, which may be due to a decrease in endogenous estrogen production. More recent evidence indicates that adipose tissue can act as an insulator, which promotes a higher core body temperature (supportive of hot flashes), although more research is needed in this area.[4]

The highest prevalence of vasomotor symptoms occurs within the first 1 to 2 years postmenopause, yet many women report experiencing hot flashes shortly before the final menstrual period. Approximately 50% of women have hot flashes for 4 to 5 years after their final menstrual period, 25% have hot flashes for more than 5 years, and 10% for as long as 15 years.[6] A small number of women report having hot flashes for decades after menopause. Some

women have relatively few hot flashes, whereas other women have very frequent hot flashes. Moreover, some women interpret these symptoms as easily managed, whereas other women report that flashes are disrupting their lives.

Women who have mild hot flashes often find relief with common sense and relatively easily instituted lifestyle changes. Women may implement the lifestyle changes listed in **Table 18-5**; if they find that these interventions are adequate, women may not require medications.[21,22,24,25]

Table 18-5	Lifestyle Interventions Used to Treat Vasomotor Symptoms
Intervention	**Comments**
Avoidance of hot flash triggers	A symptom diary to identify triggers, and thereby help the woman avoid them, and to increase personal feelings of control may be of value.
Discontinue smoking	Women who smoke tend to have more hot flashes than women who do not; in addition, the more a woman smokes, the greater her risk for more severe vasomotor symptoms.
Maintain healthy weight and fitness level	Although evidence is mixed regarding whether poor exercise habits and obesity have a causal relationship with vasomotor symptoms, these strategies may be of value and have positive effects on health in general.
Promote a cool environment	Anecdotal reports of cooler temperatures and dressing in layers of breathable clothing such as cotton suggest these interventions may help some women cope with hot flashes.
Relaxation techniques such as yoga, meditation, deep breathing, guided imagery, mindful stretching, massage, mindfulness, aromatherapy, and prayer	Methodological challenges limit the ability to evaluate the effect of these strategies, but there is little likelihood of harm and most of these techniques have little cost. These interventions may decrease anxiety.

Based on Fisher TE, Chervenak JL. Lifestyle alterations for the amelioration of hot flashes. *Maturitas.* 2012;71:217-220[21]; Smith R, Flaws JA, Gallicchio L. Does quitting smoking decrease the risk of midlife hot flashes? A longitudinal analysis. *Maturitas.* 2015;82(1): 123-127[22]; Daley A, Stokes-Lampard H, Thomas A, MacArthur C. Exercise for vasomotor menopausal symptoms. *Cochrane Database Syst Rev.* 2014;11:CD006108. doi: 10.1002/14651858.CD006108.pub4[24]; Saensak S, Vutyavanich T, Somboonporn W, Srisurapanont M. Relaxation for perimenopausal and postmenopausal symptoms. *Cochrane Database Syst Rev.* 2014;7:CD008582. doi: 10.1002/14651858.CD008582.pub2.[25]

Mood Symptoms

Experts disagree about whether changes in hormone levels during the perimenopausal/postmenopausal period actually *cause* mood symptoms. The interactions between hormones and mood are complex, as are the interactions between the "symptom clusters," including pain, vasomotor symptoms, and sleep disruption, that women may experience during the perimenopause period.[23] Moreover, many women experience coexisting stressors, and some of these stressors during this time may be caused or exacerbated by a newly empty nest, or the "sandwich generation" phenomenon of caring for both children and elderly family members.[4] In spite of the controversies surrounding this topic, several risk factors for mood disorders are well known: Women with a history of depression or

premenstrual syndrome seem to be more vulnerable to depression around the time of menopause.[11]

Lifestyle Strategies for Treatment of Perimenopausal or Menopausal Symptoms

Some symptoms experienced during menopause may be indicative of underlying pathology separate from menopause and warrant investigation. An array of healthy lifestyle strategies can be offered to women who request relief from menopausal symptoms such as hot flashes, sleep difficulties, sexual concerns, and vulvovaginal discomfort (***Table 18-5***).[21,22,24,25]. Some of these strategies, such as maintenance of a healthy weight, smoking cessation, and increased exercise, have additional benefits for cardiovascular and bone health.

Hormone Therapy for Treatment of Menopausal Symptoms

The most effective treatment for vasomotor symptoms and vaginal atrophy related to menopause is hormone therapy (HT) with estrogen or estrogen and progestin. In 2002, reports emerged from the large Women's Health Initiative (WHI) study that initiated a rapid sea change in the recommendations for women during the perimenopausal/menopausal period. The WHI was a national study supported by the U.S. National Institutes of Health (NIH), designed to address major health problems associated with morbidity and mortality among postmenopausal women. More than 160,000 women age 50 to 79 years participated in the study. One of the three major randomized controlled trials (RCTs) examined menopausal hormonal therapy (HT), specifically estrogen therapy (ET) or estrogen plus progestin therapy (EPT). The expectation, based on observational studies and case reports, was that study findings would reveal these drugs provide relief of several menopausal symptoms and decrease the risk of cardiovascular events, especially coronary heart disease.[26]

Risks and Benefits of Hormone Therapy

The risks and benefits associated with HT are complex and may be related to the age at which therapy is initiated. The findings from the WHI provide the basis for most of the projected risks and benefits from the use of HT. **Table 18-6** summarizes these risks and benefits and can be used as a resource when discussing HT with women. Although the full longitudinal study was intended to continue for 9 years, the RCT examining the effects of EPT was discontinued early because the interim analysis found an increased—not decreased—risk of coronary heart disease among women in the EPT cohort. In addition, participants in this group had statistically significant increased risks of breast cancer, stroke, and pulmonary embolism. It was determined that these risks outweighed the benefits of decreased risks of colorectal cancer and bone fracture.[27] The findings were disseminated widely, including in consumer publications. Although the absolute number of adverse events were low, both consumers and healthcare providers were concerned enough that prescriptions for menopausal hormone therapies rapidly decreased.

Table 18-6 Risks and Benefits of Hormone Therapy Based on Randomized Controlled Trials Reported by the Woman's Health Initiative

Condition	EPT	ET
Risks[a]		
Coronary heart disease	Increased	NS
Stroke	Increased	Increased
Pulmonary embolism	Increased	Increased
Venous	Increased	Increased

thromboembolism		
Breast cancer	Increased	NS
Gallbladder disease	Increased	Increased
Stress incontinence	Increased	Increased
Urge incontinence	NS	Increased
Probable dementia	Increased	NS
Benefits		
Endometrial cancer	Decreased	NA
Hip fractures	Decreased	Decreased
Total fractures	Decreased	Decreased

Abbreviations: EPT, estrogen plus progestin therapy; ET, estrogen therapy only; NA, not applicable; NS, not statistically significant.

[a] Risk based on hazard risk with statistical significant finding, although the degree of risk varies among conditions and the absolute number may be small.

Data from Rossouw JE, Anderson GL, Prentice RL, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288(3):321-333[27]; Anderson GL, Limacher M, Assaf AR, et al. Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: the Women's Health Initiative randomized controlled trial. *JAMA*. 2004;291(14):1701-1712.[28]

Two years later, the portion of the RCT that evaluated ET in women with previous hysterectomies was also terminated early.[28] Estrogen alone did not decrease the risk of cardiovascular disease in this group, and there were indications of an increased risk of stroke. However, a decreased risk of bone fractures was found, and, unlike the EPT group outcomes, no statistically significant increased risk of breast cancer was detected.

The WHI has been criticized because the average age of participants in the EPT group was more than a decade older than the average age of a woman experiencing her final menses, or menopause. This problem may affect the risk of cardiovascular disease associated with HT. Subsequent studies have found a trend toward lower rates of cardiovascular mortality in women age 50 to 59 years who start ET within the first 10 years of experiencing menopause. Therefore, some experts suggest early adoption of HT in the perimenopausal period may have some cardiovascular benefits and this is an area of ongoing research. Nevertheless, today's guidelines continue to reflect the original WHI findings and recommend that HT be limited to short-term treatment of healthy women with moderate to severe symptoms.[4] HT is not recommended for prevention of cardiovascular disease.

Another limitation of the WHI is that a single specific progesterone (medroxyprogesterone acetate [Provera]) and a single specific estrogen (conjugated equine estrogen) were used. Subsequent studies with different estrogens and progestins have yielded slightly different statistical findings, although the majority of reports are congruent with the original WHI reports.[4,29,30]

Contraindications for Hormone Therapy

Contraindications for ET/EPT include undiagnosed abnormal vaginal bleeding; known, suspected, or history of breast cancer; known or suspected estrogen-dependent cancer; current or history of deep vein thrombosis or pulmonary embolism; current or recent (within the last year) stroke or myocardial infarction; liver disease; known or suspected pregnancy; and known hypersensitivity to ET/EPT. Smoking is not a contraindication to ET/EPT, although it is a contraindication for combined hormonal contraceptive use by women older than 35 years.

Hormone Therapy Options

HT is available in several different formulations that allow for oral, transdermal, or topical application. HT is considered a safe option for healthy women who are within 10 years of menopause, younger than 60 years, and lack any contraindications. Some professional associations recommend calculating a woman's baseline risk for cardiovascular disease and breast cancer before considering HT. Online calculators are listed in the Resources section at the end of this chapter. Counseling about HT includes a discussion about the risks, benefits, side effects, contraindications, and alternatives tailored for the individual.[31] This section reviews the information that is included in this discussion.

Estrogen Therapy Versus Estrogen and Progesterone Therapy

ET is available in oral, transdermal, topical, and vaginal formulations; EPT is available in oral and transdermal formulations. For women with a uterus who are using systemic estrogen, a progestin also is needed to protect from the endometrium-building effects of estrogen and predisposition to endometrial hyperplasia.[32] Conversely, women who have undergone a hysterectomy do not usually need a progestin. Most menopause experts agree that women with a uterus who use estrogen in *nonsystemic* doses (i.e., local vaginal therapy) do not need a progestin, although some experts advocate endometrial surveillance with an ultrasonographic measurement of endometrial thickness when women use a local vaginal therapy for more than 7 to 12 months.[14]

Choosing a Hormone Therapy and Regimen

Rarely does the specific estrogen or progestin is rarely an important aspect of choice. Most of the drugs are proprietary fixed drug compositions and are usually prescribed by brand name. Some estrogens are chemically manipulated to be conjugated or esterified so that they become more water soluble; those drugs are then administered orally or vaginally. Patches, gels, rings, spray, and some creams generally contain 17ß-estradiol. Synthetic estrogens are available in some products. Progestins also vary, with some products containing either drospirenone, levonorgestrel, norethindrone acetate, or norgestimate. These products are also available in a range of doses.

Medroxyprogesterone acetate (Provera) is one of the few progestins used separately from estrogen. It is not commonly used, however, because most women using EPT rely on a single

fixed drug combination. For women who experience problematic side effects of medroxyprogesterone acetate, some novel approaches have been suggested, such as use of a levonorgestrel-containing intrauterine device (IUD) or regular endometrial biopsies or transvaginal monitoring.

None of the specific agents seems to be more effective than the others for most women. For some individuals, there are additional considerations regarding the proprietary drug Premarin and its combinations such as Premphase and Prempro. Premarin, often termed conjugated equine estrogens (CEEs), actually is a complex blend of a number of steroids, including estrogens that are derived from pregnant mares' urine and other estrogenic compounds and androgens. Some women may be uncomfortable receiving hormones from horses, especially those who are concerned about the animals' well-being. For these women, use of other estrogens can be recommended.

Recently a new fixed drug combination has become available that includes conjugated estrogens and bazedoxifene (Duavee), an estrogen antagonist/agonist (EAA) originally marketed for prevention of osteoporosis. This formulation is indicated for treatment of women with mild to severe hot flashes and to prevent osteoporosis. The package insert states that women who only require protection from osteoporosis should seek another formulation without estrogen, perhaps to reduce estrogen-related risks.[33]

Outside the United States, various other synthetic agents may be used to treat vasomotor symptoms. Tibolone, a 19-nortestosterone derivative, is used especially in Europe, but is not available in the United States.[34] Dehydroepiandrosterone (DHEA) also has been used for this indication, but studies have failed to demonstrate its effectiveness and androgenic side effects limit its use.[35]

When considering the optimal route of administration, an important first consideration is whether the woman needs *systemic* or *local* vaginal therapy. If a woman needs relief from systemic symptoms (e.g., hot flashes), she needs systemic-level dosing, which can be obtained with oral and transdermal formulations. For women with localized concerns such as vulvovaginal atrophy, topical therapy or one of the vaginal rings (e.g., Femring) is appropriate.[36]

The topical products do not result in systemic levels of hormone. Vaginal rings are to be replaced every 90 days. A vaginal tablet is available and generally is used twice a week; vaginal creams are used daily. The decision to choose oral administration versus another route can be guided by individual preference, ability to use the method, and other factors such as avoidance of the first-pass effect with transdermal products.

Clinicians should be aware that systemic HT has been associated with worsening of preexisting stress and urge urinary incontinence and with increased incidence of new urinary incontinence.[37] In contrast, local vaginal estrogen topical therapy may improve continence.[38]

A variety of dosing regimens are available for estrogen plus progestin therapy. One of the oldest EPT regimens is *cyclic EPT*, in which a woman takes estrogen for the first 25 days of each month. Progestin is added to the estrogen for the last 10 to 14 days, and no hormones are taken for the final 3 to 6 days of the month. This regimen is seldom used now because women tend to experience withdrawal bleeding and hot flashes on the hormone-free days.

With *continuous-cyclic (sequential) EPT*, women take estrogen every day and add progestin

for 10 to 14 days each month. This regimen provides predictable withdrawal bleeding but no hot flash–inducing estrogen-free days. Premphase is one brand-name product that uses this formulation; it is sometimes prescribed for women in the menopausal transition during which HT-induced endometrial growth is likely. Premphase creates a predictable pattern of bleeding during this transitional period.

Continuous-combined EPT, in which women take estrogen and progestin every day, is currently the most commonly used formulation of HT. Within this category, several brand-name drugs are available. Within a few months of using this dosing option, a woman's endometrium becomes thin and most women are amenorrheic.

Intermittent-combined EPT (also called *pulsed progestin* or *continuous-pulsed EPT*) is also available. In this regimen, women take estrogen every day; they take progestin intermittently for 3 days on, 3 days off, repeated without interruption. Only one product, Prefest, uses this regimen.

Some women prefer a non-oral systemic formulation. Available formulations in this category include skin patches that are replaced once or twice weekly and gels or spray that may be used daily.

Most HT products are also available in a variety of doses. The effective dose for symptom relief varies from woman to woman, so a certain amount of trial and error is often needed to determine the appropriate dose. Clinicians should prescribe the lowest dose of HT that effectively manages symptoms and encourage women to use this therapy for the shortest time possible. Optimally, EPT should be taken for no longer than 5 years, but the decision to discontinue therapy should be individualized. For ET, a longer duration may be safer. These guidelines are congruent with the results of a longitudinal study conducted as part of the WHI, in which women using EPT for a median of 5.6 years and women using ET only for a median of 7.2 years were found to have no increase in all-cause, cardiovascular, or cancer mortality during 18 years of follow-up.[39] The guiding principle should be to start with a low dose and titrate upward, eventually selecting the lowest dose that accomplishes the HT goal.[4] Charts listing all the potential HT products are available from several Internet sites.[40] The most important aspect is identifying a drug and regimen that provides a woman relief of her symptoms.

Pregnancy Prevention

Pregnancy is possible until 12 months after a woman experiences her final menstrual period. Women in the perimenopausal period who want to avoid pregnancy should use contraception and not rely on HT to prevent pregnancy. Although both HT and combined hormonal contraceptives contain estrogen and progestin, the dose and formulation of HT will not prevent pregnancy. Moreover, a woman whose ovaries are still functioning in a reproductive capacity is not likely to benefit from the doses of hormones found in HT because they are low in comparison to a reproductive-age woman's hormone levels. Therefore, women generally do not begin taking HT until they have reached menopause. For healthy women who experience perimenopausal symptom relief with hormonal contraception use, many clinicians recommend making the transition to HT, if needed, when the woman has reached age 55 years—the age at

which 90% of women will have reached menopause.[4]

Discontinuation of Hormone Therapy

When women discontinue HT, intuition would indicate that gradual tapering would result in a less unpleasant experience than abrupt discontinuation. In reality, evidence does not support this theory: Women's experiences seem to be no better with gradual tapering of HT than with sudden discontinuation.[4,11]

Bioidentical Hormone Therapy and Custom-Compounded Formulations

Bioidentical hormones are plant-derived hormones that are chemically identical to the endogenous hormones (e.g., estrogen, progesterone) a woman's ovaries produce during her reproductive years.[4] Many bioidentical hormone therapies are available in Food and Drug Administration (FDA)-approved, well-tested formulations. Although the list is long, a few examples include 17ß-estradiol products such as Estrace, Climara, Divigel, Evamist, and Estring; estradiol acetate products such as Femring; and oral micronized progesterone (Prometrium).[4]

Unfortunately, some consumers, healthcare providers, and pharmacists use the term "bioidentical hormone" to describe formulations that are more accurately described as "custom-compounded." Custom-compounded products were initially intended for use by women for whom standard FDA-approved products were ineffective, but post-WHI safety concerns caused an increasing number of women and clinicians to explore options other than the commercial products used in the WHI.[4] Custom-compounding of HT involves individual mixing of preparations, usually by a compounding pharmacist. Such products may include one or more hormones, sometimes delivered via nonstandard dosing routes such as subdermal implants, sublingual tablets, rectal suppositories, or nasal sprays, in an attempt to individualize therapy for a particular woman.

A number of concerns have arisen regarding custom-compounded HT. Clinicians who prescribe custom-compounded hormones often base the dosing on salivary hormone testing, even though there is no scientific evidence to support the claim that such testing can accurately dictate HT doses.[4] Moreover, no evidence supports the contention that custom-compounded hormones are safer than traditional HT, and their effectiveness is an open question given the limited research conducted on these formulations.[41] Safety concerns stem from the lack of batch-to-batch testing for consistency, purity, or dose.[42] The FDA does not regulate compounded products. Instead, individual state agencies regulate compounding pharmacies, and this regulation process varies widely from state to state.

Topical progesterone, which is typically found in the custom-compounded HT gels, creams, and lotions, does not adequately raise serum levels to protect the endometrium against estrogen's stimulatory effects, which may ultimately lead to endometrial hyperplasia and endometrial cancer in some women.[4] Custom-compounded hormones also may be substantially more expensive because they may not be covered by insurance.[41]

Several organizations—including the North American Menopause Society (NAMS), the

FDA, the American Congress of Obstetricians and Gynecologists (ACOG), the Endocrine Society, the American Medical Association, and the American Cancer Society—agree that custom-compounded therapy is no safer than traditional, commercially prepared and FDA-approved therapies and that compounding may impart additional risks.[2]

Nonhormonal Prescription Options for Treatment of Menopausal Symptoms

Lifestyle changes are not always effective and some women do not want to use or cannot use hormonal therapies. For these individual's, nonhormonal strategies are available.

Nonhormonal Treatments for Genitourinary Symptoms

The best anticipatory guidance regarding the vulvovaginal changes is to advise women to maintain adequate moisture throughout the body, as well as in the reproductive system. In contrast, moisturizers are longer-lasting products that can maintain moisture and beneficially lower the vaginal pH.[4] Water-based lubricants can be used to increase comfort during sexual activity. Oil-based products coat the vaginal lining, thereby preventing the release of natural secretions and should be avoided. These products also cause condom breakage. If possible, it is best to avoid use of antihistamines and other over-the-counter drugs that have drying effects, including douches, sprays, and colored or perfumed toilet paper and soaps.

Nonhormonal Treatments for Vasomotor Symptoms

Although some nonhormonal drugs have evidence to support their effectiveness, none is as effective as systemic HT in treating hot flashes, and all of these medications have varying side-effect profiles. Certain selective serotonin reuptake inhibitors (SSRIs)/serotonin-norepinephrine reuptake inhibitors (SNRIs) (e.g., fluoxetine [Prozac, Sarafem], paroxetine [Paxil]) have moderate effectiveness in providing relief from hot flashes and are especially useful for women with coexisting mood disorders. Women may respond rapidly to these agents, but tapering is required to discontinue them. SSRIs and SNRIs may also result in decreased libido in women.

The anticonvulsant gabapentin (Neurontin) has been found to be moderately effective for treatment of hot flashes, but its common side effect of somnolence may be problematic. Pregabalin (Lyrica), a drug used for treatment of fibromyalgia or neurologic conditions, is being investigated for potential use for vasomotor symptoms but is not recommended at this time. Methyldopa (Aldomet) has limited effectiveness and a significant negative side-effect profile; it should not be used for this indication.[4,14]

An estrogen antagonist/agonist, previously known as a selective estrogen receptor modular, is available as a treatment for women with postmenopausal dyspareunia. Ospemifene (Osphena) is a daily oral tablet that results in thickening of vaginal tissue. A potential risk with use of this medication is stimulation of the endometrium leading to increased risk of endometrial hyperplasia or cancer. Localized estrogen treatments such as creams and rings can be prescribed as an adjunctive treatment.

Complementary and Alternative Therapies

Complementary and alternative medicine (CAM) encompasses a variety of healthcare practices and products. Complementary therapies are used *in addition to* and alternative therapies are used *instead of* conventional approaches.

Despite extensive use of CAM, few of these therapies have solid evidence to support their safety and effectiveness.[43] A valuable resource for clinicians and the public about the safety and effectiveness of CAM is the National Center for Complementary and Integrative Health, which is part of the National Institutes of Health.[44]

Phytoestrogens

A large number of botanically derived phytoestrogen supplements and foods are purported to ease menopausal symptoms.[45] In addition to conflicting research findings about these substances, there is a great deal of confusion about the terminology associated with them. *Phytoestrogen* is a broad term for plant-derived compounds that have estrogenic and antiestrogenic effects. The three major classes of phytoestrogens are isoflavones, lignans, and coumestans.[46]

Isoflavones are plant-derived compounds with estrogen-like activity and a chemical structure similar to that of estradiol. Soy and red clover are the two most commonly used isoflavones for the relief of menopausal symptoms.

Soy, usually derived from whole soybeans, is the most widely consumed isoflavone-containing food. The amount of isoflavones in soy-containing foods varies widely. Moreover, the manufacturing process used for each food can have negative effects on the amount of isoflavones found in the final product. Although the research evidence about the effects of soy is conflicting and some studies have failed to find effectiveness,[45] it is possible that soy has modest benefits for the short-term (e.g., 3 months) treatment of hot flashes but not for longer periods. Moreover, a trial of 3 months is sufficient to evaluate whether a woman is likely to respond to therapy. Soy does not appear to have harmful effects on the endometrium or on the breast, even in women at increased risk for breast cancer and breast cancer survivors.[4]

Red clover contains different types of isoflavones and has different affinities for various steroid receptors when compared to soy. Evidence is mixed about effectiveness, with a few studies demonstrating small beneficial effects on hot flash reduction but most research has not found red clover to be effective.[4]

Lignans, found in flaxseed oil and whole grains, and *coumestans*, found in alfalfa sprouts and certain beans and peas, are thought to have a much lower hormonal affinity than isoflavones. These phytoestrogens are not very useful in decreasing menopausal symptoms.[4]

Herbal Therapies

Despite widespread consumer interest in herbal therapies, a lack of standardization of doses and the sparse number of high-quality, large studies that have evaluated the safety and effectiveness of herbs make determination of effectiveness challenging. In addition, herbal

therapies can cause adverse events and are associated with herb–drug interactions, although the extent of each is difficult to determine.[4]

The herbal remedies that women most often use to relieve menopausal symptoms are black cohosh, chaste tree berry, dong quai, evening primrose oil, gingko, ginseng, kava, licorice root, St. John's wort, valerian, and wild yam. **Table 18-7** lists each of these herbs' proposed effects on menopausal symptoms, dosages, and summary of safety and effectiveness.[4,46-52]

Table 18-7 Herbal Remedies Used Most Often for Relief of Perimenopausal and Menopausal Symptoms

Herb	Proposed Effects	Dose	Comments
Black cohosh (*Cimicifuga racemosa*)	Diminishes hot flashes	20 mg twice/day of proprietary standardized extract; recommended dose of Remifemin supplement is 40–80 mg/day	Possible evidence of benefit for hot flash relief. Most widely studied herb for menopausal symptoms, but recent studies have many methodological limitations. Much variation in brands and preparations.
Chaste tree berry (*Vitex agnus castus*)	Diminishes PMS symptoms; regulates menses	250 mg of crude herb or 20 mg/day of 12:1 ratio of standardized extract	Evidence of benefit for PMS symptoms. Possible benefits for regulating irregular perimenopausal bleeding. Popular in Europe and approved therapy in Germany for PMS, mastalgia, and menopause symptoms.
Ginseng (*Panax ginseng*)	Improves mood, minimizes fatigue	100–600 mg/day of standardized extract in divided doses	No data indicating effectiveness for hot flashes. Possible effectiveness for overall feelings of well-being. Significant problems with purity of preparations.
Kava (*Piper methysticum*)	Minimizes irritability, promotes sleep	150–300 mg of root extract per day in divided doses	Effective for treating anxiety but effect is small. Possible hepatotoxicity; some experts recommend avoiding completely.
St. John's wort (*Hypericum perforatum*)	Diminishes hot flashes, decreases irritability, treats depression	300 mg of standardized extract taken 3 times per day	Evidence of benefit for mood stabilization and mixed evidence for hot flash relief. Sometimes used in conjunction with black cohosh. Multiple drug-herb interactions.
Valerian (*Valeriana officinalis*)	Promotes sleep; decreases anxiety	**Insomnia:** 300–600 mg aqueous extract 30–60 min before bed; **Anxiety:** 150–300 mg aqueous extract each morning and 300–400 mg each evening	Evidence of benefit for insomnia and anxiety. Few side effects at recommended dosages and with relatively short-term use.

Abbreviation: PMS, premenstrual syndrome.

Based on North American Menopause Society. *Menopause Practice: A Clinician's Guide*. 5th ed. Mayfield Heights, OH: North American Menopause Society; 2017[4]; Borrelli F, Ernst E. Alternative and complementary therapies for the menopause. *Maturitas.* 2010;66: 333-343[46]; Guttuso T. Effective and clinically meaningful non-hormonal hot flash therapies. *Maturitas.* 2012;72:6-12[47]; Andrews JC. Vasomotor symptoms: an evidence-based approach to medical management. *J Clin Outcome Manage.* 2011;18:112-128[48]; Hall E, Frey BN, Soares CN. Non-hormonal treatment strategies for vasomotor symptoms: a critical review. *Drugs.* 2011;71:287-304[49]; Regestein QR. Is there anything special about valerian? *Menopause.* 2011;18:937-939[50]; Taavoni S, Ekbatani N, Kashaniayn M, Haghani H. Effect of valerian on sleep quality in postmenopausal women: a randomized, placebo-controlled clinical trial. *Menopause.* 2011;18: 951-955[51]; Leach MJ, Moore V. Black cohosh (*Cimicifuga* spp.) for menopausal symptoms. *Cochrane Database Syst Rev.* 2012;9: CD007244. doi: 10.1002/14651858.CD007244.pub2.[52]

There is no evidence of effectiveness for evening primrose oil (*Oenothera biennis*). Not only is there a lack of evidence for the use of dong quai (*Angelica sinensis*) or gingko (*Gingko biloba*) for symptom relief, but both of these products are also associated with heavy uterine bleeding and even potential subdural hematomas. Licorice root (*Glycyrrhiza glabra*) has not been found to be effective, and high doses or long-term use can cause hypertension and kidney, liver, or cardiac dysfunction. Some women use wild yam (*Dioscorea villosa*) as a cream based on the perception that it provides progestational effects, but this herb is not converted to

progesterone in the body.[4]

Acupuncture

Practitioners of acupuncture stimulate precise points of the body, usually by inserting fine needles into the skin. Studies show inconsistent results for the effectiveness of acupuncture for relieving menopausal symptoms. Methodological limitations, such as challenges in creating double-blind study designs, limit researchers' ability to more definitively answer questions about effectiveness. In summary, many experts agree that acupuncture is a promising therapy but requires more study to determine its effectiveness.[4,53]

Homeopathy

Homeopathy is based on the principle of "like cures like," in which practitioners use highly diluted preparations of plant extracts and minerals to promote an individual's innate healing processes. As yet, the precise mechanisms of action of homeopathy are poorly understood. A homeopathic remedy is theorized to act as a catalyst, stimulating the body to heal itself. The homeopathic remedies that menopausal women use most often to relieve symptoms are Lachesis, Pulsatilla, and Sepia. Although some trials show promising results with such therapies,[54] most homeopathy research has failed to provide robust results supporting their effectiveness in improving menopausal symptoms. Further study using high-quality study designs is needed. Nevertheless, although its effectiveness studies are ongoing, homeopathy is thought to be quite safe.[4]

Hypnosis

Among the other therapies that have been advocated for treatment of women with vasomotor symptoms is clinical hypnosis. A multiyear, NIH-funded project exploring this therapy included small RCTs that demonstrated women undergoing clinical hypnosis had a decreased number of hot flashes compared to a placebo group.[55] Additional research is needed in this area, although these results are promising.

Common Conditions Experienced by Women During Midlife and Beyond

The Study of Women's Health Across the Nation (SWAN) is an ongoing, multisite, longitudinal, epidemiologic study supported by the National Institutes of Health, which is designed to examine the health of women during their middle and older years.[56] SWAN is examining the physical, biologic, psychologic, and social changes occurring in this cohort and is providing a rich repository of information about these women. As a result of SWAN and other research studies, it has become clear that many conditions exist that are linked to aging, yet separate from menopause. As women age, a variety of chronic diseases, such as hypertension, diabetes mellitus, and cancers, are more likely to develop.[4,57]

Urinary Tract Infections

When urinary symptoms appear to be related to atrophic changes, vaginal estrogens can provide some relief of urgency, frequency, nocturia, incontinence, and recurrent urinary tract infections.[58] Systemic HT is not recommended for treatment of women with incontinence and may be deleterious.[58]

Urinary tract infections should be promptly treated because they are a common cause of sepsis among older women. Incontinence and overactive bladder are not an inevitable part of aging, and clinicians should not regard them as expected outcomes among older women.[4,59] Incontinence is discussed in more detail in the *Gynecologic Disorders* chapter.

Sexuality

The majority of women who are in midlife or older remain sexually active, and menopause is generally not related to sexual satisfaction.[60] As women age, they may place more emphasis on aspects of sex other than vaginal penetration, and the ability to recalibrate expectations allows for sexual satisfaction even if sexual activity differs from that pursued during the younger years.[60] Unfortunately, many myths about sex and aging continue to exist, including the myth that midlife and older women are uninterested in sex. According to findings of the National Social Life, Health, and Aging Project, many women are sexually active at midlife and beyond: 62% of women age 57 to 64 years reported partnered sexual activity in the previous year, as did 39% of women age 65 to 74 years, and 17% of women age 75 to 85 years.[61] More than 50% of sexually active women in all age groups reported having sexual activity 2 or 3 times per month. Health promotion for women of all ages who are in new or nonmonogamous relationships, should include discussions about the prevention of sexually transmitted infections.

Some midlife women report difficulties with sexual function. Although each woman's experience is different, the three primary sexual health concerns that midlife women report are decreased sexual desire, decreased vaginal lubrication, and inability to have an orgasm. Information regarding sexual history taking can be found in the *Introduction to the Care of*

Women chapter. Certain situations for the midlife or older woman might require even more time to evaluate in depth. These include changes in body image following a hysterectomy or mastectomy; arousal phase disorders, which can be exacerbated by anxiety or depression; and an unwelcome loss of sexual partners due to divorce or death.

Decreases in levels of ovarian hormone levels, which can vary from woman to woman, may contribute to sexual function changes. Decreased blood flow to the clitoris and loss of vaginal elasticity can contribute to discomfort during sexual activity. As testosterone levels gradually decline, many women notice a decrease in libido. Midwives should consider referring women of any age for sex therapy if they report concerns related to sexual function, especially when there is long-standing sexual dysfunction, current or past abuse, or an acute psychological event. Additional information about sexual disorders can be found in the *Gynecologic Disorders* chapter.

Among women who experience natural menopause, researchers have found differences in decline of sexual arousal related to race/ethnicity: Women of Japanese descent appear to experience a greater decline when compared with white women, whereas African American women experience a smaller decline. Women with induced menopause have reported a decline that lasted for as long as five years.[4]

Androgen Therapy

Some clinicians argue that it is beneficial for midlife women to receive androgen therapy because even a slight and gradual decrease in a postmenopausal woman's total testosterone level may be associated with decreased libido and other negative changes that can be managed with androgen therapy.[4,11] Others argue that women often require supraphysiologic dosing to appreciate any improvement in sexuality. Women's serum androgen levels do not accurately predict sexual function, and exogenous androgen use is not associated with an actual increase in sexual behavior.[4,14]

Currently there are no androgen-containing HT products for women approved by the FDA, although some clinicians prescribe custom-compounded micronized testosterone or various other androgen-containing products on an off-label basis. Side effects associated with androgen therapy include acne, growth of facial and body hair, clitoral enlargement, permanent voice deepening, emotional volatility, and deleterious effects on lipids and liver function.[4] Effects on cardiovascular disease or breast cancer, as well as long-term consequences of androgen therapy, are poorly understood. In addition, androgens do not protect the endometrium from estrogen's hyperplasia effects, and women with an intact uterus who receive estrogen–androgen therapy should also use a progestin.[14]

Endometrial Hyperplasia or Cancer

Bleeding after menopause may be an indication of endometrial or uterine malignancy or a condition of premalignancy such as atypical endometrial hyperplasia. Women who experience postmenopausal bleeding should be referred to a specialist for further evaluation. Endometrial biopsy and sonographic scanning are usually included in the initial assessment.[4] **Table 18-8**

identifies some of the critical elements to be included in a consultation or transfer note for a women with postmenopausal bleeding. In addition to the biopsy and ultrasound, other management interventions may include a dilatation and curettage and potentially a hysterectomy if a malignancy is discovered.

Table 18-8	Sample Critical Elements for a Consultation or Transfer Note for a Woman with Postmenopausal Bleeding
Date or year of final menstrual period	
Risk factors for endometrial cancer, including genetics, family history, personal past history, nulliparity, and obesity	
Current and past use of hormone therapy, including hormonal contraception and specific hormonal therapy	
Current medications, including tamoxifen	
Last Pap test and results	
Description of bleeding, including time of first discovery, duration, amount, and association with coitus	
Care provided before transfer: procedures and results; laboratory results or if pending; including pregnancy test, endometrial biopsy, and/or transvaginal sonogram	
Description of pelvic findings	
Assessment/diagnosis of the situation	
Summary of rationale for transfer and request for information about the treatment plan for follow-up	

Sleep Disturbances

Midlife women frequently report problematic sleep patterns, defined as an inadequate number of hours of sleep, sleep of poor quality, or an inability to function in an alert state during desired waking hours. Women might experience insomnia as a prolonged time needed to fall asleep, as inability to stay asleep all night, or as early awakening without being able to get back to sleep. Estimates of sleep difficulties vary widely, but as many as 48% of women in the United States age 40 to 64 years report sleep difficulties, including fewer hours of sleep than desired, more frequent insomnia, and more frequent use of prescription sleep aids when compared to premenopausal women.[4]

Sleep difficulties can have adverse effects on quality of life. Primary sleep disorders such as restless legs syndrome are common in midlife women. Night sweats secondary to hot flashes can be managed with other treatments for vasomotor conditions. However, night sweats are not the only etiology of sleep disorders.

Women who report problems sleeping in addition to diminished capacity to enjoy life, energy loss, appetite disturbance, or increased somatic complaints, or who have poorly diagnosable physical problems, may be experiencing major depression. Thyroid dysfunction, autoimmune disorders, infections such as HIV and tuberculosis, illnesses causing chronic pain, and medication effects are all worthy of consideration when providing care to women reporting sleep difficulties.[4] Likewise, chronic illnesses such as cardiac and mood disorders and anxiety from stressful life events can be related to sleep difficulties.

Women with mild sleep disturbances or women who are motivated to avoid medication therapy are often successful at improving the quality and quantity of their sleep with lifestyle

changes. Avoiding caffeine in the evening may improve sleep patterns; women should be aware that caffeine can be found in energy drinks, some nonprescription pain relievers, premenstrual syndrome symptom-relief remedies, weight-control products, alertness aids, cold medications, and diuretics. Moreover, caffeine has a long half-life and it can take several hours before the stimulant effects wane.[4] Alcohol can initially assist a person in falling asleep, but it can also cause fragmented sleep and early awakening. Nicotine also disrupts sleep, as do drugs such as marijuana, heroin, and prescription opioids.

A light snack with protein and complex carbohydrates, or a source of tryptophan such as milk or chamomile tea, may improve the ability to fall asleep. Engaging in vigorous exercise or stressful activities close to bedtime can cause difficulties in relaxing and falling asleep, but regular, moderate exercise earlier in the day can promote healthy sleep habits. Erratic sleep hours, as well as naps, can negatively affect sleep quality. A regular schedule capitalizes on the light/dark cycle. A warm bath or other pleasant ritual, as well as reserving the bedroom for only sleep and sex, can condition better sleep patterns.[4]

Weight Changes

Midlife women frequently report weight gain, with the average weight gain during the menopause transition being approximately 5 pounds. However, evidence does not support a causal relationship: Neither menopause nor HT appear to *cause* weight gain.[4] Instead, aging and lifestyle factors are more likely to cause an increase in weight. Lean muscle mass decreases with age, lowering a woman's metabolic rate. This reduced metabolic rate, combined with a more sedentary lifestyle, causes women to burn fewer calories and gain weight if they do not reduce their caloric intake.[62]

Regardless of cause, it is clear that many women in the United States are overweight or obese. These women are at an increased risk for adverse health consequences, including cardiovascular disease, hypertension, type 2 diabetes, certain cancers, and premature death. Thus, diagnosis and management of obesity and overweight, including counseling about lifestyle management strategies, are vitally important.

Hair and Skin Changes

Sun damage, redistributed or decreased subcutaneous fat, skin laxity from weight changes, and decreased underlying muscle tissue cause most of the skin changes that women notice during midlife years. Other potential midlife skin changes include dryness, acne, hair loss, brittle and slow-growing nails, and decreased wound healing, all of which may contribute to psychological distress or altered body image.[4]

The midwife is likely to find a variety of benign skin lesions when examining the skin of women in their midlife years. Some examples are skin tags (fibroepitheliomas) or benign, soft, brown, pedunculated polyps usually found on eyelids and the neck. Seborrheic keratosis or sharply defined, flat, light or dark brown papules are usually found on the trunk. Campbell de Morgan spots (cherry angiomas) are red dome-shaped papules also found primarily on the trunk. Acne rosacea, an inflammatory condition of the sebaceous glands, likewise has an

increased incidence with age.

Although the great majority of skin lesions are benign, the clinician should always consider the possibility of a dermatologic malignancy and consult with or refer the woman to a dermatologist as indicated. This vigilance is particularly important when a woman has a lesion that has asymmetry, border irregularities, color variations, a diameter greater than 6 mm, or a sudden increase in size.[4] If any vulvar lesions have an atypical appearance, vulvar cancer should be considered and vulvar biopsy and/or referral to a specialist arranged.

Thyroid Function Changes

The incidence of thyroid dysfunction increases as women age, and screening for thyroid dysfunction is more cost-effective in perimenopausal and postmenopausal women than it is in younger women.[4] Relying on classic symptoms of thyroid disease to prompt screening is problematic in this age group because other common perimenopausal symptoms such as changes in menstrual bleeding characteristics, sleep difficulties, fatigue, mood changes, heat intolerance, and palpitations overlap with symptoms of thyroid dysfunction. Thus, a higher index of suspicion is needed to diagnose and treat thyroid disease early in the older woman compared to her younger counterpart.[4]

Oral Health

The oral mucosa contains estrogen receptors, so variations in hormone levels can have a direct effect on the oral cavity.[63] Postmenopausal women experience periodontal disease more frequently and more severely. Saliva naturally decreases during menopause and may lead to oral problems such as xerostomia, burning mouth syndrome, an increase in dental caries, dysesthesia, taste alterations, gingivitis, periodontitis, and osteoporotic jaw. A thorough oral health history, clinical examination of the oral cavity during a physical examination, and regular dental care provided by a dentist are indicated.

Bone loss in postmenopausal women is directly correlated to tooth loss. The loss of 1% of the body's total bone mineral density quadruples the risk for tooth loss.[4] Prevention of osteopenia and osteoporosis as well as treatment of women with osteoporosis will facilitate maintaining bone and dental health. Adequate vitamin D levels help to prevent and treat osteoporosis-associated periodontal disease. Regular and frequent periodontal/dental examinations as well as meticulous care at home should be stressed.[4]

Bone Health, Osteopenia, and Osteoporosis

In addition to oral health, bone health has major ramifications for the older woman. Osteoporosis is a deceptively silent disease. In this disorder, bone mass and strength are lost and bone quality deteriorates—changes that increase the risk that the bone will fracture.[64] **Table 18-9** defines key terms related to osteoporosis.[64-66] Most women do not have any symptoms and may not realize they have osteoporosis until a fracture occurs. Approximately 2 million osteoporosis-related fractures occur annually in the United States, which leads to significant morbidity among women.[65] Hip fracture is the most costly and dramatic type of fracture. Thus, preventing fracture—particularly hip fracture—is a women's health priority.

Table 18-9 **Osteoporosis Definitions**

Term	Definition
Bone mineral density (BMD)	Surrogate marker for bone strength and resistance to fracture
	Measured by dual-energy X-ray absorptiometry
	Usually measured and reported for the hip, femoral neck, and spine
	Results reported as standard deviations from the mean of a reference population
Dual-energy X-ray absorptiometry (DEXA or DXA)	Gold-standard diagnostic technology for measuring BMD
FRAX	Online fracture risk assessment tool to assist in clinical decision making for screening and treatment decisions
	Calculates absolute fracture risk/estimate of risk for fracture in the next 10 years
	Calculations include age, bone density, and risk factors
Osteoblasts	Bone-building cells (*"**B**lasts **b**uild"*)
Osteoclasts	Bone-breakdown cells (*"**C**lasts **c**ut down" or "**C**lasts **c**arve out"*)
Osteopenia	Low bone mass
	T-score between –1.0 and –2.5
Osteoporosis	Skeletal disease of low bone mass and deterioration of bone micro-architecture
	Resulting bone fragility increases the risk that bone will fracture
	BMD T-score < –2.5 constitutes osteoporosis
T-score	Comparison with mean peak BMD of a normal, young, same-sex population
	Number of standard deviations that a person's BMD differs from the mean peak BMD of a normal young adult of the same sex
	Most commonly used measurement when making clinical decisions about midlife and older women
Z-score	Comparison with a reference population of the same age, gender, and ethnicity
	Not commonly used when evaluating midlife and older women; can be misleading because bone density is often low in older adults
	Used primarily for children, teens, and young adults

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 129: osteoporosis. *Obstet Gynecol.* 2012;120:718-734[64]; Tufts G. New treatment approach for osteopenia. *J Midwifery Womens Health.* 2011;56:61-67[65]; North American Menopause Society. Management of osteoporosis in postmenopausal women: 2012 position statement of the North American Menopause Society. *Menopause.* 2012;17:25-54.[66]

Humans build bone throughout childhood, adolescence, and young adulthood, with bone mass peaking by age 18 to 25 years. Genetic factors, and to a lesser extent nutrition, physical activity, and general health, determine the amount and quality of bone that a woman has at when bone is maximal. Throughout the bone formation process, osteoclasts help to remove old bone and osteoblasts help to build new bone. With increasing age and as women become postmenopausal, more bone is removed than is built; the result can be weakened bone structure and decreased bone mass. In turn, bone becomes more fragile and is more prone to fracture even without significant trauma.[64] Certain risk factors predispose individuals to experience an even greater extent of bone loss and/or an even higher risk of fracture. **Table 18-10** summarizes risk factors for osteoporosis and osteoporosis-related fractures.

Table 18-10 Risk Factors for Osteoporosis and Osteoporotic Fractures in Women

Nonmodifiable Risk Factors	Potentially Modifiable Risk Factors
Age ≥ 65 years	Smoking
Personal history as an adult with fracture without substantial trauma	Body mass index < 20 or weight < 127 pounds
Family history of osteoporosis	Eating disorder or excessive exercise-induced amenorrhea
Hip, spine, or wrist fracture without substantial trauma in a first-degree relative	Chronic glucocorticoid use (e.g., prednisone > 5 mg/day for > 3–6 months)
Race: white and Asian women are at greatest risk, followed by Hispanic and African American women	Multiple risk factors for falling (e.g., decreased leg or arm muscle strength, diminished vision, environmental hazards, impaired cognition)
Sex: female	Chronic illnesses: rheumatoid arthritis, hyperparathyroidism, impaired absorption syndromes
Late menarche (> 15 years) or early menopause (< 45 years)	Heavy alcohol use (≥ 3 drinks/day)

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 129: osteoporosis. *Obstet Gynecol.* 2012;120:718-734[64]; Tufts G. New treatment approach for osteopenia. *J Midwifery Womens Health.* 2011;56:61-67[65]; North American Menopause Society. Management of osteoporosis in postmenopausal women: 2012 position statement of the North American Menopause Society. *Menopause.* 2012;17:25-54.[66]

Clinicians usually diagnose and manage women with low bone mass by measuring bone mineral density (BMD) with dual-energy X-ray absorptiometry (DXA), an enhanced type of X ray. Examples of indications for BMD are listed in **Table 18-11**. Although BMD is not a perfect surrogate for fracture risk, a person's BMD is more predictive of fracture than cholesterol level is predictive of myocardial infarction.[67]

Table 18-11 Considerations for Ordering Bone Mineral Density Testing

At menopause: women with a history of fracture or with a parent who had a hip fracture or multiple vertebral or nonvertebral fragility fractures
Before menopause or during menopausal transition: women with specific risk factors for increased fracture risk such as anorexia, long-term glucocorticoid use, metabolic bone disease, hyperthyroidism, low body weight, prior low-trauma fracture, or high-risk medication
For treatment decisions: to monitor effects of osteoporosis therapy, if being considered for medications, or if evidence of bone loss would lead to treatment

As indicated by FRAX: clinicians can use FRAX to determine which women younger than 65 years should have BMD testing; if FRAX indicates that a woman has a 9.3% or higher 10-year risk of major osteoporotic fracture, it is reasonable for that woman to have a DXA
To monitor effects of DMPA on bone density

Abbreviations: BMD, bone mineral density; DMPA, depot medroxyprogesterone acetate; DXA, dual-energy X-ray absorptiometry; FRAX, World Health Organization's Fracture Risk Assessment Tool.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 129: osteoporosis. *Obstet Gynecol.* 2012;120:718-734[64]; Tufts G. New treatment approach for osteopenia. *J Midwifery Womens Health.* 2011;56:61-67[65]; North American Menopause Society. Management of osteoporosis in postmenopausal women: 2012 position statement of the North American Menopause Society. *Menopause.* 2012;17:25-54.[66]

If bone mass is placed on a continuum in which normal bone mass is 1 standard deviation (SD) above and below 0, *osteopenia* represents the next finding on the continuum. Osteopenia is defined as decreased bone mass ranging from 1 SD below 0 to 2.5 SD below 0. This condition is three times more prevalent than osteoporosis. Moving even farther away from normal bone mass on the continuum, *osteoporosis* represents even more significantly decreased bone mass, 2.5 SD below 0 and lower (**Figure 18-2**). A T-score is the most commonly used reporting method for the results of the DXA measurements.[64] Generally, DXA measurements are taken at the femoral neck, at the posterior–anterior lumbar spine, and as a total hip measurement; the lowest of the three values determines the diagnostic category to which the individual is assigned.[64]

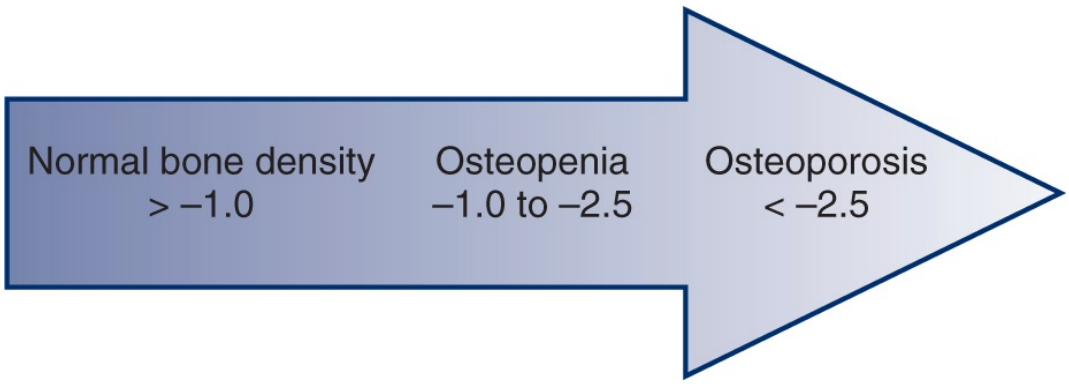


Figure 18-2 Bone mineral density continuum.

Data from National Osteoporosis Foundation. *Clinician's Guide to Prevention and Treatment of Osteoporosis.* Washington, DC: National Osteoporosis Foundation; 2010.[68]

FRAX: The Fracture Risk Assessment Tool

Clinically, the important goal related to bone health is *fracture prevention*, rather than simply promoting a specific BMD score. BMD is just one aspect of a woman's bone health, as other factors also influence fracture risk. The World Health Organization's Fracture Risk Assessment Tool, also known as FRAX, was developed to assist clinicians in estimating the likelihood of a major (e.g., hip, spine, forearm) fracture in the next 10 years.[64] Economic modeling for cost-effectiveness determines the cut-offs for treatment recommendations. For the U.S. population, the National Osteoporosis Foundation recommends treatment for persons with a 3% or higher

chance of breaking a hip in the next 10 years or a 20% or greater chance of breaking any major bone in the next 10 years.[68]

Risk factors in the FRAX algorithm include age, gender, smoking, consumption of three or more alcoholic drinks per day, rheumatoid arthritis, low BMI, prolonged corticosteroid use, secondary osteoporosis, parent with a hip fracture, and lower BMD.[65] The algorithm does not take into account certain aspects of these risk factors, such as the amount of tobacco use or corticosteroid use, and it does not consider some risk factors at all (e.g., activity level; risk for falling; and intake of caffeine).[69]

The National Osteoporosis Foundation provides several clinical caveats to consider when using FRAX. When caring for women in the United States, for example, clinicians should consider that FRAX is intended for postmenopausal women and not for younger adults or children. For women currently or previously taking osteoporosis medications, the midwife should exercise caution in interpreting FRAX scores because FRAX has not been validated in this population. Moreover, while the intent of FRAX is to make clinical decisions more straightforward, all treatment decisions require careful clinical judgment of characteristics of the individuals. FRAX does not capture all risk factors; conversely, its recommendations do not mandate treatment.[69]

Lifestyle and Nonpharmacologic Strategies to Promote Bone Health

Robust scientific evidence supports several lifestyle changes and nonpharmacologic strategies to promote bone health for all women. These strategies are generally inexpensive, have minimal inherent risks, and can confer benefits for other aspects of a person's health. They include consuming a healthy diet with an adequate amount of certain nutrients, participating in regular weight-bearing exercise, maintaining a healthy weight, and avoiding tobacco use and excessive alcohol intake.

Of the various dietary nutrients, calcium and vitamin D are the most vital for bone health, and consumption of a diet rich in these nutrients and/or supplement intake of them lowers fracture risk.[65] Major organizations such as the National Osteoporosis Foundation, the National Academy of Sciences, and the Institute of Medicine recommend that women older than age 50 years consume 1200 mg of calcium per day and 800–1000 IU of vitamin D per day.[68] The best sources of vitamin D and calcium are in foods. Vitamin D and calcium supplements for the purpose of improving bone health have not been shown to be effective and are not recommended for healthy adults.[70] Recommendations regarding the use of Vitamin D for falls prevention in women 65 years and older are described in the *Health Promotion Across the Lifespan* chapter.

Muscle-strengthening and weight-bearing exercise has multiple benefits for the bone health (and overall well-being) of midlife and older women. Improved strength, balance, agility, and posture can reduce the risk of falling; falling has the potential to bring significant negative sequelae, such as fractures, pain, disability, and death. Exercise also has a modest effect on bone density; that is, bone mass increases in response to activities that place stress on bones.[65]

Body weight less than 127 pounds and a BMI less than 20 kg/mm^2 are risk factors for low

bone mass and increased fracture risk. The benefits of gaining weight should be discussed with very thin women, with recommendations made for approaches that can be used to achieve a healthy weight.[65,68]

In addition to their many other deleterious health effects, smoking and excessive alcohol intake have significantly negative effects on women's bones. Women smokers have lower bone mass, lose bone more quickly, and have significantly higher fracture risk than nonsmokers.[68] While moderate alcohol intake does not have any recognized detrimental effects on bones, consuming three or more alcoholic drinks per day increases the risk of osteoporotic fracture and falling.[65,68]

Pharmacologic Therapies for Osteoporosis Management

A focus on lifestyle strategies may be all that is necessary to promote bone health and minimize fracture risk for midlife and older women at low risk for fracture. However, midwives should consider pharmacologic therapy for women with any of the following: osteoporosis (T-score of –2.5 or lower), history of a low-trauma hip or vertebral fracture, or osteopenia (T-score between –1 and –2.5) *if* FRAX indicates a 10-year probability of 3% or greater of a hip fracture or a 10-year probability of 20% or greater of a major osteoporosis-related fracture.[65,68,69]

Estrogen in the form of ET or EPT has long been an FDA-approved agent for preventing osteoporosis, however, it is not approved as a treatment for this condition. Aside from estrogen, a variety of FDA-approved medications are available to prevent and/or treat osteoporosis, including bisphosphonates, raloxifene (Evista), calcitonin (Fortical), and teriparatide (Forteo). **Table 18-12** summarizes the medications in each category, dose, and basic prescribing issues. Adherence to pharmacologic osteoporosis treatment is often poor, with adherence rates in 6- to 12-month studies ranging from less than 25% to as high as 81%.[68] Thus, if the midwife and the woman decide that medication is indicated, it is important that the woman be educated about her fracture risk and the purpose of therapy, and then identify and eliminate barriers to appropriate use of that agent.[69] Dosing frequency seems to affect adherence, such that weekly dosing enhances adherence for many women when compared to daily dosing.[65,68]

Table 18-12 Medications for Treatment of Women with Osteoporosis

Medication Generic (Brand)	Dose	Comments
Bisphosphonates		
Alendronate (Fosamax)	Prevention: 5 mg daily or 35 mg weekly tablet or oral solution Treatment: 10 mg daily or 70 mg weekly tablet or oral solution	Bisphosphonates are considered by many experts to be first-line therapy for osteoporosis
Ibandronate (Boniva)	Prevention: 2.5 mg daily or 150 mg monthly tablet Treatment: 2.5 mg daily or 150 mg monthly tablet or 3 mg every	Choice of bisphosphonate is often

	3 months IV	made based on the desired dosing regimen
Risedronate (Actonel)	Prevention and treatment: 5 mg daily; 35 mg weekly; 35 mg weekly packaged with 6 tablets of 500 mg calcium carbonate; 75 mg on two consecutive days every month; and 150 mg monthly tablet	Take 30–60 min before eating in the morning, with 8 ounces water; sit/stand upright for 30–60 min
Zoledronic acid (Reclast)	Prevention: 5 mg IV every 2 years Treatment: 5 mg IV annually	Side effects for all oral bisphosphonates: difficulty swallowing, esophageal inflammation, gastric ulcers
Calcitonin		
Salmon calcitonin (Miacalcin; Fortical)	Treatment (> 5 years postmenopause): 200 IU daily nasal spray	FDA approved for treatment of women with vertebral osteoporosis but not osteoporosis at nonvertebral sites (e.g., hip) Other agents are more reliable at both vertebral and nonvertebral sites; calcitonin is not a first-tier therapy
Estrogen Agonist/Antagonists (EAAs) [previously known as selective estrogen receptor modulators (SERMs)]		
Raloxifene (Evista)	Prevention and treatment: 60 mg daily	Not only inhibits bone resorption, but also decreases risk of breast and uterine cancers Effective at reducing the risk of vertebral fractures but not hip or other nonvertebral fractures Can cause hot flashes Contraindicated in women with venous thromboembolism history
Parathyroid Hormone		
Teriparatide (Forteo)	Treatment (postmenopausal women at very high fracture risk): 20 mcg daily SQ	Indicated for women with established osteoporosis at very high risk of primary fracture Indicated for women with continued fractures despite use of other antiresorptive therapies

Abbreviations: FDA, U.S. Food and Drug Administration; IV, intravenous; SQ, subcutaneous.

Based on North American Menopause Society. Management of osteoporosis in postmenopausal women: 2012 position statement of the North American Menopause Society. *Menopause.* 2012;17:25-54[66]; National Osteoporosis Foundation. *Clinician's Guide to Prevention and Treatment of Osteoporosis.* Washington, DC: National Osteoporosis Foundation; 2010.[68]

Conclusion

Menopause is a healthy, natural life process. Similar to pregnancy, menopause is a time when women often present to the healthcare system. Midwives are well situated to care for women at midlife. Midwifery care during this phase of life includes development of an individualized plan for health promotion and disease prevention, as well as health screening and early identification and management of disease. As primary care providers, midwives have an opportunity to provide leadership in the delivery of holistic, woman-centered health care to women as they age without imposing negative biases.

Resources

Organization	Description	Webpage
North American Menopause Society (NAMS)	Organization focused on midlife and older women and that publishes the MenPro App, which can assist in shared decision making.	http://www.menopause.org
	Menopause Practice: A Clinician's Guide is a publication available for download that includes background information, evidence for health care, and guidelines.	http://www.menopause.org/publications/clinical-care-recommendations
FRAX: World Health Organization	*Fracture Risk Assessment Tool* is an instrument for determining risk of bone fractures.	http://www.shef.ac.uk/FRAX
Investigation of Women with Postmenopausal Uterine Bleeding	*Clinical Practice Recommendations* are guidelines for assessment and management of women with postmenopausal uterine bleeding.	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3951032/
National Center for Complementary and Integrative Health at the National Institutes of Health	Information about complementary and integrative health care.	https://nccih.nih.gov/health/menopause
Smiles for Life	*A National Oral Health Curriculum* has information for about oral health for all ages.	http://www.smilesforlifeoralhealth.org
American College of Cardiology and American Heart Association Task Force on Practice Guidelines	Heart Risk Calculator	http://www.cvriskcalculator.com
National Cancer Institute	Breast cancer risk assessment tool	https://www.cancer.gov/bcrisktool/
National Osteoporosis Foundation (NOF)	Information for professionals and patients	https://www.nof.org

References

1. Shifren JL, Gass MLS; NAMS Recommendations for Clinical Care of Midlife Women Working Group. The North American Menopause Society recommendations for clinical care of midlife women. *Menopause*. 2014:21(10);1038-1062.
2. Rousseau ME. Women's midlife health: reframing menopause. *J Midwifery Womens Health*. 1998:43(3):208-223.
3. Rousseau ME. Evidence-based practice in women's health: hormone therapy for women at menopause. *J Midwifery Womens Health*. 2001;46(3):167-180.
4. North American Menopause Society. *Menopause Practice: A Clinician's Guide*. 5th ed. Mayfield Heights, OH: North American Menopause Society; 2017.
5. Broekmans FJ, Soules MR, Fauser BC. Ovarian aging mechanisms and clinical consequences. *Endocr Rev.* 2009;30:465-493.
6. Fritz MA, Speroff L. Menopause and perimenopausal transition. In: *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:673-748.
7. Cockerham A. Adenomyosis: a challenge in clinical gynecology. *J Midwifery Womens Health*. 2012;57:212-220.
8. Solomon DA, Ruppert K, Greendale YL, Selzer F, Finkelstein JS. Medication use by race and ethnicity in women transitioning through the menopause: a Study of Women's Health Across the Nation drug epidemiology study. *J Womens Health* 2016;25(6):599-605.
9. Nosek M, Kennedy HP, Beyene Y, Taylor D, Gilliss C, Lee K. The effects of perceived stress and attitudes toward menopause and aging. *J Midwifery Womens Health*. 2010;55(4):328-334.
10. Hale GE, Robertson DM, Burger HG. The perimenopausal woman: endocrinology and management. *J Steroid Biochem Molec Biol*. 2014;142:121-131.
11. Harlow SD, Gass M, Hall JE, et al. Executive summary of the Stages of Reproductive Aging Workshop + 10: addressing the unfinished agenda of staging reproductive aging. *Fertil Steril*. 2012;97:843-851.
12. Singer D. "It's not supposed to be this way": psychological aspects of a premature menopause. *Counsel Psychother Res*. 2012;12(2):100-108.
13. Pickar JH, Archer DF, Kagan R, Pinkerton JV, Taylor HS. Safety and benefit considerations for menopausal hormone therapy. *Expert Opin Drug Saf*. 2017;16(8):941-954.
14. Fritz MA, Speroff L. Postmenopausal hormone therapy. In: *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:749-858.
15. Nelson HD. Menopause. *Lancet*. 2008;371:760-770.
16. Wildman RP, Sowers MR. Adiposity and the menopausal transition. *Obstet Gynecol Clin North Am*. 2011;38:441-454.
17. Nappi RE, Kokot-Kierepa M. Vaginal Health: Insights, Views & Attitudes (VIVA): results from an international survey. *Climacteric*. 2012;15:36-44.
18. Portman D. Genitourinary syndrome of menopause: new terminology for vulvovaginal atrophy from the International Society for the Study of Women's Sexual Health and the North American Menopause Society. **Menopause**. 2014;21(10):1063-1068.
19. Gandhi J, Chen A, Dagur G, et al. Genitourinary syndrome of menopause' an overview of clinical manifestations, pathophysiology, etiology, evaluation, and management. **Am J Obstet Gynecol**. 2016;215(6):704-711.
20. Freeman EW, Sherif K. Prevalence of hot flushes and night sweats around the world: a systematic review. *Climacteric*. 2007;10:197-214.
21. Fisher TE, Chervenak JL. Lifestyle alterations for the amelioration of hot flashes. *Maturitas*. 2012;71:217-220.
22. Smith R, Flaws JA, Gallicchio L. Does quitting smoking decrease the risk of midlife hot flashes? A longitudinal analysis. *Maturitas*. 2015;82(1):123-127.
23. Cray LA, Woods NF, Herting JR, Mitchell ES. Symptom clusters during the late reproductive stage through the early postmenopause: observations from the Seattle Midlife Women's Health Study. *Menopause*. 2012;19:864-869.
24. Daley A, Stokes-Lampard H, Thomas A, MacArthur C. Exercise for vasomotor menopausal symptoms. *Cochrane Database Syst Rev*. 2014;11:CD006108. doi: 10.1002/14651858.CD006108.pub4.
25. Saensak S, Vutyavanich T, Somboonporn W, Srisurapanont M. Relaxation for perimenopausal and postmenopausal symptoms. *Cochrane Database Syst Rev*. 2014;7:CD008582. doi: 10.1002/14651858.CD008582.pub2.
26. Alexander IM. The history of hormone therapy use and recent controversy related to heart disease and breast cancer arising from prevention trial outcomes. *J Midwifery Womens Health*. 2012;57(6):547-557.
27. Rossouw JE, Anderson GL, Prentice RL, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288(3):321-333.
28. Anderson GL, Limacher M, Assaf AR, et al. Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: the Women's Health Initiative randomized controlled trial. *JAMA*. 2004; 291(14):1701-1712.
29. Marjoribanks J, Farquhar C, Roberts H, Lethaby A, Lee J. Long-term hormone therapy for perimenopausal and postmenopausal women. *Cochrane Database Syst Rev*. 2017;1:CD004143. doi: 10.1002/14651858.CD004143.pub5.
30. Boardman HMP, Hartley L, Eisinga A, et al. Hormone therapy for preventing cardiovascular disease in post-menopausal

women. *Cochrane Database Syst Rev*. 2015;3:CD002229. doi: 10.1002/14651858.CD002229.pub4.
31. Charbonneau DH. Health literacy and the readability of written information for hormone therapies. *J Midwifery Womens Health*. 2013;58(3):265-270.
32. Furness S, Roberts H, Marjoribanks J, Lethaby A. Hormone therapy in postmenopausal women and risk of endometrial hyperplasia. *Cochrane Database Syst Rev*. 2012;8:CD000402. doi: 10.1002/14651858.CD000402.pub4.
33. Parish SJ, Gillespie JA. The evolving role of oral hormonal therapies and review of conjugated estrogens/bazedoxifene for the management of menopausal symptoms. *Postgrad Med*. 2017;129(3):340-351.
34. Formoso G, Perrone E, Maltoni S, et al. Short-term and long-term effects of tibolone in postmenopausal women. *Cochrane Database Syst Rev*. 2016;10:CD008536. doi: 10.1002/14651858.CD008536.pub3.
35. Scheffers CS, Armstrong S, Cantineau AEP, Farquhar C, Jordan V. Dehydroepiandrosterone for women in the peri- or postmenopausal phase. *Cochrane Database Syst Rev*. 2015;1:CD011066. doi: 10.1002/14651858.CD011066.pub2.
36. Lethaby A, Ayeleke RO, Roberts H. Local oestrogen for vaginal atrophy in postmenopausal women. *Cochrane Database Syst Rev*. 2016;8:CD001500. doi: 10.1002/14651858.CD001500.pub3.
37. Nelson HD, Walker M, Zakher B, Mitchell J. Menopausal hormone therapy for the primary prevention of chronic conditions: a systematic review to update the U.S. Preventive Services Task Force Recommendations. *Ann Intern Med*. 2012;157:104-113.
38. Weinstein MM. Hormone therapy and urinary incontinence. *Menopause*. 2012;19:255-256.
39. Manson JE, Aragaki AK, Rossouw JE, et al. Menopausal hormone therapy and long-term all-cause and cause-specific mortality: the Women's Health Initiative randomized trials. *JAMA*. 2017;318(10):927-938.
40. North American Menopause Society. Hormone products for postmenopausal use in the United States and Canada. 2012. Available at: http://www.menopause.org/docs/professional/htcharts.pdf. Accessed September 15, 2017.
41. Gaudard AMIS, Silva de Souza S, Puga MES, Marjoribanks J, da Silva EMK, Torloni MR. Bioidentical hormones for women with vasomotor symptoms. *Cochrane Database Syst Rev*. 2016;8:CD010407. doi: 10.1002/14651858.CD010407.pub2.
42. Pattimakiel L, Thacker HL. Bioidentical hormone therapy: clarifying the misconceptions. *Cleve Clin J Med*. 2011;78:829-836.
43. Moore TR, Franks RB, Fox C. Review of efficacy of complementary and alternative medicine treatments for menopausal symptoms. *J Midwifery Womens Health*. 2017;62(3):286-297.
44. National Center for Complementary and Integrative Health. Available at: https://nccih.nih.gov/health. Accessed September 15, 2017.
45. Lethaby A, Marjoribanks J, Kronenberg F, Roberts H, Eden J, Brown J. Phytoestrogens for menopausal vasomotor symptoms. *Cochrane Database Syst Rev*. 2013;12:CD001395. doi: 10.1002/14651858.CD001395.pub4.
46. Borrelli F, Ernst E. Alternative and complementary therapies for the menopause. *Maturitas*. 2010;66:333-343.
47. Guttuso T. Effective and clinically meaningful non-hormonal hot flash therapies. *Maturitas*. 2012;72:6-12.
48. Andrews JC. Vasomotor symptoms: an evidence-based approach to medical management. *J Clin Outcome Manage*. 2011;18:112-128.
49. Hall E, Frey BN, Soares CN. Non-hormonal treatment strategies for vasomotor symptoms: a critical review. *Drugs*. 2011;71:287-304.
50. Regestein QR. Is there anything special about valerian? *Menopause*. 2011;18:937-939.
51. Taavoni S, Ekbatani N, Kashaniayn M, Haghani H. Effect of valerian on sleep quality in postmenopausal women: a randomized, placebo-controlled clinical trial. *Menopause*. 2011;18:951-955.
52. Leach MJ, Moore V. Black cohosh (*Cimicifuga* spp.) for menopausal symptoms. *Cochrane Database Syst Rev*. 2012;9:CD007244. doi: 10.1002/14651858.CD007244.pub2.
53. Dodin S, Blanchet C, Marc I, et al. Acupuncture for menopausal hot flushes. *Cochrane Database Syst Rev*. 2013;7:CD007410. doi: 10.1002/14651858.CD007410.pub2.
54. Nayak C, Singh V, Singh K, et al. Management of distress during climacteric years by homeopathic therapy. *J Altern Complement Med*. 2011;17:1037-1042.
55. Elkins GR, Fisher WI, Johnson AK, Carpenter JS, Keith TZ. Clinical hypnosis in the treatment of postmenopausal hot flashes: a randomized controlled trial. *Menopause*. 2013;20(3):291-298.
56. Study of Women's Health Across the Nation (SWAN). Available at: http://www.swanstudy.org. Accessed September 14, 2017.
57. Rousseau ME, Gottlieb SF. Pain at midlife. *J Midwifery Womens Health*. 2004;49(6):529-538.
58. Rahn DD, Carberry C, Sanses TV, et al. Vaginal estrogen for genitourinary syndrome of menopause: a systematic review. *Obstet Gynecol*. 2014;124(6):1147-1156.
59. Cody JD, Jacobs ML, Richardson K, Moehrer B, Hextall A. Oestrogen therapy for urinary incontinence in post-menopausal women. *Cochrane Database Syst Rev*. 2012;10:CD001405. doi: 10.1002/14651858.CD001405.pub3.
60. Thomas HN, Hess R, Thurston RC. Correlates of sexual activity and satisfaction in midlife and older women. *Ann Fam Med*. 2015;13(4):336-342. doi: 10.1370/afm.1820.

61. Shifren JL. Increasing our understanding of women's sexuality at midlife and beyond. *Menopause*. 2011;18:1149-1151.
62. Hirschberg AL. Sex hormones, appetite and eating behaviour in women. *Maturitas*. 2012;71:248-256.
63. Suri V, Suri V. Menopause and oral health. *J Mid-Life Health*. 2014;5(3):115-120.
64. American College of Obstetricians and Gynecologists. Practice Bulletin No. 129: osteoporosis. *Obstet Gynecol*. 2012;120:718-734.
65. Tufts G. New treatment approach for osteopenia. *J Midwifery Womens Health*. 2011;56:61-67.
66. North American Menopause Society. Management of osteoporosis in postmenopausal women: 2012 position statement of the North American Menopause Society. *Menopause*. 2012;17:25-54.
67. Pollycove R, Simon JA. Osteoporosis: screening and treatment in women. *Clin Obstet Gynecol*. 2012;55:681-691.
68. National Osteoporosis Foundation. C*linician's Guide to Prevention and Treatment of Osteoporosis*. Washington, DC: National Osteoporosis Foundation; 2010.
69. Dunniway DL, Camune B, Baldwin K, Crane JK. FRAX counseling for bone health behavior change in women 50 years of age and older. *J Am Acad Nurse Pract*. 2012;24:382-389.
70. Zhao JG, Zeng XT, Wang J, Liu L. Association between calcium or vitamin D supplementation and fracture incidence in community-dwelling older adults: a systematic review and meta-analysis. **JAMA**. 2017;318(24):2466-2482.

PART IV

Antepartum

JOYCE E. ROBERTS

It is generally accepted that prenatal care is essential for achieving the best outcomes of pregnancy. However, the quality of prenatal care, when it is initiated, the number of "visits," the qualifications and expertise of the caregiver, the context of the care (e.g., birth center, clinic, hospital based, private or public), as well as the accessibility of secondary, consultative or medical care are all important to achieve positive health outcomes. Ideally, prenatal care will occur within a collaborative, multidisciplinary context wherein various health professionals are available to contribute to timely and accurate diagnosis of problems and offer appropriate interventions. Ultrasound, genetic diagnosis, and prenatal educational resources are especially important.

The chapters in this section address a wide range of healthcare services, including genetics, pregnancy, and medical complications that affect women during pregnancy. Prenatal care includes the consideration of guidelines, such as those developed by the U.S. Preventive Services Task Force, the assessment of pregnancy status, interpretation of screening tests, consideration of diagnostic strategies, and decisions about when to seek consultation and how to engage in collaborative care when the woman's needs merit medical care and management of complications.

The general health and reproductive history of a pregnant woman are critical antecedents to the healthy progression of a pregnancy and positive birth outcomes. It would be ideal if prenatal care was always preceded by preconception care, but often this is not the case. Lack of preconception care makes prenatal care especially important for achievement of optimal pregnancy outcomes, as well as an occasion to influence the future health of the woman—indeed, the health of her children and family and, ultimately, society—through timely recognition of health problems and the promotion of positive health practices. This type of care is especially critical as a means to identify chronic diseases and health behaviors that might be modified or addressed during the course of prenatal care. Key behaviors to be considered

include nutritional practices, substance use, and exercise.

The chapters in this section provide specific information regarding genetics; anatomic and physiological changes during pregnancy; the interpretation of pregnancy status, including the assessment of key physical parameters; and guidance regarding the recognition of health problems and complications unique to pregnancy.

A key initial consideration is the gestational age of the pregnancy—that is, the age of the fetus. Assessment and determination of gestational age is used to organize the topics addressed in this section and incorporated in the accompanying figures and tables that summarize key information. Gestational age is an essential "lens" through which the midwife should "hear" and "view" the information acquired in conversation with the woman and in clinical observation. It is a key parameter that helps the midwife achieve the purposes of prenatal care—namely, accurately assessing the status of pregnancy during a prenatal visit, recognizing problems through the interpretation of the woman's symptoms and the results of diagnostic tests, and offering timely interventions to achieve healthy pregnancy outcomes and a satisfying pregnancy and birth experience for the expectant mother.

Two basic dimensions of pregnancy are considered in providing prenatal care. The first is the physical health of the pregnant woman, the status of her pregnancy, and the growth and well-being of her fetus, including various screen modalities, laboratory tests, and genetic screening. The second is the experiential aspects of pregnancy and the woman's responses to her pregnancy. The chapters in this section provide essential content about the scope and content of prenatal care with consideration of the woman's emotional status, as well as the physical dimensions of pregnancy and fetal growth. This content includes the assessment of pregnancy status, the genetic basis of the fetal condition, the critical features of diagnostic tests, and the screening, recognition, and management of health problems and complications of pregnancy within a care setting that allows for consultation and multidisciplinary care. This information is presented in a systematic fashion that will enable the reader to interpret the objective data related to prenatal assessment and care and to use it in a way that reflects the best-practice principles used in providing prenatal care. Detailed tables and charts summarizing screening criteria, specific tests, and the range of results, along with listings of additional resources related to prenatal care, are included.

Given the limited accuracy of diagnostic testing and the uncertainty of the outcomes of interventions, it is essential that the midwife deliberately engage the woman in her own assessment of her situation and shared decision making. The midwife should ensure that the woman understands the information and alternatives available to her, and that the woman considers interventions in accordance with her beliefs and values.

These chapters will enable midwives to address the challenges of providing prenatal care—specifically, obtaining and interpreting information about the ongoing pregnancy, knowledgably recognizing health problems and complications of pregnancy, and using a deliberative approach within the interactive context of ongoing prenatal visits with pregnant women and their support persons. In addition, midwives are encouraged to maintain an orientation geared toward facilitating women's "readiness" for labor and ability to deal with the challenges of early parenting, including breastfeeding.

CHAPTER

19

Anatomy and Physiology of Pregnancy

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Introduction

Pregnancy is a time of profound anatomic and physiologic change. In addition to the reproductive organs, all maternal physiologic systems make adaptions needed to support the developing fetus and, at the same time, maintain maternal homeostasis. A thorough understanding of these changes is an essential foundation for all healthcare providers—including midwives—who care for women during pregnancy. This chapter provides an overview of changes in the reproductive organs, the effect of the major hormones of pregnancy, fetal development, maternal adaptations that take place during pregnancy.

The Reproductive Organs During Pregnancy

Pregnancy lasts approximately 266 days or 38 weeks from ovulation (postconceptional weeks). This translates into 10 lunar months or 9 calendar months (because some months have 5 weeks). If dating occurs from the last normal menstrual period, some sources note the duration of human pregnancy is 280 days or 40 completed weeks' gestation. When counting gestation in weeks, it is important to remember that the number changes after the week is completed. Therefore 23 5/7 weeks' gestation is 23 completed weeks since the last menstrual period and an additional 5 days. This translates to 21 completed weeks and 5 days since conception.

Over the course of pregnancy, a woman's breasts grow and prepare for lactation. The uterus increases to approximately 5 times its normal size; at 38 gestational weeks, the uterus measures approximately 32 centimeters long, 22 centimeters across, and 24 centimeters wide. The cervix must first act as a barrier maintaining the uterine contents. However, at the end of pregnancy, the cervix becomes soft and short, opening to allow passage of the fetus during birth. These remarkable changes are the result of a complex interplay of hormonal stimulation that has important clinical implications for all women, but especially those who have congenital uterine anomalies, as well as those who experience miscarriage, preterm labor, preeclampsia, and other pregnancy complications.

The Breast

Under the direction of several different pregnancy hormones, the breast undergoes two distinct developmental changes to prepare for lactation during pregnancy. Both stages—mammogenesis and lactogenesis I—include hyperplasia and hypertrophy.[1] Hyperplasia refers to an increase in the number of cells, or cellular proliferation. Hypertrophy refers to enlargement of the cells; that is, cells grow in size.

Mammogenesis begins early in pregnancy. Breasts enlarge via cellular hyperplasia, and the breast lobules increase in size. The nipples become erectile, the areola becomes proportionately larger and darker, and superficial veins can become visible. During this process, an individual may feel her breasts are tender or even painful. Alveoli expand and proliferate at the end of breast lobules; the lobules proliferate as well.

Toward the middle of pregnancy, the alveoli epithelial cells change into secretory epithelium, which is the first stage of lactogenesis. Toward the end of pregnancy, the alveoli secrete colostrum but are primarily quiescent secondary to inhibition by progesterone, one of the primary pregnancy hormones. After birth, the influence of progesterone abruptly ceases and lactogenesis II—that is, the onset of milk production—begins. A more detailed description of lactogenesis II is presented in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

The Uterus

The three layers of the uterus (endometrium, myometrium, and perimetrium) become clearly defined over the course of pregnancy. The uterus grows at a steady, predictable rate during pregnancy, with its expansion first becoming detectable at approximately 5 weeks' gestation. The initial uterine growth occurs in the anteroposterior diameter, while the isthmus or lower segment of the uterus can become very soft. This softening results in marked compressibility in the lower uterine segment that is present for a short period of time at approximately 4 to 6 weeks after the first day of the woman's last menstrual period started, and approximately 2 weeks after conception. This unusual compressibility, called *Hegar's sign*, is a probable sign of pregnancy. As seen in **Figure 19-1**, during a bimanual examination, the lower segment of the uterus is so soft that it can be easily compressed between the fingers of the examiner's two hands (one on the abdomen and the other gloved hand in the posterior fornix of the vagina).

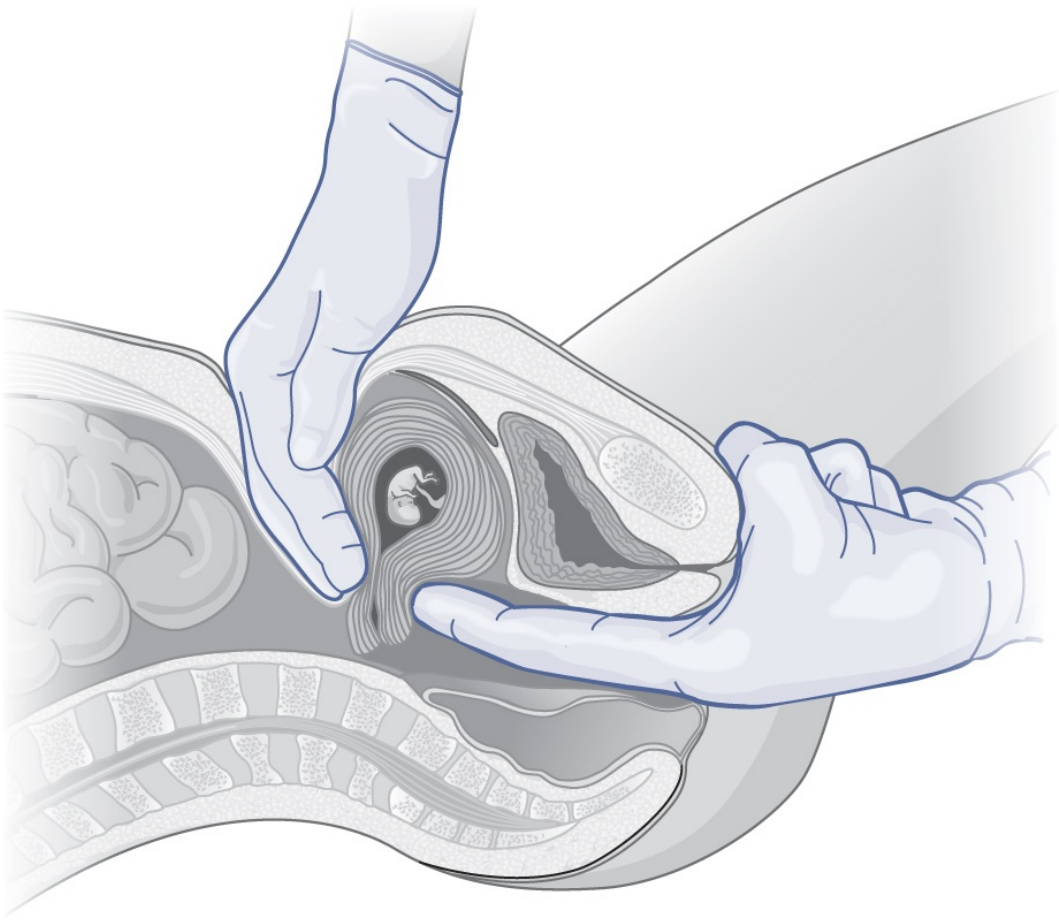


Figure 19-1 Hegar's sign.

The uterine shape changes from the nonpregnant pear shape to a ball or sphere in the first trimester, and then expands to an elongated cylinder. The anatomic location of the uterus in relation to maternal anatomy is illustrated in **Figure 19-2**.

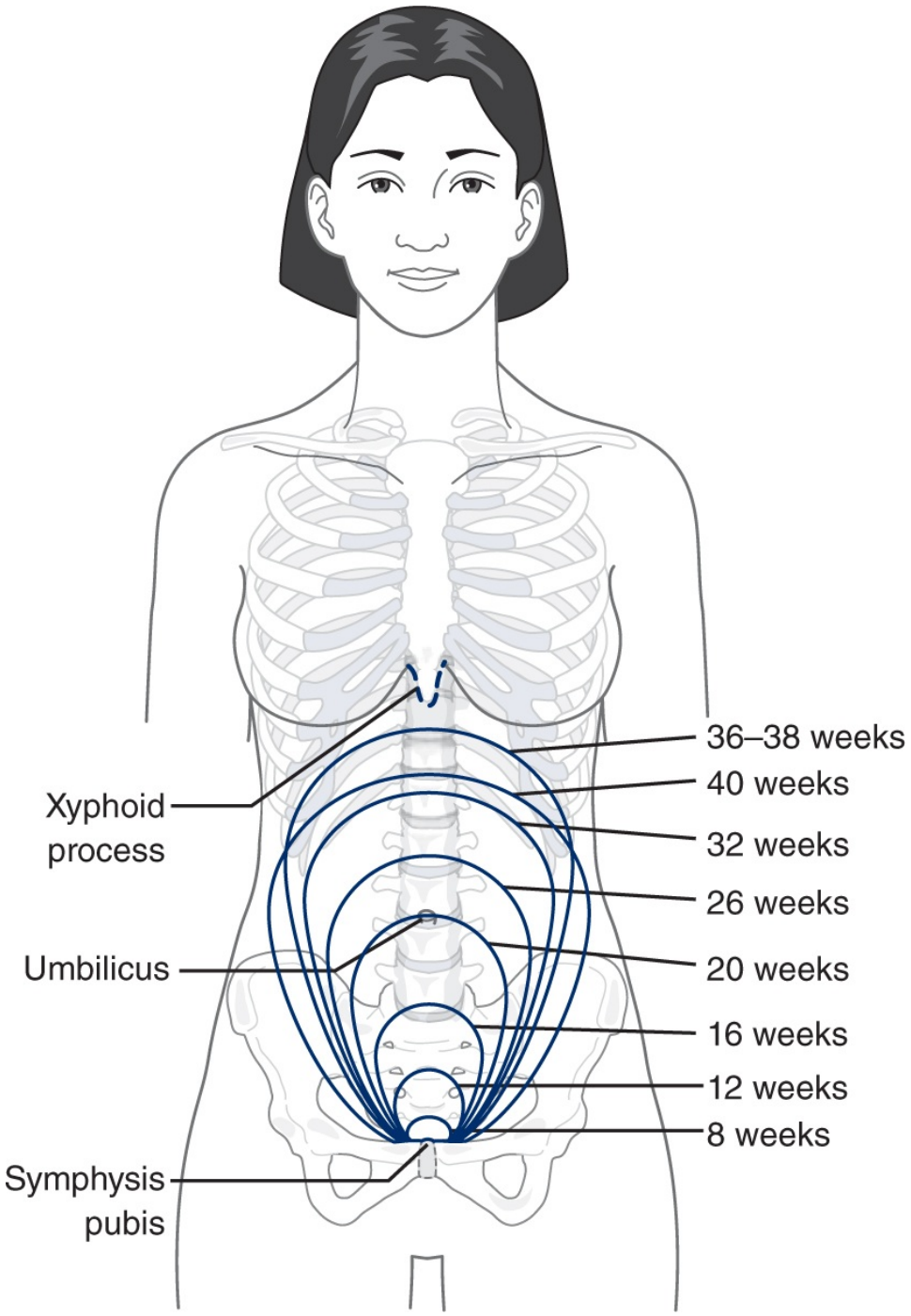


Figure 19-2 Approximate normal fundal heights during pregnancy.

The uterine round ligaments attach on either side of the uterus just below and in front of the insertion of the fallopian tubes; they then cross the broad ligament in a fold of peritoneum, pass through the inguinal canal, and insert in the anterior (upper) portion of the labia majora on either side of the perineum. The ligaments are composed largely of smooth muscle that is continuous with the smooth muscle of the uterus. The round ligaments hypertrophy during pregnancy and stretch as the uterus enlarges. During periods of rapid uterine growth, women may feel stretching or sharp pain in the inguinal area when moving or turning, which is secondary to additional torsion or stretching of the round ligament.

Growth of the uterus is due to two processes: (1) estrogen- and progesterone-induced

hyperplasia of uterine smooth muscle cells within the myometrium during early pregnancy and (2) hypertrophy of the uterine muscles later in pregnancy. The muscles increase their content of actin, myosin, sarcoplasmic reticulum, and mitochondria, which collectively serve as the machinery used to contract the muscles during labor and birth, as described in the *Anatomy and Physiology During Labor and Birth* chapter. The myometrium thus has both properties of contractility and elasticity. Contractility allows for lengthening and shortening, whereas elasticity refers to the ability to stretch.

Transformation of the Endometrium into the Decidua

The secretory endometrium contains columnar epithelium, epithelial cells, and nonresident or migratory immune cells and spiral arteries. The radial arteries that supply the myometrium branch into basal arteries, which become the spiral arteries that supply part of the myometrium and the endometrium.[2]

The endometrial changes that allow and facilitate implantation are collectively termed the *decidual reaction*. The name "decidua" was chosen for this process because, like the leaves on deciduous trees, the transformed tissue is shed after birth.[3] The decidual reaction occurs in response to estrogen, progesterone, and a complex dance or "cross-talk" of locally produced chemicals generated by the blastocyst and maternal endometrium.

Approximately 8 days after ovulation, the secretory endometrium provides a 4- to 5-day "window of opportunity" for blastocyst implantation.[4] During this short span of time, immune cells congregate, the endometrial glands become more secretory, and the epithelial surface develops small protrusions called pinopods and cell-adhesion molecules.[5] The pinopods absorb fluid from the uterine cavity and likely play key roles in attracting the blastocyst. Once the blastocyst is in contact with the endometrium, the pinopods and trophoblastic protrusions interact to facilitate implantation (**Figure 19-3**).[6] It is thought that physiologic variations in the development of the endometrial "window of opportunity" could result in failure of implantation; this hypothesis is currently the subject of infertility research.

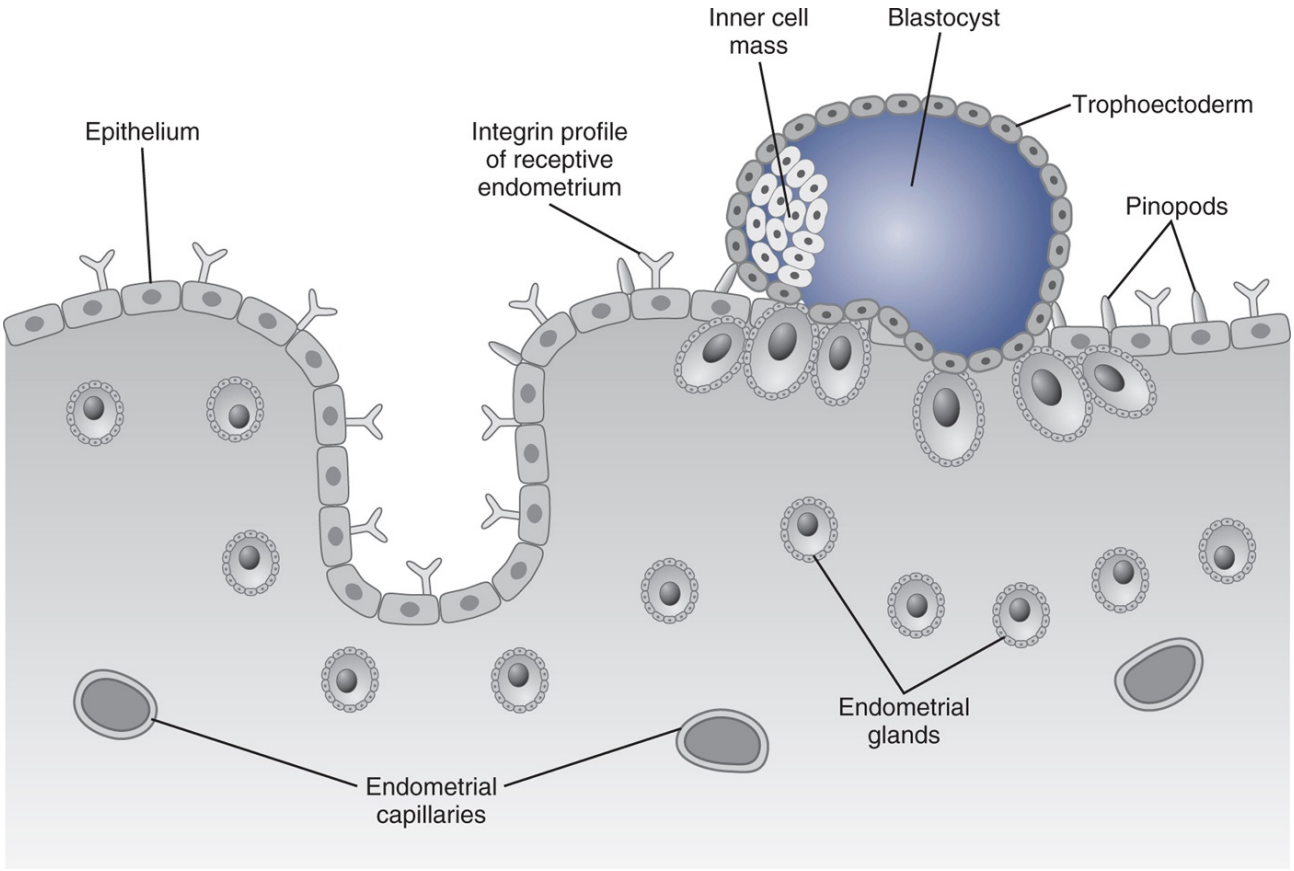


Figure 19-3 Implantation of the blastocyst.

The decidual reaction starts once the blastocyst is present at the endometrial site of implantation. The endometrial stromal cells become larger, round, decidual cells that have a membrane to which the trophoblast can anchor itself, and another surge of chemical "cross-talk" occurs.[7]

The decidua fulfills multiple roles, including a key role in directing trophoblastic invasion and provision of nutrition to the embryo in the early stages of embryology. The entire maternal decidua is divided into three regions: the *decidua basalis*, the *decidua capsularis*, and the *decidua parietalis*. These three regions are named for their positional relationship to the conceptus. The decidua basalis is the site where implantation occurs and is the future site of the maternal potion of the placenta. It is subdivided into a *zona compacta* and a *zona spongiosa*, the latter of which is the site of detachment of the placenta in the third stage of labor. The decidua capsularis lies like a capsule around the chorion, whereas the decidua parietalis is found on the opposite uterus wall. At approximately the fourth month of gestation, the decidua capsularis comes into contact with the decidua parietalis. The merging of these two causes the uterine cavity to become obliterated.

Uterine Innervation

Changes in uterine innervation are among the most interesting physiologic effects of pregnancy. The uterus is primarily innervated by the sympathetic nervous system, with some fibers coming from the parasympathetic nervous system and cerebrospinal tract. Innervation is not consistent

throughout the uterus; for example, more nerve fibers are found near the cervix as compared to the fundus. Sympathetic nerve fibers in the uterine fundus virtually disappear during pregnancy, whereas those in the cervix remain.[8,9] Although the reasons for these changes are not clear, it is postulated that the lack of sympathetic innervation protects the uterus from sympathetic stimulation via catecholamines and resultant contraction activity. Uterine contractions during pregnancy and labor occur secondary to endocrine stimulation rather than via muscle fiber nerve stimulation.

Cervical Remodeling During Pregnancy

In nonpregnant women, the cervix is an average of 3 centimeters long.[10-12] In essence, it is a tubular structure, made up of two major structures: the *ectocervix* and the *endocervix*. The ectocervix, which is visible from the vagina, comprises an external layer of columnar epithelial cells and a layer of squamous epithelial cells. The endocervix is the internal, canal-like portion of the cervix, which opens into the uterus. It is covered with a single layer of columnar epithelial cells. The cervical openings are referred to as the *internal os* and *external os*, as described in the *Anatomy and Physiology of the Female Reproductive System* chapter. In nulliparous women, the opening of the endocervix at the external os is pinpoint and circular in size; in contrast, in multiparous women, the external os has a slit-like appearance.

Glandular tissue in the cervix produces thick, tenacious mucus, which forms the mucus plug sealing the endocervical canal. This mucus plug helps prevent ascending bacteria or pathogens from entering the uterine cavity during pregnancy.

During pregnancy, the cervix is composed primarily of connective tissue covered by a thin layer of smooth muscle, which itself is covered by the columnar epithelial cells. The extracellular connective tissue within the cervix contains protein (in the form of collagen and elastin) and proteoglycans (primarily hyaluronic acid and decorin); it is referred to as the *extracellular matrix*. Approximately 80% of the cervix comprises extracellular matrix and 15% is smooth muscle, but the distribution is quite heterogeneous.[10-14] The area closest to the internal os has more smooth muscle, whereas the area closer to the external os has more extracellular matrix. The strength of the collagen tissue depends on the type and number of crosslinks between the collagen microfibrils.

Over the course of pregnancy, labor, birth, and the postpartum period, the cervix normally undergoes four distinct phases: (1) softening, which is also called *remodeling*; (2) accelerated softening at the end of pregnancy, referred to as *ripening*; (3) dilation; and (4) repair.[13] Under the influence of estrogen in pregnancy, the cervix first begins to soften approximately 4 weeks after the first day of a woman's last menstrual period. This cervical softening, called *Goodell's sign*, is one sign of pregnancy.[11] As vascularization increases, a cyanosis or bluish-purple discoloration called *Chadwick's sign* develops. Chadwick's sign is usually first evident at 6 to 8 weeks' gestation.

Following the initial relatively rapid softening, the cervix continues to soften throughout the pregnancy, albeit at a slower rate. The collagen becomes more soluble and compliant, but does not lose structural integrity. Several factors can adversely influence cervical architecture during pregnancy, including infection, genetic factors, and previous surgical procedures that involved the cervix. Usually the cervix remains between 30 and 40 millimeters in length throughout gestation. Women whose cervix shortens to less than 20 millimeters are at increased risk for preterm labor.

Cervical Ripening Prior to Labor

"Cervical ripening" is the term used to describe the process of accelerated remodeling and softening that begins weeks prior to the onset of labor. Cervical remodeling and activation of

uterine contractions are the two primary physiologic events that are associated with the initiation of parturition or labor. Cervical ripening results from a series of interactions between hormonal and mechanical factors that have not been fully elucidated. As progesterone levels fall and estrogen levels rise, the water content and vascularization of the cervix increase and the collagen cells become disorganized, which results in a marked reduction in the mechanical strength of the collagen bundles.

The local paracrine activity of prostaglandins PGE2 and PGF2a also influence the cervical ripening and the onset of labor. Fetal production of corticotropin-releasing hormone and cortisol causes an upregulation of prostaglandin receptors in the cervix and uterus. PGE2 facilitates cervical vasodilation. Finally, production of pro-inflammatory cytokines leads to infiltration of the cervix by leukocytes and macrophages. These cells release enzymes that facilitate alterations in extracellular matrix proteins, loosening of collagen fibers, and a reduction in collagen content.

Cervical Effacement and Dilatation

Toward the end of gestation and extending into early labor, the cervix shortens or thins so that there is no discernable length between the external os and the internal os. This process, which occurs in response to uterine contractions, is referred to as *effacement*. The cervix also begins to *dilate* as the process continues. Effacement and dilatation generally occur during early labor in nulliparous women but may occur prior to progressive labor in multiparous women. However, the onset of labor is not often discrete. There is significant variability between individuals with regard to the degree of effacement and dilatation that has occurred when labor begins.

Many women notice an increase in vaginal secretions during pregnancy called *leukorrhea*. This phenomenon likely occurs because, in addition to normal vaginal secretions produced by the epithelial cells, the cervical glands secrete an increased amount of mucus at this time in order to form the cervical mucus plug. These cervical and vaginal secretions likely form the basis of the physiological discharge of leukorrhea.

Hormones of Pregnancy

The placenta is the primary physiologic interface between the maternal and fetal compartments and is a central mediator for chemical messages between the fetus and the pregnant woman. The primary hormones of pregnancy produced by the placenta are estrogen, progesterone, human chorionic gonadotropin (hCG), and human placental lactogen (hPL). Each of these hormones has a major role in supporting pregnancy (**Table 19-1**). In addition, the placenta and the fetus synthesize other chemical mediators that act both locally and systemically to support growth and development of the fetus and placenta itself; many of these mediators are still being discovered.

Table 19-1 **Major Hormones and Functions During Pregnancy**

Hormone	Source	Selected Functions During Pregnancy
Human chorionic gonadotropin (hCG)	Syncytiotrophoblast	Stimulates production of progesterone from the corpus luteum
	Placenta	Prevents degeneration of the corpus luteum, thereby ensuring ongoing estrogen and progesterone production
		Promotes formation of syncytiotrophoblast during trophoblastic invasion
		Aids spiral artery remodeling during the process of trophoblastic invasion
		Promotes angiogenesis in uterine vasculature
		Stimulates thyroid production of thyroxine in the first trimester
		Suppresses myometrial contractions
Human placental lactogen (hPL)	Placenta	Increases insulin resistance
		Stimulates production of growth hormones
Progesterone	Corpus luteum	Promotes systemic vasodilation
	Placenta	Has anti-inflammatory actions to protect trophoblast from being rejected
		Supports decidualization within the endometrium
		Prevents myometrial contractility
		Inhibits uterine production of prostaglandins
		Supports mammary growth for lactation
		Withdrawal at term leads to uterine contractions
Estrogen	Ovaries	Softens collagen fibers in cervix and ligaments
	Corpus luteum	Increases uterine blood flow
	Placenta	Promotes growth of the uterus and breast glandular tissue
	Fetus	Increases production of insulin-like growth factors
		Enhances myometrial contractility
		Increases myometrial sensitivity to oxytocin, may upregulate oxytocin receptors

Human Chorionic Gonadotropin

Human chorionic gonadotropin is a glycoprotein with both alpha and beta subunits. The alpha subunit is structurally the same as luteinizing hormone (LH), follicle-stimulating hormone (FSH), and thyroid-stimulating hormone (TSH). The beta unit can have various forms depending on the tissue that produces it and is the basis of modern qualitative pregnancy tests.[15-17] Serial plasma levels of beta-hCG can be used to provide quantitative levels used to diagnose and monitor development of the early conceptus.

Human chorionic gonadotropin is secreted by the syncytiotrophoblast tissue within the blastocyst before implantation occurs, and then later by the placenta. The first role of hCG is to sustain estrogen and progesterone production in early pregnancy by preventing degeneration of the corpus luteum. The level of the free beta unit of hCG (beta-hCG) rises in early pregnancy and is first detectable approximately 8 to 10 days after ovulation, or shortly before the first missed menses. This timing coincides with implantation of the fertilized ovum.[18] This level doubles approximately every 48 to 72 hours in 85% of women with normal pregnancies, maintaining this rate of increase until it reaches a peak of approximately 100,000 mIU/mL at 8 to 11 gestational weeks. At this point, the plasma level of beta-hCG slowly decreases to a stable level of approximately 20,000 mIU/mL (**Figure 19-4**).[18]

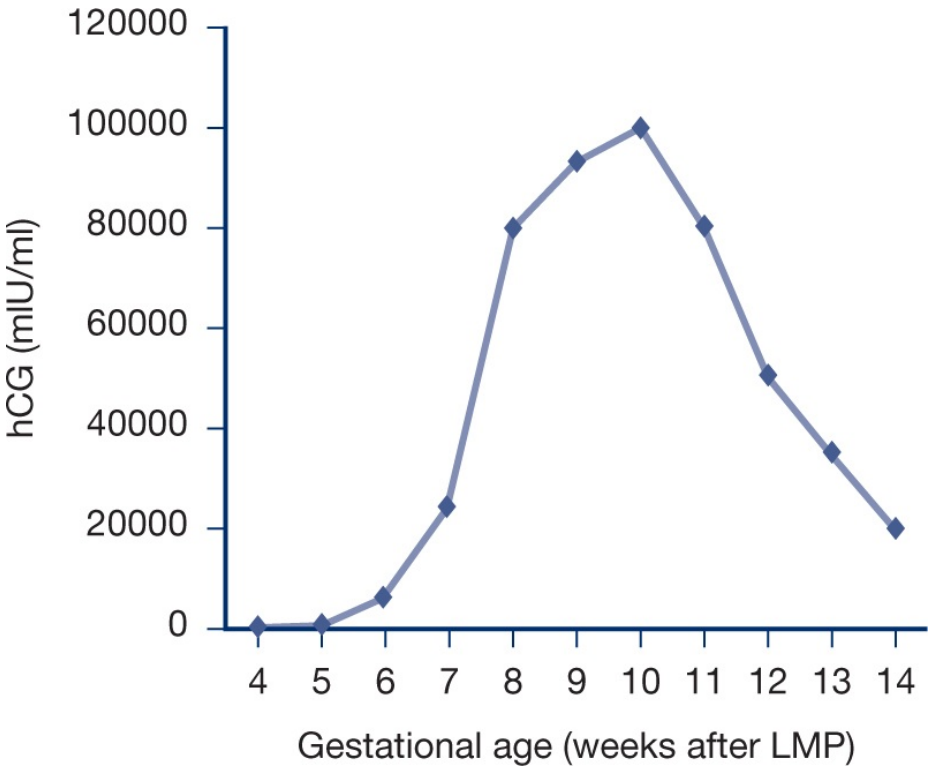


Figure 19-4 Human chorionic gonadotropin values in the first trimester of pregnancy.

Abbreviations: hCG, human chorionic gonadotropin; LMP, last menstrual period.

The characteristic doubling time of hCG has been used in serial measurements of blood values to assess the viability of pregnancy. Because there is wide individual variability in hCG levels, however, some patterns of serial hCG levels (e.g., slower than expected increases) can be difficult to interpret. Moreover, although an hCG level may be obtained every 2 days if clinically indicated, assessing these values every 48 hours can sometimes be too soon to see a

full doubling of the hCG plasma level.[19] Serial quantitative measures of the beta subunit are used to determine the viability of a pregnancy, as described in the *Prenatal Care* and *Medical Complications in Pregnancy* chapters. Plasma levels of the beta subunit are also used as a marker for tumors that have an embryologic origin, such as choriocarcinoma and hydatidiform mole.

The natural rise and fall of hCG levels also corresponds to the natural history of nausea and vomiting in the first trimester. Although the direct etiology of nausea and vomiting in pregnancy is not known, it has been postulated that hCG may play a role. Nevertheless, studies that have linked hCG levels to nausea symptoms have not shown a direct correlation between the two. Women who have severe nausea and vomiting or hyperemesis may also have subclinical hyperthyroidism; in such women, the alpha subunit of hCG is able to stimulate the thyroid gland as though it were TSH.[20]

Human Placental Lactogen

Human placental lactogen, which is structurally similar to growth hormone, ensures adequate fetal nutrition by altering maternal glucose metabolism so that glucose is available for fetal uptake. This transformation is achieved primarily by mobilizing fatty acids from lipids to provide an alternative fuel for the pregnant woman while sparing glucose for the fetus. hPL also increases maternal insulin resistance, thereby assuring consistent blood levels of glucose for fetal use. Glucose is transported across the placenta via facilitated diffusion.

The fetus relies primarily on glucose for nutritional needs; fetal production of glucose either does not occur or is undetectable.[21] It appears that the fetal liver does not develop the mechanism necessary for gluconeogenesis until just before birth.

The hPL-induced insulin resistance results in an increase in maternal insulin levels. This, in turn, stimulates amino acid production, thereby ensuring that amino acids are also available for fetal growth and development.

hPL is secreted by the placental syncytiotrophoblast. This agent is detectable in maternal circulation at 6 to 8 gestational weeks, with levels increasing in direct proportion to placental growth.

Progesterone

Progesterone and estrogen—both steroid hormones—act as intracellular chemical messengers by binding to intracellular receptors. These hormones influence many aspects of DNA transcription and cellular activities.

Progesterone is essential for maintenance of pregnancy in all mammals, and its name is derived from this function: “pro-gestational steroidal ketone.” This hormone maintains the uterus in a quiescent state and helps suppress the maternal immune response to fetal antigens so that the fetal tissue is not rejected. Progesterone is produced by the corpus luteum in early pregnancy. The placenta then assumes the progesterone production at some point during gestational weeks 7 to 9, an event called the “luteal-placental shift.” Additional functions of progesterone during pregnancy are listed in **Table 19-1**.

Low levels of progesterone in pregnancy are associated with an increased risk of spontaneous abortion. Women with luteal-phase defects can have progesterone levels that drop in early pregnancy, leading to spontaneous abortions. Progesterone supplementation has been shown to be effective for treating threatened miscarriage.[22,23] Progesterone is also used to prevent preterm birth in women who had a previous preterm birth.[24] Alternatively, administration of mifepristone, a progesterone receptor antagonist, will cause miscarriage.

Estrogen

Three types of naturally occurring estrogen have been identified: estrone, estradiol, and estriol. Estriol (E_3) is the primary estrogen of pregnancy. Estrogen production during pregnancy entails a three-part interplay between the woman, fetus, and placenta; each of these three separate but interrelated factors completes part of estrogen synthesis but not all of it. Estrogen is initially synthesized in the corpus luteum until the eighth or ninth week of gestation. At that point, the fetal adrenal glands are mature enough to produce the necessary estrogen precursors, and the placenta is able to produce and excrete the active forms of estrogen. Placental estrogen production depends on input from both the fetal and maternal adrenal cortex because the placenta cannot produce the androgenic C19 steroid dehydroepiandrosterone (DHEA), and its sulfoconjuate, DHEA-S, which are essential substrates of estriol.

Selected effects of estrogen during pregnancy are listed in **Table 19-1**. Estrogen encourages growth of breast tissue, stimulates uterine contractility, and increases uterine receptiveness to oxytocin. For most of pregnancy, however, the uterus remains refractory to the effects of estrogen—a consequence of the influence of progesterone, which ensures that the uterine myometrium has very few estrogen receptors.

Fertilization and Implantation

The processes that make it possible for the single cell that results from the fusion of the ovum and the sperm to develop and mature within the course of months are the subject of many fields of study, including genetics, embryology, and fetology. It is beyond the scope of this chapter and this text to cover these topics in detail. Instead, this chapter presents an overview of the knowledge needed by a practicing midwife with regard to the milestones of embryologic and fetal development and the intricate and vulnerable steps that create organs and organ systems. This understanding will help the midwife discuss embryonic and fetal development with the woman and her family and also provides general knowledge regarding the causes of congenital malformations.

Fertilization

Fertilization is the process of fusion of two haploid (containing 23 chromosomes) cells—a sperm and an ovum—to form a diploid (containing 46 chromosomes) cell or zygote. Once a sperm cell binds to receptors on the oocyte membrane, its nucleus is pulled into the cytoplasm of the oocyte and the oocyte membrane depolarizes, causing destruction of other sperm cells. This prevents the binding of other sperm cells and, therefore, polyspermy. Once the sperm enters the ovum, the oocyte, which was arrested in the metaphase of the second meiotic division, completes metaphase. As the nuclei of the ovum and the sperm swell, fertilization occurs, the nuclear membranes disappear, and pairing of the chromosomes occurs to create the diploid zygote (**Figure 19-5**).

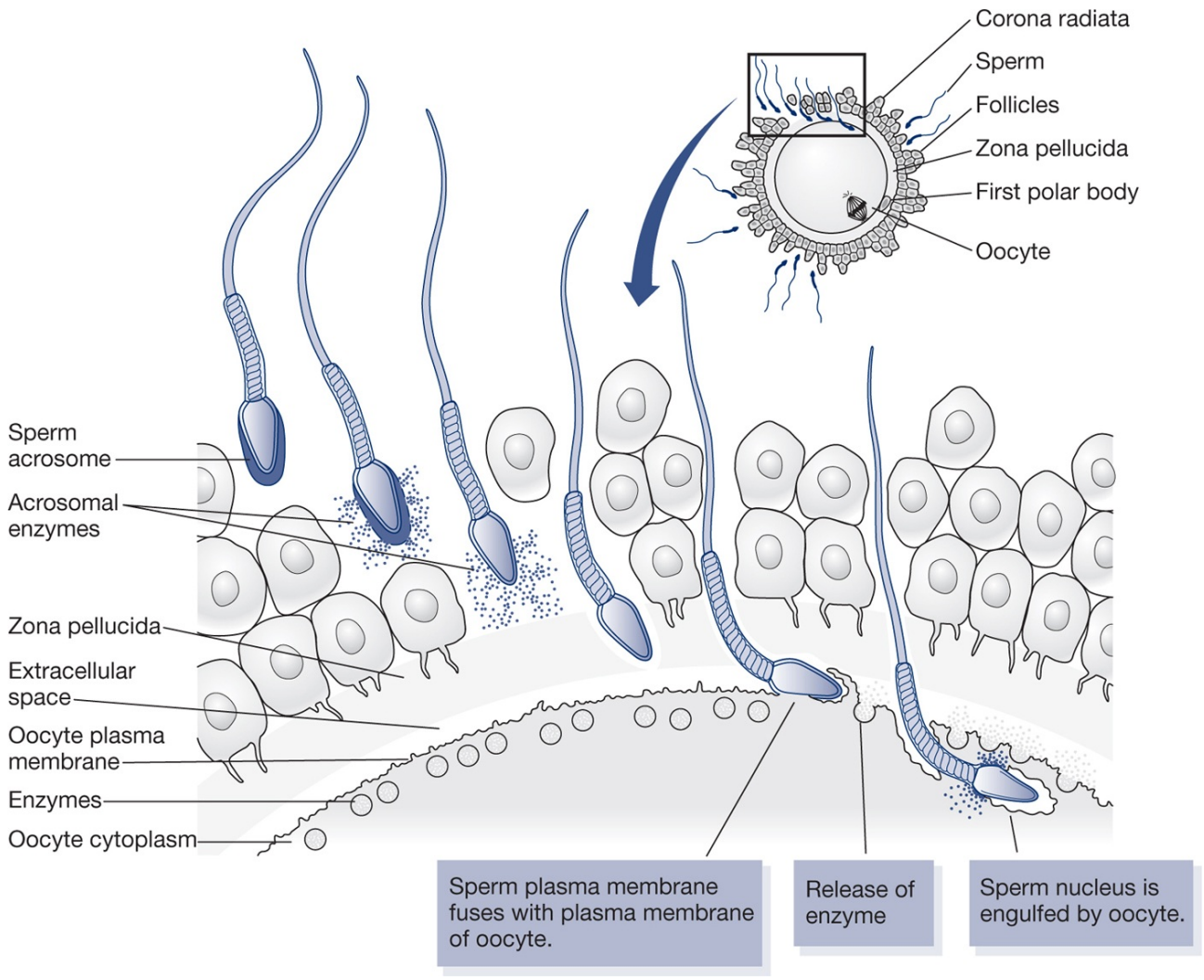


Figure 19-5 The process of fertilization.

Fertilization usually occurs in the fallopian tube and takes approximately 18 to 24 hours. The fertilized oocyte becomes known as a *zygote*. The zygote begins as a single cell that contains 46 chromosomes: 23 from the ovum and 23 from the sperm. At this stage, the genetic code for that individual is formed. The 23rd pair of chromosomes determines the fetus's sex. The ovum contains only an X chromosome, so the X or Y chromosome that pairs with the maternal X chromosome is donated by the sperm.

Blastocyst

As the zygote moves through the fallopian tube into the uterine cavity, the cells divide (rapid mitotic activity) and create additional cells. These cells, known as blastomeres, are held together by the zona pellucida (an extracellular glycoprotein matrix) and form a solid ball of 12 to 16 cells known as the *morula*. As the morula enters the uterus, which occurs approximately 4 days after fertilization, intracellular fluid increases and a central cavity forms. The zygote is now called a *blastocyst*. The blastocyst is composed of four components: (1) the zona pellucida, (2) an inner cell mass, (3) an outer layer of cells called the trophoblast, and (4) a fluid-filled cavity. The trophoblast eventually forms the placenta and chorion, while the inner

cell mass develops into the embryo and amnion.

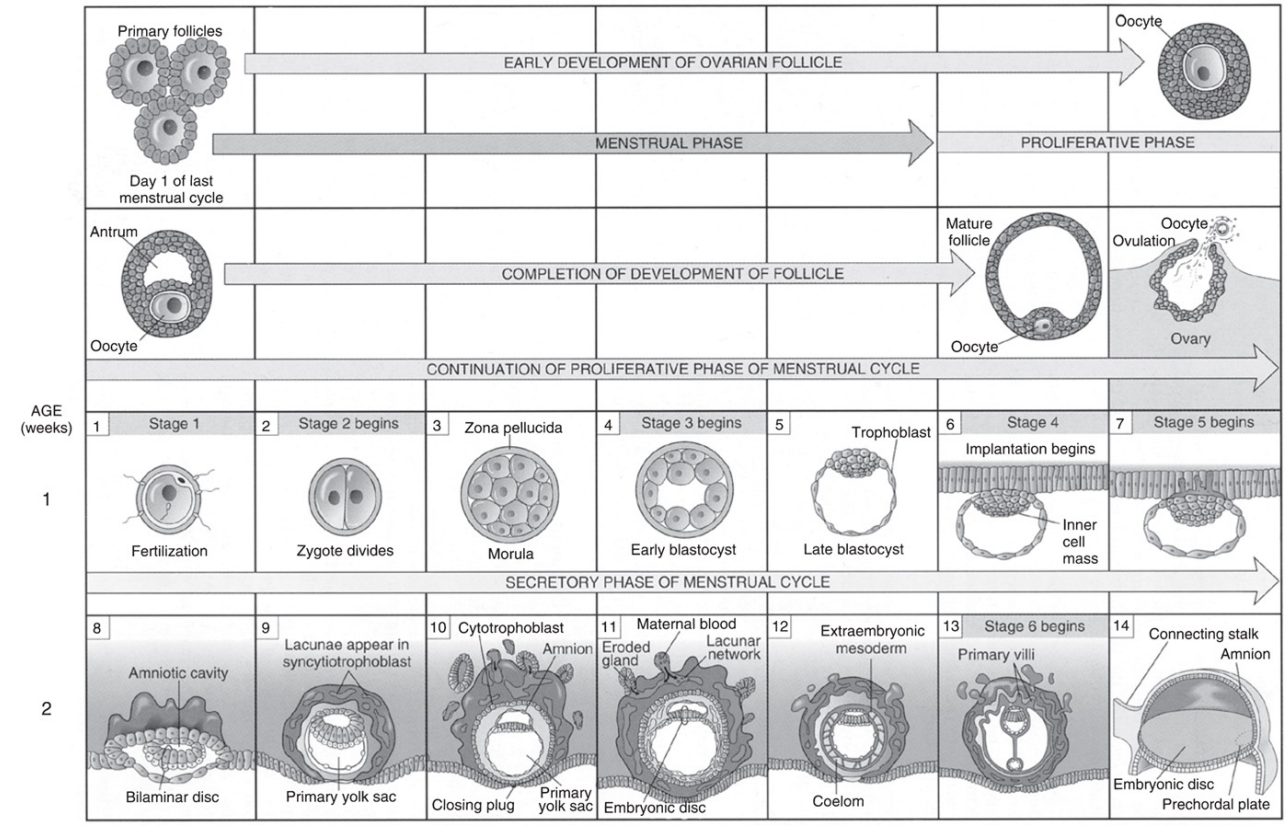
The blastocyst is said to “hatch” when it sheds the zona pellucida. At approximately 10 to 12 days post conception or 1 to 2 gestational weeks, the blastocyst implants in the decidua.[25] The blastocyst is guided to a receptive area, usually in the fundus, for implantation. It orients itself so that the embryonic pole is closest to the endometrium, and the process of implantation begins.

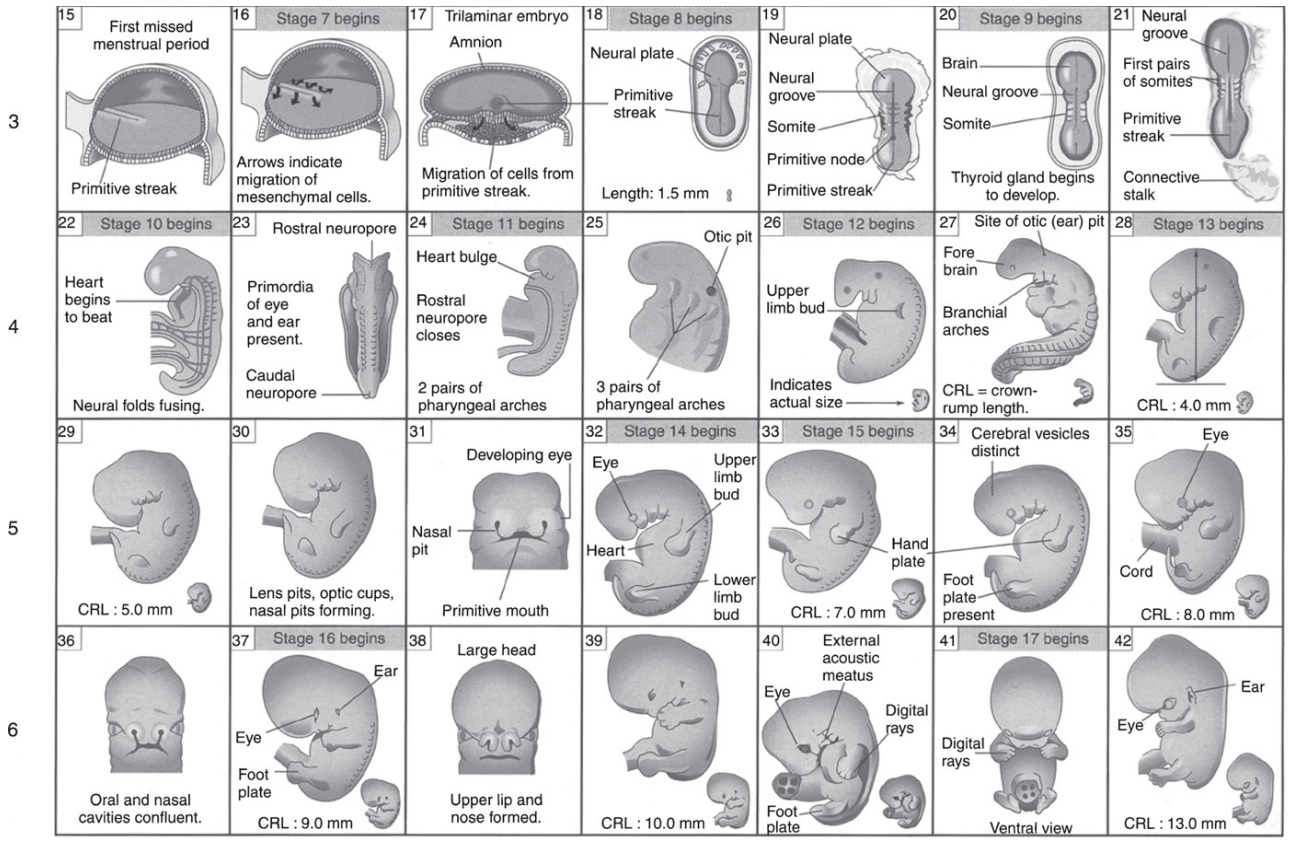
The Trophoblast and Implantation

The trophoblast is unique tissue playing an essential role in pregnancy. The trophoblast, in contact with the endometrium, first differentiates into two distinct tissues: the syncytiotrophoblast and the cytotrophoblast. The *syncytiotrophoblast* is a syncytium or multinucleate protoplasmic mass formed by the fusion of cells, some of which were endometrial decidual cells. The syncytiotrophoblast burrows into the endometrium and remains in contact with maternal blood throughout gestation.[25] This tissue becomes the primary source of hCG.[26] The implantation process can result in light vaginal bleeding or spotting, referred to as “implantation bleeding,” and can be mistaken for a light menses.

As the syncytiotrophoblast invades the endometrium, it disrupts maternal endometrial capillaries, portions of which are engulfed and become the lacunae. This process initiates the lacunar stage, which is marked by growth of small vacuoles in the syncytiotrophoblast that multiply and eventually fuse to form a system of *lacunae*. Initially, these lacunae fill with a substance derived from glandular secretions of the endometrium and a filtrate of maternal blood that diffuses through the trophoblastic tissue and serves to nourish the embryo. The lacunar networks enlarge and communicate with each other as they evolve into the intervillous space (IVS). *The intervillous space* will fill with maternal blood and bathe the placental villi as they form into this space (**Figure 19-6**).[27]

TIMETABLE OF HUMAN PRENATAL DEVELOPMENT
1 TO 6 WEEKS





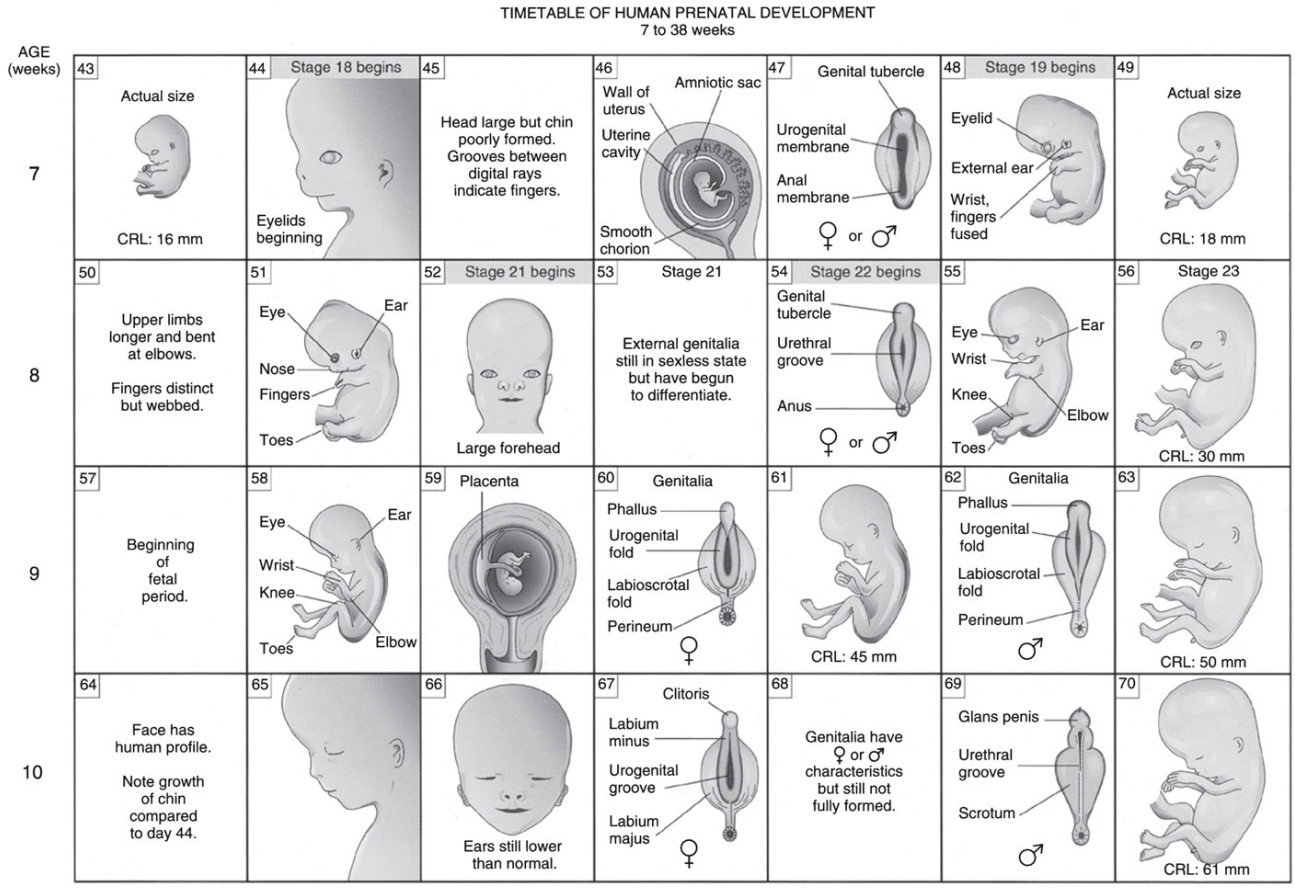


Figure 19-6 Development of an ovarian follicle containing the oocyte, ovulation, and phases of the menstrual cycle are illustrated. Human development begins at fertilization, approximately 14 days after the onset of the last menstruation. Cleavage of the zygote in the uterine tube, implantation of the blastocyst, and early development of the embryo are shown.

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The *cytotrophoblast* tissue is the point of contact between fetal and maternal tissues. The cytoblast differentiates into extravillous trophoblasts, and these trophoblasts then form columns of cells that become the anchoring villi of the placenta (**Figure 19-7**).[17,25] Proliferative extravillous cytotrophoblast cells also invade the spiral arteries, that developed in the luteal phase to provide blood to the endometrium. When the cytotrophoblastic cells reach the spiral arteries, the cytotrophoblastic cells replace the endothelial layer of the spiral artery and begin the process of remodeling those arteries into the low-pressure, high-volume deinnervated open vessels needed to sustain the developing fetus.[28-30]

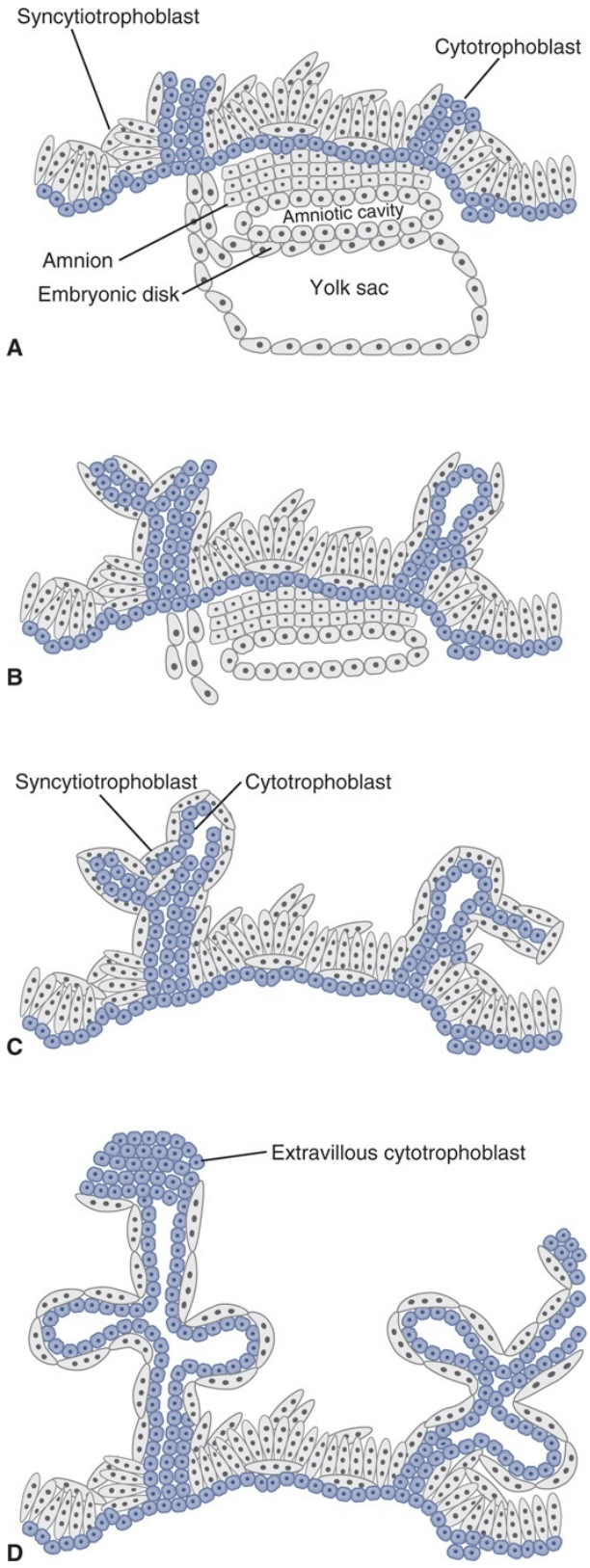


Figure 19-7 **A.** Cytotrophoblast columns in early implanted embryo. **B.** Extension of columns and differentiation of peripheral cells. **C.** Folding of extensions caused by shape of syncytiotrophoblast cells. **D.** Formation of trophoblastic villi.

Reproduced with permission from Cole LA. hCG, the wonder of today's science. *Reprod Biol Endocrinol*. 2012;10:24-28.[17] Published by BioMed Central.

This cytotrophoblastic invasion of the spiral arteries occurs in two waves. The first wave takes place in the first weeks following implantation, as cytotrophoblast cells move down the endothelial lining of the spiral arteries in the decidua. The second wave occurs between 12 and 20 weeks of gestation.[25] During this wave, the cytotrophoblast extends into the myometrial portion of the spiral arteries (**Figure 19-8**).[28-31] The changed spiral artery architecture accommodates a remarkable change in uterine blood flow. Prior to pregnancy, uterine blood flow is approximately 50 mL per minute; by comparison, at the end of pregnancy, uterine blood flow is approximately 750 mL per minute.

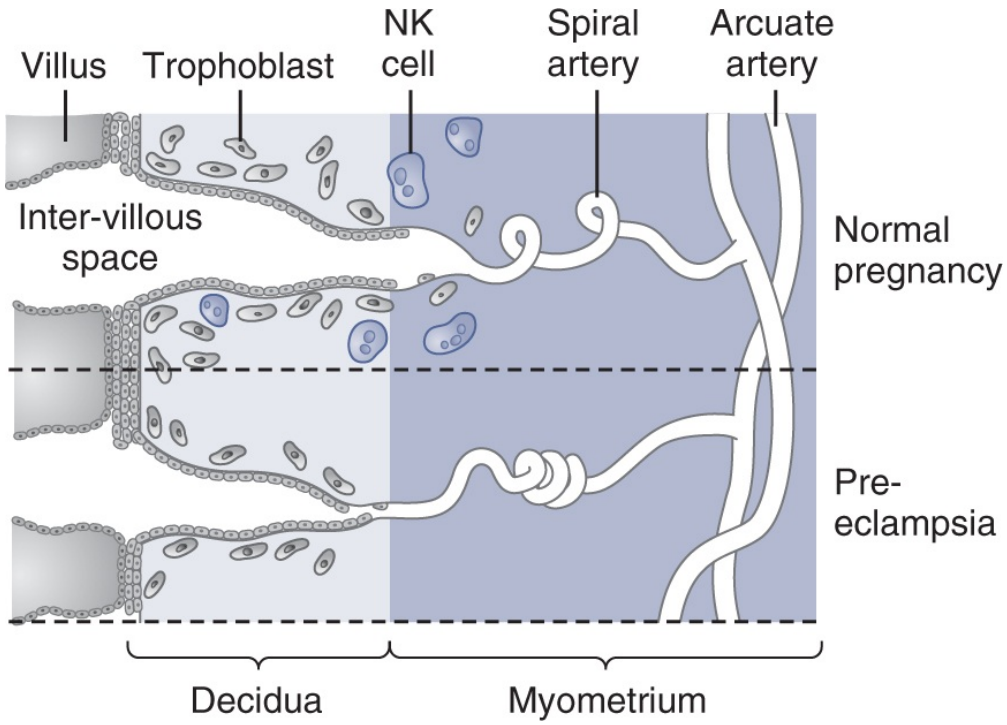


Figure 19-8 Spiral artery invasion in normal and preeclamptic pregnancies.

Based on Jain A. Endothelin-1: a key pathological factor in pre-eclampsia. *Reprod BioMed Online.* 2012;25:443-449.[31]

Complications of pregnancy can occur if cytotrophoblast cells fail to fully invade or deinnervate the spiral arteries.[29] The genesis of preeclampsia is cytotrophoblast invasion, wherein the second wave does not occur and the maternal spiral arteries remain small, high-resistance vessels. These vessels culminate in a small placenta secondary to limited maternal blood flow. The effects of preeclampsia become evident when the nutritional needs of the fetus are no longer met by the small placenta.[31] When the trophoblastic tissue invades indiscriminately, without the normal complex immunologic checks and balances that occur between the maternal and trophoblastic tissues, placenta accreta can develop. Abnormal placentation can also cause fetal growth restriction and has more recently been associated with preterm premature rupture of membranes and preterm labor.[31]

The Placenta

The placenta has been recognized as an important organ for centuries and has been imbued with many different meanings. In some cultures, the placenta is considered the alter ego or "secondary self."[32] In early Egypt, the placenta was described as the "external soul." In other cultures, it is referred to as "the tree of life," because the vessels that branch out from the insertion point on the maternal surface of the placenta look like branches of a tree.

At term, the placenta weighs approximately 500 grams, is 18 to 22 centimeters in diameter, and is approximately 2 to 2.5 centimeters thick.[33,34] This organ has a villous surface area of approximately 1.8 m^2 at term.[33] Clinical implications of common variations in placental gross anatomy and systematic evaluation of the placenta following birth are reviewed in the *Third Stage of Labor* chapter.

The placenta is a vital endocrine, hemochorial, villous organ with four well-known functions:

- Produces hormones critical for maintenance of pregnancy, and other bioactive substances.[25]
- Transports substances between the maternal and fetal circulations, including acting as the respiratory organ for gas exchange.
- Metabolizes and synthesizes agents necessary for sustaining pregnancy.
- Provides an immunologic barrier between the maternal and fetal systems.

Many of these functions overlap (**Figure 19-9**).

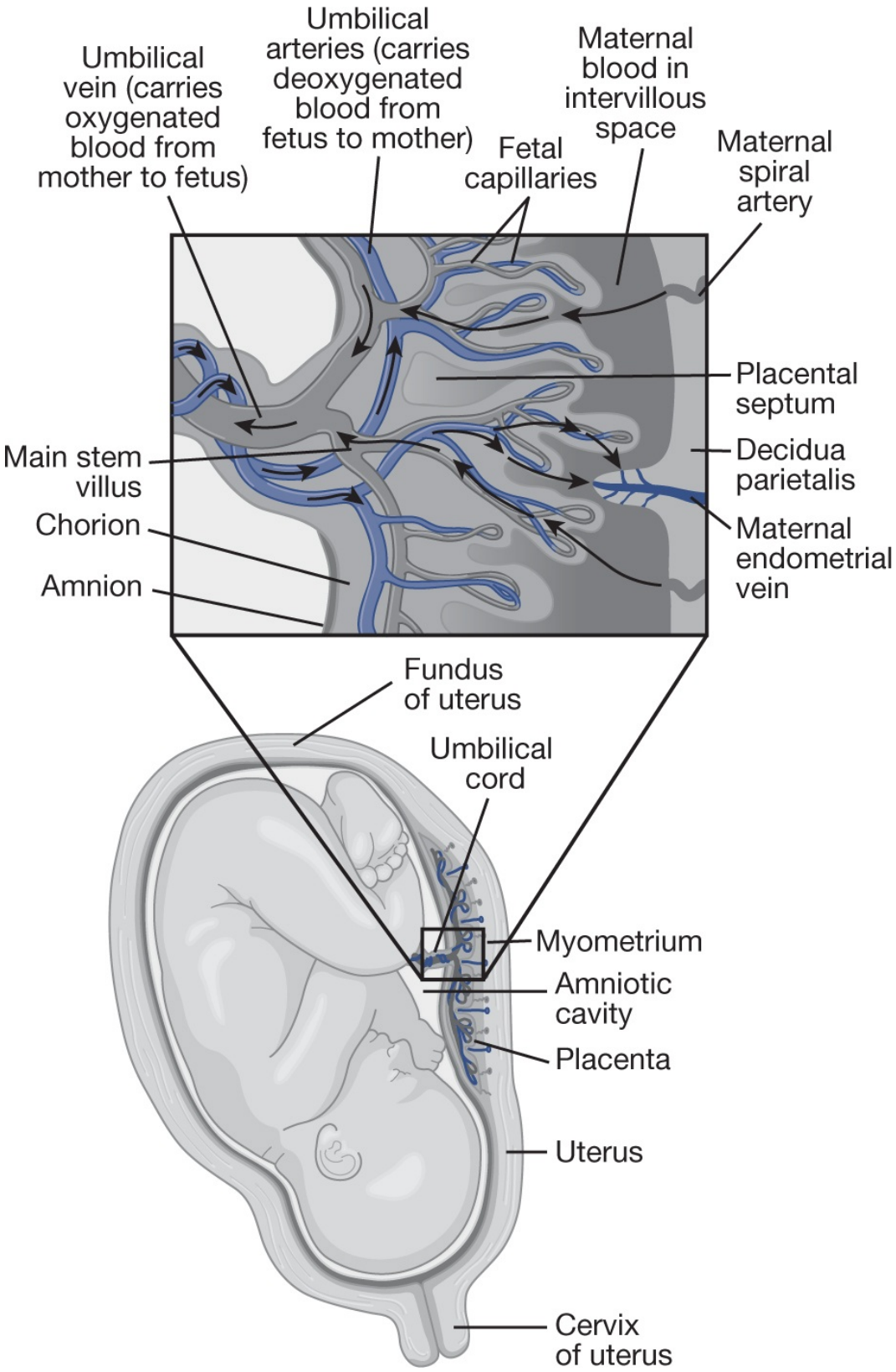


Figure 19-9 Transverse view of a placenta at a full-term pregnancy. Maternal blood flows into the intervillous spaces in spurts from the spiral arteries, and exchange of nutrients and gases occurs with the fetal blood as the maternal blood flows around the branch villi. The two umbilical arteries carry poorly oxygenated fetal blood to the placenta; the one umbilical vein carries oxygenated blood to the fetus. The placental cotyledons are separated from each other by septa projections of the decidua basalis. Each cotyledon consists of two or more main stem villi and branches.

Recent research regarding placental function suggests that the placenta may also play a more complex role during pregnancy. Instead of being just a passive filter or barrier, which has historically been the perceived role of the placenta, this amazing organ integrates a wide range of chemical messages produced by the fetal and maternal systems and actively adapts to different conditions.[25,35] In short, the placenta plays an active and independent role in fetal growth and development.

Development of the Placenta

The cytotrophoblast lines the decidua and firmly anchors the placenta. The extraembryonic mesoderm—another tissue from the original blastocyst—grows into the cytotrophoblastic columns to form villi that float freely in the intervillous space. The fetal vessels that form within the villi are separated from maternal blood by three layers of cells: (1) fetal endothelial cells, (2) connective tissue, and (3) cytotrophoblastic cells that are eventually called the chorionic villi epithelium.

Blood flow begins in the placenta when the intervillous spaces become filled with maternal blood. Initially, the preponderance of fluid in the intravillous space consists of maternal serum and secretions from the endometrial glands, until fetal vessels begin to function at 8 to 9 weeks' gestation. Common variations in placental morphology that have clinical implications are reviewed in the *Third Stage of Labor* chapter.

Placental Transport Mechanisms

Placental function involves multiple transport mechanisms, including facilitated diffusion, passive diffusion, active transport, pinocytosis, endocytosis, bulk flow, solvent drag, accidental capillary breaks, and independent movement.[25,35] These mechanisms are described in **Table 19-2**.

Table 19-2 Placental Transport Mechanisms

Transport Mechanism	Description of Mechanism	Examples of Substances Transported via This Mechanism
Simple diffusion	Transfer of substances across a membrane down a concentration gradient (from an area of higher concentration of the substance to an area of lower concentration of the substance)	Oxygen, carbon dioxide, electrolytes, water, certain medications including analgesic and anesthetic agents
Facilitated diffusion	Transfer of substances across a membrane down a concentration gradient (such as diffusion), but in a manner that allows for more rapid or more specific transfer	Substances such as glucose that are essential for rapid fetal growth but are present in low concentrations in the maternal blood
Active transport	Transport against a concentration gradient, which requires energy	Transfer to the fetus of substances that are present in higher concentrations in the fetus than in the pregnant woman, such as iron and ascorbic acid
Pinocytosis	A form of endocytosis that allows small particles to be brought into a cell. The cell membrane folds around the	Immunoglobulin G (IgG) maternal antibodies, phospholipids used to make cell membranes,

	particle and becomes an intracellular membrane.	lipoproteins used to transport cholesterol
Breaks between cells	Active capillary breaks allow fetal and maternal cells to mix	Cell trafficking may play a role in fetal immunity and in maintaining maternal tolerance to the fetal allograft
Bulk flow	Movement of water and some solutes in water via aqueous pores	Free movement of water and some solutes maintains equal osmolality in the fetal and maternal compartments

Uteroplacental Circulation and Fetal Respiration

The uteroplacental circulation is the lifeline for the fetus. As such, physiology affecting fetal respiration deserves detailed attention. Fetal respiration depends on four factors: (1) adequate maternal blood flow into the intervillous space; (2) sufficient functional placental villi for gas exchange; (3) adequate diffusion, facilitated diffusion, and active transport of gases, nutrients, and fetal waste products; and (4) unimpaired fetal circulation through the placenta and umbilical cord.

The fetus can use either oxygen or glucose to produce the energy needed for growth and metabolic processes. Carbon dioxide (CO_2) and water (H_2O) are the end products of using oxygen for aerobic metabolism. When the fetus does not have sufficient oxygen, glucose is used to create adenosine triphosphate (ATP) for energy. This process is called anaerobic metabolism, and one of its end products is lactic acid ($C_3H_6O_3$), which has important clinical implications that are reviewed in more detail in the *Fetal Assessment During Labor* chapter.

Immunology of the Placenta

The fetus is considered a semi-allogenic graft, meaning that it consists of foreign tissue from the same species but has different antigens. The processes that ensure the woman's body does not reject the fetus have not been yet fully identified. Most importantly, the trophoblast does not express the cell surface antigens that are the usual targets for maternal antibodies. Additionally, the hormones of pregnancy alter the maternal immune response in ways that enable tolerance to contact with the cytotrophoblast and syncytiotrophoblast.

There appears to be more cellular traffic between the fetus and the pregnant woman than was originally hypothesized.[36] Fetal cells can be found in many maternal tissues and can persist in a mother for decades. Similarly, maternal cells enter the fetal circulation via mechanisms that have not been fully elucidated and may support the development of fetal immunity. The implications of this microchimerism are unknown.[36]

Role of the Placenta in Fetal Development and Programming

The placenta synthesizes many substances, including glycogen, cholesterol, and fatty acids, which contribute nutrients and energy for the embryo and growing fetus. Placental metabolism uses approximately 40% to 60% of the oxygen and glucose that is delivered into the intervillous spaces.[35] The placenta can also alter its metabolism in the presence of stressors

such as hypoxia.[35] More importantly, the placenta is instrumental in making heritable changes in gene expression (e.g., epigenetic changes) of the fetal genome that affect postnatal life.[37,38] This developmental plasticity in placental function is an area of current research interest.

Fetal Origins of Adult Disease/Fetal Programming

In the 1990s, physician and researcher David Barker published a theory that was later known by various names. Initially, it was best known as "Barker's hypothesis." Today, alternative nomenclature—such as "developmental origins of health and disease" (DOHaD), "fetal programming," and "the thrifty genotype"—is more commonly used because research has established that Barker's work is no longer a hypothesis. Barker's major finding was that fetuses who were undernourished in utero were at increased risk for chronic diseases such as obesity, diabetes, coronary heart disease, and hypertension when they grew to adulthood in a country with abundant food.[38] His work linked birth records with later health and death records in the United Kingdom. Approximately a decade after his initial publication, Barker described his basic methodology and acknowledged his debt to the meticulous and complete records of midwives, without whom he would not have had adequate data to discover the associations.[39]

Subsequent to Barker's initial work, many links have been discovered between the fetal environment and several major chronic diseases that arise during adulthood.[40-47] When a pregnant woman experiences poor nutrition, such as during war or related to severe poverty, her placenta will lower production of enzymes that protect the fetus from cortisol. More exposure to cortisol in utero enables the fetus to survive low protein intake during gestation, but increases the risk of cardiovascular disorders when that fetus becomes an adult.[43] Specific physiologic changes related to these processes are under investigation and much remains to be learned.

Current research suggests many types of prenatal stresses—not just nutrition—can permanently affect fetal physiology and infant development, especially in the areas of vascular, metabolic, and endocrine changes. Prenatal stressors may include stress hormones, environmental toxins, and even paternal genes. Currently, DOHaD is the subject of a great deal of research, including that which focuses on the evaluation of epigenetic mechanisms and the investigation of maternal, paternal, and fetal genes.

Barker's work is of importance to midwifery because it underscores the importance and complexity of preconceptional and prenatal care. In an era when prenatal visits are becoming fewer in number and shorter in duration, it is the midwife's responsibility to promote health care both for the current generation of women and for the future of society's health.

Role of the Placenta in Pregnancy-Related Complications

In the short-term, the placenta plays a central role in many pregnancy-related complications. As described, the origins of preeclampsia occur early in pregnancy although signs and symptoms develop much later. Poor maternal nutrition can result in small, poorly functioning placentas that provide inadequate nutrients to the fetus, resulting in fetal growth restriction.

Women with diabetes and hyperglycemia may experience microvascular and macrovascular placental changes, such as thickening of the basement membrane of cells, and these placentas also may not sustain normal fetal growth.

A more complex example is illustrated in the relationship between obesity and poor maternal and newborn outcomes. The placentas of women who are obese express placental genes differently than the placentas of women who are lean, and they tend to show metabolic and oxidative stress leading to cell injury and inflammation of the placenta.[48] The low-level inflammation damages the endothelial cells that line blood vessels, which in turn, adversely affects delivery of oxygen and nutrients to the fetus.

Amniotic Fluid

Amniotic fluid serves multiple functions during pregnancy, including cushioning and protecting the fetus, providing space for fetal movement and growth, and maintaining consistent temperature and pressure.[49] Substances found in amniotic fluid include electrolytes, urea, creatinine, bile pigments, renin, glucose, hormones, fetal cells, lanugo, and vernix caseosa. The fluid's osmolality and composition evolve throughout the course of the pregnancy and are similar to the characteristics of dilute fetal urine in a term gestation.

In the latter half of pregnancy, amniotic fluid is primarily produced by the fetus in the form of urine (700–800 mL) and lung fluid. The average amount of amniotic fluid at term is 700–800 mL.[50] Amniotic fluid is removed via fetal swallowing and diffusion across the placenta. The fluid secretions from the fetus's lungs contain phospholipids, including lecithin and sphingomyelin, which are components of surfactant—a substance that is essential for the function of the neonatal lungs. As pregnancy advances, the absolute and relative amounts of lecithin in amniotic fluid increase. The ratio of lecithin to sphingomyelin is used as means of assessing fetal lung maturity to help guide decisions about delivery prior to term. While the amount of fluid varies in the third trimester and begins to decrease after 40 gestational weeks, the amount of fluid that is turned over (produced and removed) remains relatively constant, at approximately 1000 mL per day.

Amniotic fluid is essential for normal fetal growth. *Oligohydramnios* at term is defined as an amniotic fluid index of 5 centimeters or less (via ultrasound assessment). Oligohydramnios in the second trimester can inhibit normal fetal lung development. Etiologies of this disorder include fetal renal abnormalities, heart disease, and fetal growth restriction. Maternal conditions that can cause oligohydramnios include severe hypertension, dehydration, and renal disorders.

Polyhydramnios is defined as an amniotic fluid index of more than 24 centimeters and is caused by too much production or too little removal of amniotic fluid. Modest polyhydramnios can be idiopathic, whereas oligohydramnios is most likely to be associated with an abnormal condition in the fetus or the pregnant woman. Conditions that can cause polyhydramnios include maternal hyperglycemia, obstructed fetal swallowing, fetal cardiac failure, and severe fetal anemia.

The Umbilical Cord

The umbilical cord has two arteries and one vein surrounded by a gelatinous collagen material called *Wharton jelly*. The vessels within the cord are longer than the cord itself, so they coil in a spiral fashion as the cord lengthens. This coiling may protect the blood flow within the cord if it is subjected to tension or compression. The maximum length of the cord averages 55 to 60 centimeters. The cord epithelium, which is formed by the amnion, typically inserts into the placenta centrally but may insert at many portions of the placenta. The only clinically relevant aspect of cord insertion occurs when the cord inserts marginally (at the placental edge) or when the insertion is velamentous, wherein the cord inserts into the membranes. With velamentous cord insertion, fetal vessels run through the membrane unprotected by Wharton's jelly. When umbilical cord vessels run through the membranes across the cervix, the presentation is termed vasa previa. Although vasa previa is very rare, fetal mortality occurs approximately 50% of the time when the vessels rupture.[51]

The Embryo

Organogenesis

Returning to the blastocyst, the *embryonic period*, which is the period of organogenesis, occurs between 2 and 8 weeks following fertilization. The *fetal period*, marked by growth and tissue differentiation, starts at 8 weeks after fertilization and extends to birth. The age of the embryo in "gestational weeks" refers to the number of weeks after fertilization in the embryology and basic science literature. Clinical obstetric texts use the term "gestational weeks" to define the number of weeks after the woman's last menstrual period. This 2-week discrepancy can be confusing unless the reader understands which terminology the author is using. This chapter follows the convention used by embryologists for this section of this chapter.

The embryonic disc within the blastocyst gives rise to three germ layers: (1) the endoderm, (2) the mesoderm, and (3) the ectoderm. The third week of embryo development is marked by a period of rapid growth, during which the mesoderm, ectoderm, and endoderm begin to undergo the dramatic transformations that form specific embryonic structures (**Table 19-3**). Most functional organs and organ systems are formed from all three embryonic germ layers. Each germ layer contributes a specific feature, but the germ layers do not produce specific structures separately from each other.

Table 19-3 Differentiation of Embryonic Germ Layers

Ectoderm	Mesoderm	Endoderm
Central and peripheral nervous system	Connective tissues	Epithelium of the digestive system (except the mouth and anus, which are involutions of the ectoderm)
Epidermis including hair, nails, and sebaceous glands	Muscle tissue	Liver and pancreas
Epithelium of sensory organs	Skeleton (bone)	Respiratory system, including alveolar cells of the lung
Nasal and oral cavities	Cardiovascular	Thymus, thyroid, parathyroid, and pancreas
Salivary glands	Lymphatics	
Adrenal medulla	Urogenital structures (gonads and kidney)	
Parts of the pituitary gland	Serous lining of body cavities (peritoneum, pleural, and pericardium)	

Morphogenesis

The genetically controlled process during which cells and cell groups take on a specific form, shape, and function is known as *morphogenesis*. Initially, the cells in the embryoblast are all the same; that is, they are stem cells, capable of becoming any type of body cell. These unspecialized cells must proceed through two distinct phases: (1) determination, which restricts the cell to a specific type, and (2) differentiation, in which the morphologic and functional characteristics specific to that cell type develop.

Cell differentiation often involves a process called induction wherein cells in a local environment signal one another to develop in a specific manner. The signaling cell is called the inductor, and the cells that respond to induction are called the inducers. If disruption occurs during any of the differentiation sequences, the next step in the process will not proceed in the usual fashion and an abnormality will develop. Complete organ agenesis may occur when the process is disturbed at an early stage. For example, anencephaly is a form of organ agenesis wherein the brain does not form. Some disruptions in differentiation sequences will result in termination of the pregnancy, whereas others may produce a fetal defect that can be undetectable, minor, or clinically significant.

In addition to cell differentiation and induction, several other cellular mechanics are involved in morphogenesis, including proliferation, migration, adhesion, and folding. Some of these processes are summarized in **Table 19-4**.

Table 19-4 Selected Cellular Mechanisms Involved in Morphogenesis

Cell Mechanism	Description	Congenital Defects That May Result from Faulty Mechanism
Cell proliferation (hypertrophy)	A rapid increase in the number of cells by cell division and growth	Inhibition of cell proliferation can occur when there is a lack of space. A diaphragmatic hernia will allow abdominal contents to be in the thoracic cavity; pulmonary hypoplasia occurs when the lungs are not allowed to continue cell proliferation.
Cell differentiation	Process by which pluripotent cells become more specialized cells	Prenatal exposure to high levels of methylmercury is thought to result in failure of cell differentiation in the central nervous system, causing neurologic and developmental defects.
Apoptosis	A genetically determined process of cell self-destruction. Cells produce enzymes that lead to their dissolution.	Excessive enzymatic release can lead to excessive cellular destruction and result in defects such as limb shortening; deficient enzymatic release can lead to defects such as bowel atresia, syndactyly, or imperforate anus.
Migration	A dynamic and cyclical process in which layers migrate to a strategic location along the developing embryo. The cell extends protrusions at its front and attaches to the substratum on which the cell is migrating.	Central nervous system abnormalities, such as lissencephaly (lack of grooves on the brain)
Adhesion	The interaction of specific mechanisms on one cell and complementary adhesion molecules on the membrane of another cell	Cleft palate and neural tube defects occur as a result of alteration in cell recognition and the adhesion process.
Folding	As new cells form, the embryo is forced to conform to the available space. The embryo folds in both the transverse and longitudinal planes. Structures within the embryo also must fold to conform to the space available to them.	Congenital heart defects Diverticula

The Fetus

Fetal Growth

All major organs have formed by the beginning of the fetal period. Fetal growth involves both hyperplasia (cellular division that yields a significant increase in cell numbers) and hypertrophy (an increase in cell size). At approximately 32 weeks' gestation, hypertrophy dominates.[52] The rate and amount of fetal growth are determined by many factors, including genetics, placental metabolism, maternal conditions, maternal behavior, and environmental factors. For example, there is a well-known correlation between maternal smoking and impaired fetal growth.[53]

Adequate fetal growth is also directly associated with optimal functioning of the placenta and the uterine vascular system. Alterations in the trophoblastic invasion of the maternal spiral arterioles as well as inadequate development of the chorionic villus can result in impaired fetal growth.

Fetal Movement and Behavioral States

Limb movement develops at 9 weeks, with reflective leg movements occurring at 14 weeks. Hand-to-face movements become apparent by 12 to 13 weeks, while limb, head, and torso movements develop by 12 to 16 weeks. Fetuses begin to suck on fingers by 15 weeks and continue to develop more complex movement patterns after 24 weeks, when respiratory movements occur.[54]

Discrete fetal behavior states involving sleep–wake patterns and behavioral patterns begin to occur by 32 weeks. Distinct fetal heart rate patterns, eye movement, gross body movement, and quiet states have been catalogued.[55,56] Although women often report less fetal movement close to term, the underlying fetal physiology of this maternal perception is likely the development of fetal cycling, in which the fetus exhibits distinct quiet states and distinct active states. The average time the fetus spends in the quiet state ranges from 20 to 40 minutes.[54,55]

Maternal Adaptations to Pregnancy

Pregnant women experience a wide variety of anatomic and physiologic alterations over the course of pregnancy and postpartum. While many of these changes are normal symptoms of pregnancy, others herald the onset of an abnormal processes. A thorough knowledge of the maternal anatomic and physiologic adaptations that occur during pregnancy is, therefore, an essential foundation for midwifery practice. This section presents a broad overview of some of the clinically important anatomic and physiologic pregnancy adaptations. Management of pregnancy symptoms are described in the *Prenatal Care*, *Pregnancy-Related Conditions*, and *Medical Complications in Pregnancy* chapters.

Musculoskeletal Changes

The musculoskeletal changes that occur during pregnancy are primarily related to weight gain, the growing uterus, the softening effects of progesterone on cartilage in joints, and the laxity of ligaments induced by estrogen and relaxin.[56] These changes result in lordosis, kyphosis, and altered gait that gradually increase as pregnancy progresses.

Changes within the pelvic girdle are the most profound. The sacroiliac joint widens and has more mobility. The symphysis also widens, and the pelvis develops an anterior tilt. Many of the common complaints of pregnancy can be attributed to these anatomic changes, including pelvic pain, sciatica back pain, and carpal tunnel syndrome. A careful history and physical examination is needed to rule out more serious problems for which pregnant women are at increased risk, such as herniated discs and peripheral nerve injury.

Integumentary Changes

Pregnancy is associated with many changes in the integument. Hyperpigmentation occurs as estrogen, progesterone, and melanocyte-stimulating hormone induce melanocytes to make and deposit pigment. This phenomenon results in darkening of the areola, the change of the linea alba to the *linea nigra*, and *melasma* or *chloasma* (irregular areas of pigmentation on the cheeks), which is also called the “mask of pregnancy.”[57-59] Intertriginous areas such as the axillae, genitalia, perianal region, and inner thighs may also become darker during pregnancy.[59]

Thinning of the elastin fibers in connective tissue under the skin predisposes pregnant women to striae gravidarum (stretch marks). As the size of the abdomen and breasts increase, elastin fibers at the dermal–epidermal junction stretch and shift from perpendicular to parallel, which can create striae.

Vascular nevi called spider angiomas are common as blood vessels dilate and proliferate. These small red lesions have a central puncta and branches that extend from the center and disappear after birth.

Cardiovascular Changes

Cardiovascular changes in the pregnant woman begin early in the first trimester.[57] Blood volume increases by 40% to 50% over the course of pregnancy, reaching a maximum by 32 weeks' gestation. Plasma accounts for 75% of this increase. Cardiac output increases 30% to 50% to approximately 4 to 6 L/min, primarily as a result of increased stroke volume.[60] The increase in cardiac output begins in the first trimester and peaks at approximately 25 to 30 weeks' gestation, when the total blood volume is approximately 5000 to 6000 mL. Heart rate increases by approximately 10 beats/min, and blood pressure decreases gradually from prepregnancy values as early as 7 weeks' gestation. This decrease reaches a nadir in the second trimester. The decrease in blood pressure is presumed to occur in concert with expansion of the low-pressure placental compartment, and lower systemic vascular resistance induced by progesterone.[60]

Anatomically, the heart is displaced upward (cephalad) and rotated to the left as the uterus enlarges. Mild pulmonic or tricuspid regurgitation often occurs. Several common signs and symptoms are related to these changes. Systolic ejection murmur that is loudest along the left sternal boarder is a common finding in pregnant women and is attributed to the dramatic increase in cardiac output. These murmurs are clinically benign. A third heart sound may be auscultated in many pregnant women. The decrease in systemic vascular resistance in combination with pressure on the vena cava from the growing uterus is responsible for the dependent edema that most pregnant women experience in the third trimester and contributes to the development of varicosities, hemorrhoids, labial varicosities, and increased risk for venous thrombosis.

Careful monitoring of cardiovascular changes can facilitate early detection of abnormalities. For example, if a woman's blood pressure fails to decrease during the second trimester, she may have chronic hypertension or be at increased risk for developing preeclampsia. Cardiovascular changes can also increase the risk for adverse outcomes in women who have preexisting cardiomyopathies; these women should be referred promptly to a physician.[58]

Hematologic Changes

Two aspects of hematologic changes in pregnancy have important clinical implications. First, pregnancy is a hypercoagulable state, as evidenced by the increased clotting factors, decreased fibrinolysis, and decreased anticoagulant activity noted in the pregnant woman. Clotting factors I, II, VII, VIII, IX, and XII are more abundant in such women, whereas protein S levels and activated protein C levels fall. These alterations in the coagulation cascade are likely protective, geared toward preventing hemorrhage at birth. Nevertheless, they increase a woman's risk for venous thromboembolism in the prenatal and postnatal periods. In addition, significant placental damage and obstetric hemorrhage are associated with early onset of disseminated intravascular coagulation (DIC), which is not a common component of surgical or traumatic hemorrhage. DIC occurs because the tissue factor in the placenta acts as a potent activator of the coagulation cascade, which is already in a hypercoagulable state.[58]

The second clinically important hematologic change in pregnancy relates to iron metabolism and iron-deficiency anemia. During pregnancy, the plasma volume increases and this expansion exceeds the increase in red cell mass, which results in a physiologic hemodilution. The

physiologic hemodilution has a positive effect on placental perfusion, with blood becoming less viscous.[61] In addition, the maternal hemoglobin (Hgb) concentration decreases by approximately 2% to 10%. The drop in hemoglobin occurs because the fetal uptake of iron is usually more than maternal absorption of iron can replace. The net result is a decrease in the woman's hematocrit of approximately 3% to 5%, which reaches a nadir late in the second trimester or early in the third trimester.

Because iron is not easily absorbed, fetal uptake can deplete a woman's iron reserves despite the fact that iron absorption in the second and third trimesters increases more than fivefold.[62] Thus iron stores can be easily depleted in pregnancy and, in turn, iron-deficiency anemia is common in pregnancy.

Additional hematologic changes include a decrease in the concentration of plasma proteins, especially albumin. Lower albumin result in lower colloid oncotic pressure, which in tandem with decreased venous resistance, facilitates the development of dependent edema.

Respiratory Changes

Pregnant women commonly experience dyspnea even when at rest. The actual etiology is unknown, but most sources assume this condition arises secondary to the added respiratory effort and work of breathing.[63] Pregnancy is associated with an increase in minute ventilation (i.e., the volume of air inhaled and exhaled in a minute) and in pulmonary blood volume, both of which can contribute to the sensation of dyspnea. The etiology of these changes is largely unknown, but it is thought to relate to an enhanced sensitivity to carbon dioxide levels as well as to hypoxia. As the uterus expands into the abdominal cavity in the second and third trimesters, the respiratory effort required by the woman further contributes to dyspnea. Physiologic dyspnea can be distinguished from pathologic dyspnea by the respiratory rate. Tachypnea is a sign of possible respiratory compromise.

An increase in thoracic diameter and cephalad rise in the diaphragm (as much as 4 centimeters) changes the pregnant woman's lung capacity. Tidal volume increases by 30% to 40%, and vital capacity increases slightly (**Figure 19-10**).[63,64] This hyperventilation probably occurs secondary to the effects of progesterone and places the woman in a state of respiratory alkalosis, which has the effect of improving carbon dioxide transfer from the fetus to the maternal circulation.

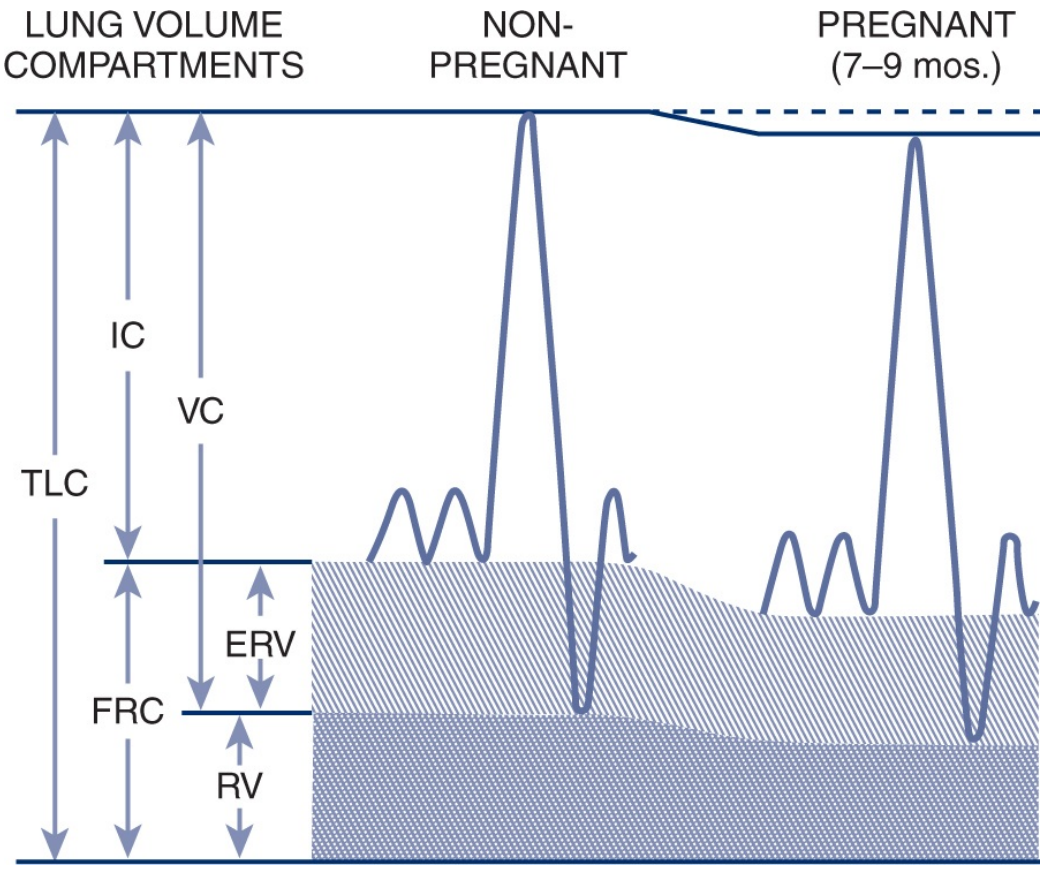


Figure 19-10 Changes in lung volumes in women who are 7–9 months pregnant compared with volumes in nonpregnant women.

Abbreviations: ERV, expiratory reserve volume; FRC, functional residual capacity; IC, inspiratory capacity; RV, residual volume; TLC, total lung capacity; VC, vital capacity.

Under the influence of estrogen and increased blood volume, nasal passages become edematous and hyperemic in pregnancy. Women often report more congestion and/or rhinitis, sometimes confusing the symptoms with an upper respiratory infection or allergies.

Changes in the Oral Cavity

During pregnancy, bleeding gums, especially after brushing teeth, is related to estrogen- and progesterone-mediated inflammation and hyperemia. Elevated levels of estrogen create a favorable environment for the growth of bacteria that can cause gingivitis and gingival inflammation. Women should receive regular dental care during pregnancy.

Gastrointestinal Changes

During pregnancy, decreased peristalsis caused by relaxation of the smooth muscle of the large bowel in the presence of increased amounts of progesterone, as well as changes in fluid reabsorption increases the risk of constipation. The displacement and compression of the bowel by the enlarging uterus or presenting part may also contribute to decreased motility in the gastrointestinal tract and, therefore, to constipation. Specifically, the stomach is moved superiorly and the intestines are displaced laterally. Another factor that may contribute to

constipation is the decrease in gastric emptying time and gastric motility secondary to the smooth muscle relaxation effects of progesterone. Thus, constipation is common, especially in the first trimester when the growing uterus also places pressure on the descending colon.

Several physiologic changes of pregnancy predispose the pregnant woman to heartburn (pyrosis): The lower esophageal sphincter is relaxed, gastrointestinal motility is slower, esophageal function and peristalsis change, and the angle of the gastroesophageal junction is altered as the stomach is displaced by the enlarging uterus. Women with preexisting gastroesophageal reflux disorder may find the condition aggravated throughout pregnancy. Flatulence and gas pain is more common during pregnancy due to decreased motility from the effect of progesterone relaxing smooth muscle and from the displacement of and pressure on the intestines by the enlarging uterus.

Renal Changes

Two different aspects of renal changes in pregnancy are responsible for symptoms that must be carefully evaluated to determine normal versus abnormal alterations. First, a marked increase in renal plasma flow is the natural consequence of arterial vasodilation and increased cardiac output. Renal blood flow increases 60% to 80% above prepregnant levels in the first and second trimesters, and 50% above prepregnant levels in the third trimester. The glomerular filtration rate (GFR) increases by 50% over prepregnant levels, peaking at 12 gestational weeks.[65] The obvious physiologic consequences of these changes are the commonly reported symptoms of urinary frequency and nocturia that frequently occur at two different times during the prenatal period. Frequency during the first trimester is due to hormonal changes affecting levels of renal function as well as developing hypervolemia and bladder compression within the pelvis due to uterine growth. Bladder compression resolves as the uterus becomes an abdominal organ in the second trimester. Urinary frequency during the third trimester occurs most often among primiparous women, after engagement has occurred when the presenting part descends into the pelvis and causes direct pressure against the bladder.

Nocturia also can be caused by increased urine production at night. Venous return from the extremities is facilitated when the woman lies in a recumbent lateral position while sleeping at night, when the uterus is not pressing against the pelvic vessels and inferior vena cava, which results in an increase in urinary output. Additionally, pregnant women have an increase in sodium excretion at night, with an associated increase in fluid excretion, which may also explain nocturia.

The increased flow may be great enough that the descending tubule is unable to reabsorb all glucose. This resultant physiologic glycosuria is usually intermittent, but it affects as many as 20% of pregnant women. Similarly, protein reabsorption is not as efficient as it is in the nonpregnant state. A small amount of urinary protein in a sample of concentrated urine can cause a dipstick test to be positive, which may be falsely interpreted as a urinary tract infection or even proteinuria associated with preeclampsia. Serum creatinine likewise falls; thus, plasma values for creatinine that would be considered normal in a nonpregnant woman may actually reflect renal dysfunction in a pregnant woman.

Pregnant women are at increased risk for urinary tract infections because the ureters,

urethra, and bladder dilate under the influence of progesterone and ascending infections can occur, including pyelonephritis. The bladder becomes hyperemic and urinary stasis can occur, which occasionally results in stress incontinence. Thus, physiologic changes in pregnancy cause symptoms that may be normal, a sign of urinary tract infection, or a sign of renal dysfunction. Urinary tract infections are also associated with preterm labor. Therefore, careful attention to the history, physical examination, and adjunct measures of urinary function are necessary to adequately care for women with urinary symptoms.

Metabolic Changes

Among the many important endocrine and metabolic changes in pregnancy are changes that occur in the hypothalamus, pituitary, and adrenal glands, often in an interrelated manner. Calcium metabolism and the renin–angiotensin system both exhibit significant alterations in ways that facilitate fetal growth and development.

Thyroid Metabolism

Although all endocrine organs undergo changes in pregnancy, thyroid changes are particularly of note. Because both hypothyroid and hyperthyroid states can adversely affect the fetus, it is critical that a euthyroid state be maintained throughout pregnancy.

The thyroid is the first endocrine gland to appear in the fetus, but the fetus does not start secreting thyroid hormone until approximately 18 to 20 weeks' gestation.[66,67] Consequently, the fetus is dependent on maternal thyroid hormone for the critical metabolic functions fulfilled by this hormone. A woman's thyroid slightly increases in size early in pregnancy and may be palpable on an initial prenatal visit as a smooth and regular-shaped mass. The basal metabolic rate also increases by 20% to 25% during pregnancy.

Several changes occur in the production and transport of thyroxine during pregnancy. The alpha unit of hCG stimulates the thyroid in the same manner as TSH, which causes an increase in total thyroxine (T_4) levels (i.e., subclinical hyperthyroidism). Sensing the increase in total thyroxine, the pituitary reduces production of TSH. At the same time, higher levels of plasma albumin and thyroxine-binding globulin (TBG) bind more thyroxine in serum. Overall, the level of free thyroxine remains normal despite lower levels of TSH and higher levels of total thyroxine (**Figure 19-11**).[67,68]

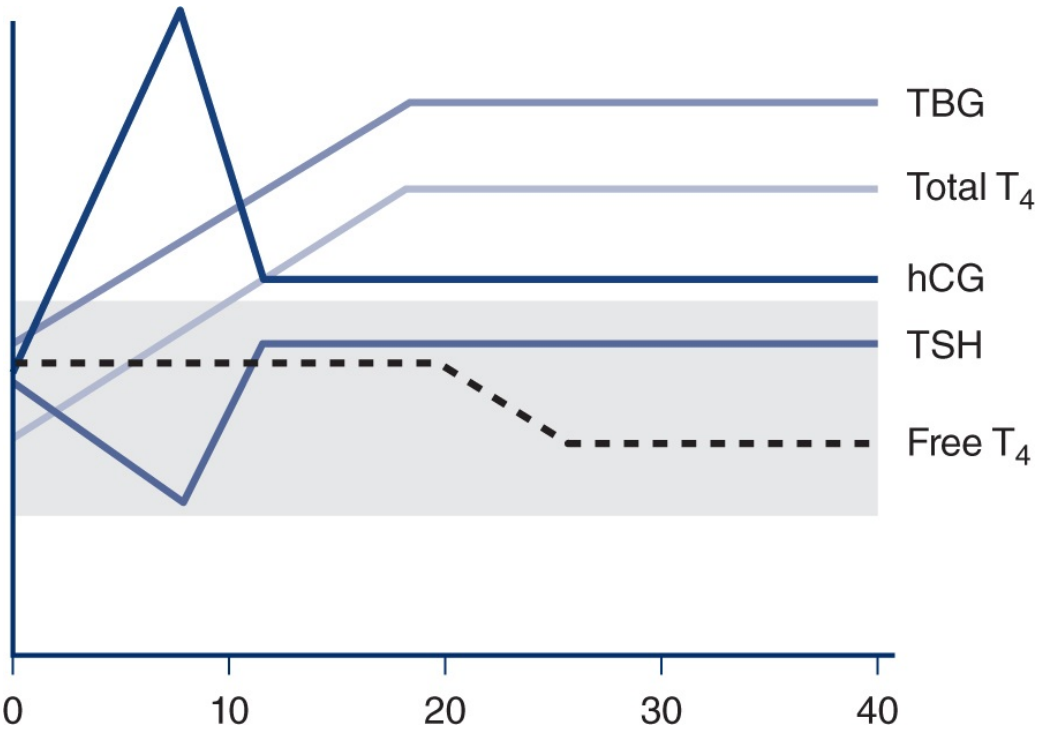


Figure 19-11 The pattern of changes in serum concentrations of thyroid function studies and hCG according to gestational age. The shaded area represents the normal range of thyroid-binding globulin, total thyroxine, thyroid-stimulating hormone, or free T_4 in the nonpregnant woman.

Abbreviations: hCG, human chorionic gonadotropin; T_4, thyroxine; TBG, thyroid-binding globulin; TSH, thyroid-stimulating hormone.

Reproduced with permission from Casey MB, Leveno K. Thyroid disease in pregnancy. *Hil Obstet Gynecol.* 2006;108:1283-1292.[67]

The TSH level reaches a nadir at approximately 10 gestational weeks.[66-68] Subsequently, as hCG levels decline, TSH levels rise to reach the nonpregnant level by the third trimester and total thyroxine levels decline to a normal value. Plasma values for thyroid function are trimester specific and cannot be determined via one measurement. In general, an assessment of TSH and free T_4 or total T_4 is needed to interpret thyroid function during pregnancy.[66-68]

Glucose and Lipid Metabolism

Glucose metabolism is significantly altered in pregnancy. Glucose is the primary energy source for the fetus and placenta, and is transferred across the placenta via facilitated diffusion. Thus, glucose delivery to the fetus depends on a concentration gradient between the maternal and fetal circulations. The hormone hPL induces maternal insulin resistance and hepatic glucose formation, both of which raise glucose levels in maternal circulation. hPL acts by initiating hyperplasia within the beta cells of the maternal pancreas, which in turn enables greater production of insulin. Hepatic glucose production increases by as much as 30% in the third trimester. Because of the ongoing fetal use of glucose provided by the maternal circulation, a pregnant woman will experience fasting glucose levels that are lower than usual, yet because of the insulin resistance, postprandial glucose levels can be higher.[69]

Lower fasting blood glucose levels can worsen nausea and vomiting in early pregnancy but

may also contribute to the pregnant woman's enhanced appetite and her need to eat more often. As the placenta increases in size and function, maternal hyperinsulinemia must likewise increase to keep pace. Women who are unable to sufficiently raise their insulin production will develop gestational diabetes in the latter half of pregnancy.

Lipid metabolism is also altered significantly during pregnancy.[70] Body fat accumulates during the first two-thirds of pregnancy, but this growth then stops or declines during the last third of pregnancy. Thus, the pregnant woman is first in an *anabolic* state and then later in a *catabolic* state.[71] During the period of rapid lipolysis, free fatty acids and glycerol are generated. Free fatty acids are converted to ketones, and glycerol is converted to glucose. The free fatty acids are used to synthesize triglycerides, which causes triglyceride levels to increase.[71]

Immunologic Changes

Pregnancy is essentially an immunologic paradox: First, how does the pregnant woman avoid rejecting the fetus given that fetal cells and maternal cells are in direct contact in the maternal spiral arteries and intervillous space? Second, how do the immunologic changes that take place to accommodate the fetus, a semi-allograft, affect the maternal immune response? Immunologic changes in pregnancy are implicated in several important clinical disorders, including recurrent miscarriage, Rh sensitization, and preeclampsia.

The immune response is usually subclassified as either innate immunity or adaptive immunity; the latter is subdivided into two components called cell-mediated immunity and antibody-mediated immunity. The innate immune response is the first line of defense against "non-self" invaders, which includes inflammation and phagocytosis. *Cell-mediated immunity* is responsible for elimination of intracellular microbes and involves several immune lymphocytes, including natural killer (NK) cells and T cells. *Antibody-mediated immunity* involves the production of antibodies by B cells; these antibodies then target extracellular microbes or antigens.

In general, innate immunity is enhanced during pregnancy, whereas adaptive immunity is less functional. In particular, cell-mediated (T helper 1) responses are somewhat suppressed compared to antibody-mediated (T helper 2) responses, which are more responsive. This change in the cell-mediated and antibody-mediated responses is referred to as a *Th1 to Th2 shift*.[71-73] These changes increase the risk of maternal infection, especially with regard to viral infections such as influenza or varicella. At the same time, the Th1 to Th2 shift results in improvement of some autoimmune disorders such as rheumatoid arthritis.

Chemotaxis is delayed in the pregnant woman, which can delay the maternal response to some infections. The total white blood cell count is elevated largely due to increased numbers of polymorphonuclear neutrophils, monocytes, and granulocytes. Many of the bioactive agents produced by the fetus and placenta effect subtle shifts in maternal immunity. Although little overall change occurs in the maternal immune response, subtle changes in each of the three types of immunity have important clinical implications (**Table 19-5**).

Table 19-5 **Changes in the Immune System During Pregnancy**

Primary Host Defense Mechanisms
Increased number of white blood cells (primarily polymorphonuclear leukocytes), which enhances the pregnant woman's nonspecific immune response.
Delayed chemotaxis (the movement of phagocytes to the site of foreign invasion), which may delay the maternal response to infection.
Decreased number of natural killer cells, which may delay the maternal response to infection.
Reduced levels of plasma IgG. The hemodilution of pregnancy and passive transfer of Ig antibody to the fetus reduces maternal blood levels of IgG.
Cell-Mediated Immunity
Although the overall number of lymphocytes remains unchanged, there is a decreased number of T-helper cells (CD4 cells) relative to the number of T-suppressor cells (CD8 cells).
With fewer CD4 cells, the B-cell function may be slightly impaired.
Antibody-Mediated Immunity
Overall, the antibody-mediated response is unchanged.
Clinical Implications
Small increase in risk for developing gram-negative organism infections and mycotic or fungal infections
Increased morbidity from gram-negative infections, H1N1 flu virus, and varicella if infection occurs
Increased infectivity with certain pathogens, including herpes simplex virus, poliovirus, cytomegalovirus, malaria, and hepatitis
Changes in autoimmune disease characteristics (rheumatoid arthritis often improves during pregnancy, while systemic lupus erythematosus is more likely to flare)

Abbreviation: IgG, immunoglobulin G.

The Fetus as an Allograft

The first part of the pregnancy immunologic story occurs during implantation and is not yet fully understood. In simple terms, trophoblastic tissue does not express the cell membrane proteins that would stimulate an innate or cellular immune response. The mechanisms by which the fetus eludes detection as a foreign object are quite complex.[72] In addition, changes in the cell-mediated immune response result in a concentration of NK cells in the decidua; these cells, however, are a variant of NK cells that have minimal cytotoxic abilities but a refined ability to control trophoblast invasion and remodel uterine vasculature. T cells and B cells become scarce in the uterine environment during this period.

Fetopelvic Relationships

The fetus can lie in numerous positions in relationship to the maternal abdomen pelvis. A few of these positions preclude a vaginal birth; others are associated with a longer labor. For this reason, it is important to know all possible fetopelvic relationships and their clinical significance. The terminology used to describe fetopelvic relationships is listed in **Table 19-6**.

Table 19-6 Fetopelvic Relationships

Term	Definition
Asynclitism	Oblique presentation of the fetal head. When the fetal head is tilted laterally toward the fetal shoulder, the biparietal diameter is not parallel to the planes of the pelvis. The sagittal suture will not be palpable as midway between the front and back of the pelvis. Asynclitism is called anterior when the anterior parietal bone is the point of presentation; it is called posterior when the posterior parietal bone is the presenting part of the fetal head.
Attitude	Relation of fetal parts to each other. The basic attitudes are flexion and extension. The fetal head is flexed when the chin is close to the chest; it is extended when the occiput is closer to the cervical spine.
Cephalic Prominence	Fetal part of head most easily felt (prominent) during Leopold's maneuvers and used to determine attitude. When the cephalic prominence is felt on the same side as fetal small parts, the head is flexed; when on the same side as fetal back, the head is extended. When the cephalic prominence is not palpable on one side or the other, it often is called a military attitude.
Denominator	An arbitrarily chosen point on the presenting part of the fetus that is used to describe fetal position. The denominator for a vertex presentation is the occiput; for a breech presentation, it is the sacrum. The denominator of a face presentation is the mentum or chin.
Engagement	The point at which the widest diameter of the presenting part is at or below the pelvic inlet.
Lie	Relationship of the long axis of the fetus to the long axis of the pregnant woman. The three possible lies are longitudinal, transverse, or oblique.
Position	Relationship of the denominator to the front, back, or sides of the maternal pelvis.
Presentation	The part of the fetus that presents first to the maternal pelvis. The three possible presentations are cephalic, shoulder, and breech. Breech presentations are further subdivided based on presentation of the buttocks or feet.
Presenting part	The most dependent part of the fetus that is closest to the maternal cervix.
Station	The number of centimeters above or below the plane between the ischial spines of the presenting part. The ischial spines are designated as 0 station; the centimeters above the spines are –1, –2, –3, –4, and –5; and the centimeters below the ischial spines are +1, +2, +3, +4, and +5, which is when the fetal presenting part is visible at the vaginal introitus.

The Fetal Skull

The fetal skull is composed of five bones—two frontal bones, two parietal bones, and one occipital bone—that may be palpated during labor to identify the position of the fetus and assess labor progress. In addition, the two temporal bones are located inferior to the parietal bones on each side but are not involved in the anatomic markers important during labor assessment (**Figure 19-12**). The bones meet at the frontal suture, located between the two

frontal bones; at the sagittal suture, located between the two parietal bones; at the two coronal sutures, where the parietal and frontal bones meet on either side of the head; and at the two lambdoid sutures, where the parietal bones and the upper margin of the occipital bone meet on either side of the head.

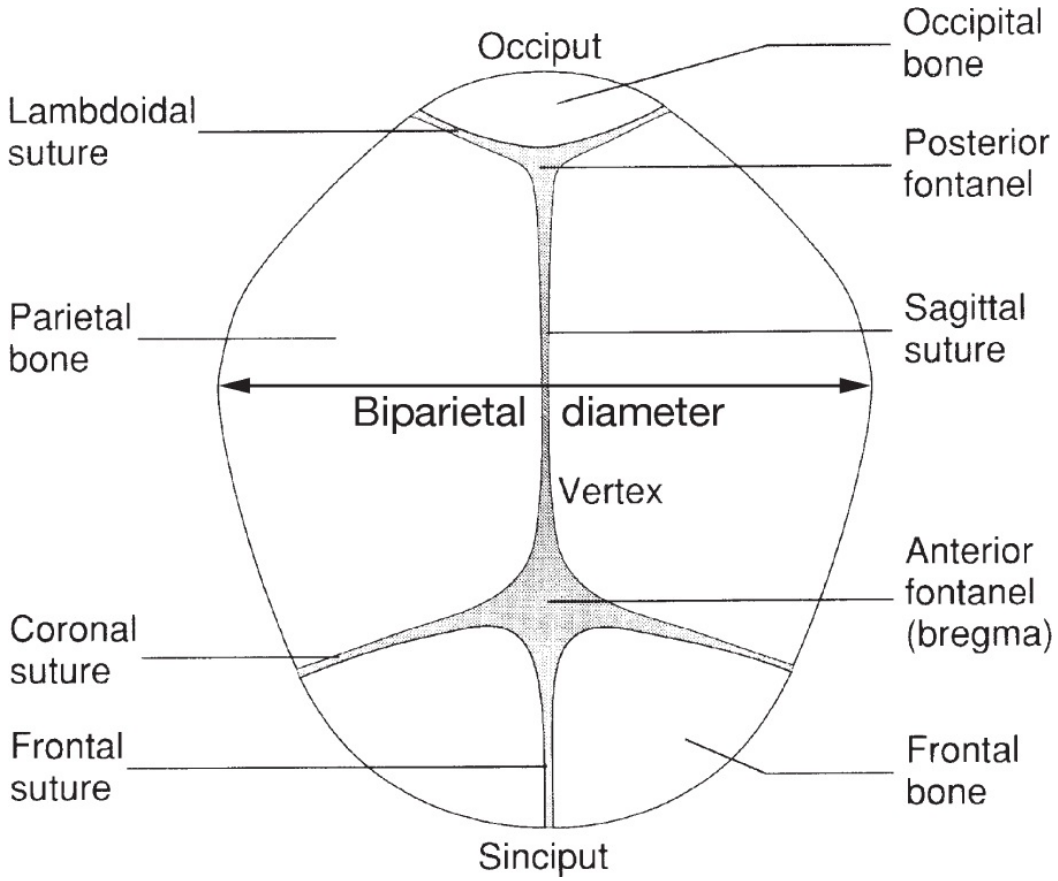


Figure 19-12 Fetal skull: landmarks, bones, fontanelles, sutures, and biparietal diameter.

Two fontanels—areas formed by the meeting of sutures—are found on either end of the sagittal suture. The anterior fontanel is the largest, formed as the frontal, sagittal, and two coronal sutures come together in a diamond-like shape.

The four sutures can be palpated coming from the four corners of diamond shape, with markers of the frontal suture being more subtle and sometimes difficult to palpate. The sagittal and two lambdoid sutures meet to form the posterior fontanel in a triangle shape; the sagittal and lambdoid sutures can be palpated from the three corners of the triangle.

The fetal head also has several diameters of importance in providing maternity care, which are shown in **Figure 19-12** and **Figure 19-13**.

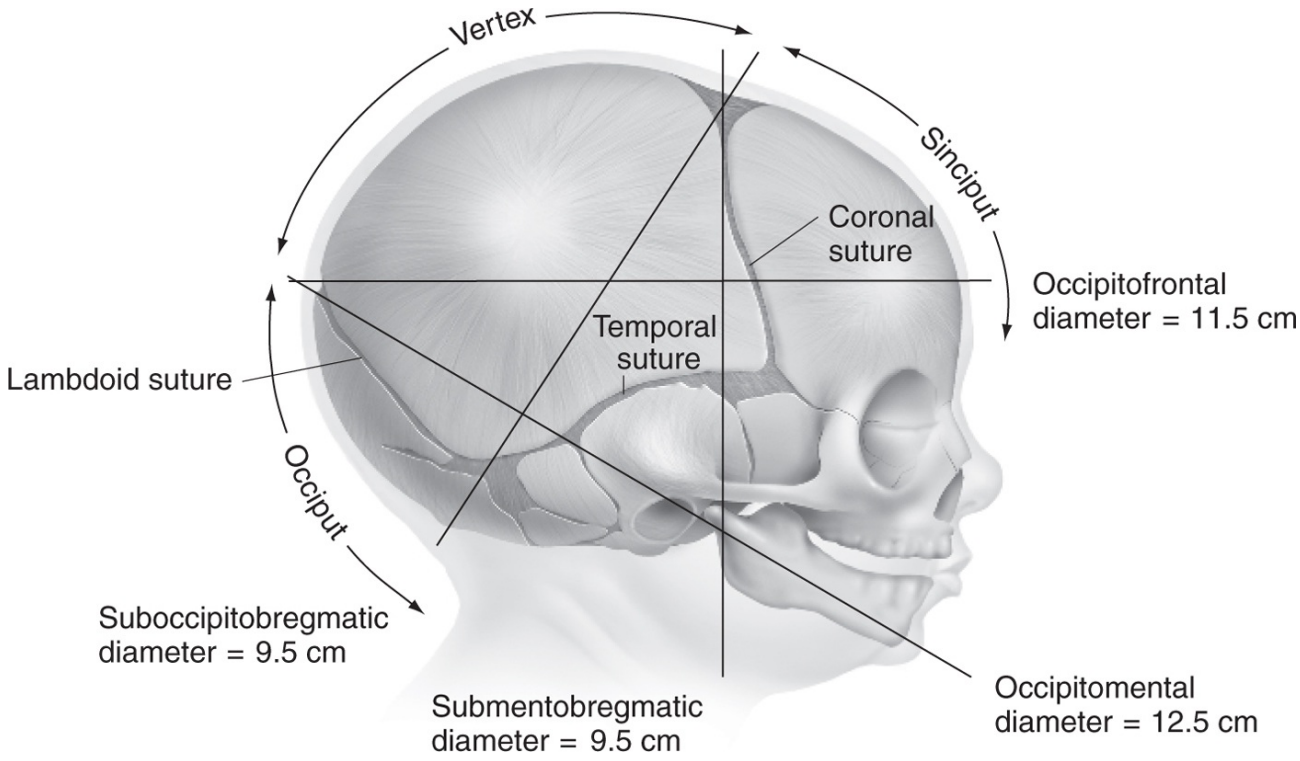


Figure 19-13 Average diameters of the full-term fetal head.

Lie, Presentation, Denominator, and Position

Determination of the lie, presentation, and position of the fetus require an understanding of terms and the anatomic landmarks of the fetal skull in relation to the maternal pelvis.

Lie is the relationship of the long axis of the fetus to the long axis of the pregnant woman. Three possible lies are longitudinal, transverse, and oblique (**Figure 19-14**). Transverse and oblique lies in labor are abnormal conditions requiring collaboration with or referral to a physician because they will likely necessitate cesarean section.

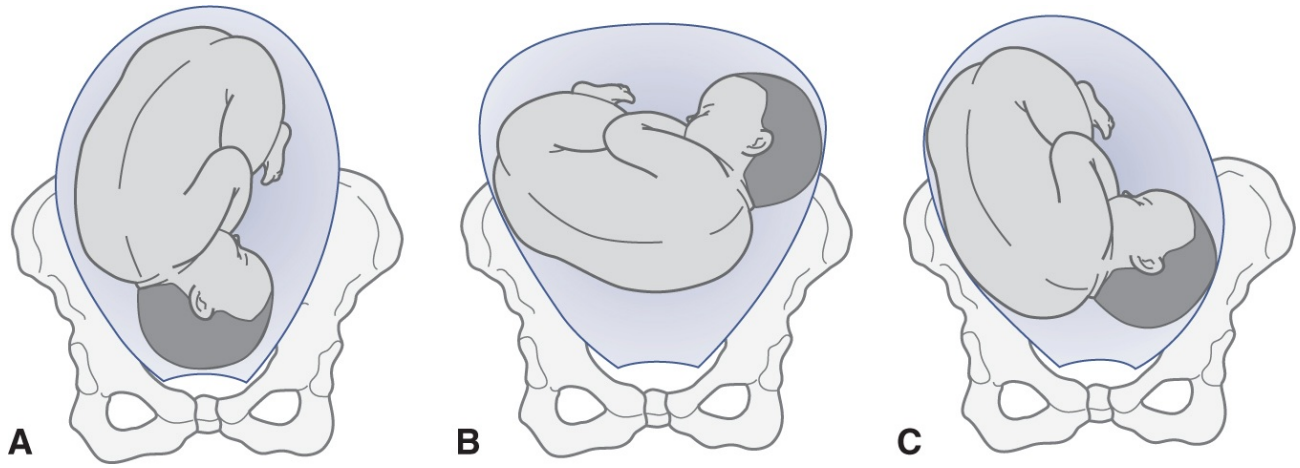


Figure 19-14 Lies. **A.** Longitudinal. **B.** Transverse. **C.** Oblique.

Presentation is determined by the presenting part—that is, the part of the fetus that first

enters the pelvic inlet. The three possible presentations are cephalic, breech, and shoulder. Cephalic and breech presentations are each further subdivided: A cephalic presentation can be vertex, sinciput, brow, or face (**Figure 19-15**), and a breech presentation can be frank (legs extended), full/complete (legs flexed), or footling (single or double). Approximately 3.0% to 3.5% of women enter labor with a breech presentation and 0.5% with a face presentation. Approximately 0.5% of women enter labor with a shoulder presentation. The midwife collaborates with a physician in the management of women with a noncephalic presentations.

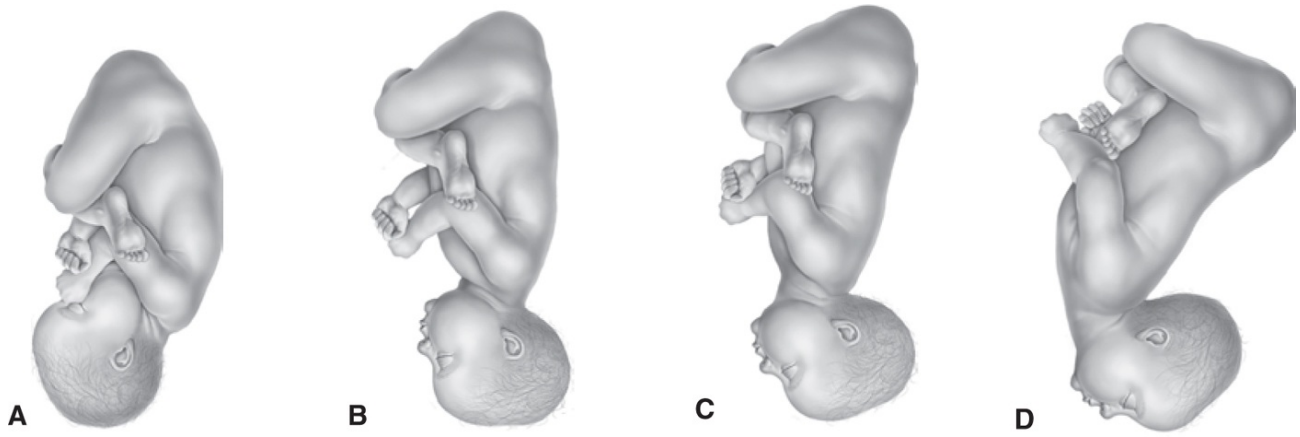


Figure 19-15 Attitude of the fetus in various presentations. **A.** Vertex. **B.** Sinciput (military). **C.** Brow. **D.** Face.

The *attitude* of the fetus is its characteristic posture, determined by the relationship of the fetal parts to one another and the effect this has on the fetal vertebral column. The attitude of the fetus varies according to its presentation. For example, a fetus in a vertex presentation has a well-flexed head, flexion of the extremities over the thorax and abdomen, and a convex curved back. By comparison, the straight upright attitude of a fetus with a sinciput presentation has resulted in the classically defined military attitude. Finally, a fetus with a face presentation has an acutely extended head, flexion of the extremities on the thorax and abdomen, and a vertebral column that is arched to some degree.

Fetal *position* is named using three letters in the following order: the first reference is to the side of the maternal pelvis (**L**eft or **R**ight); the second reference is the denominator (**O**cciput, **S**acrum, or **M**entum); and the third reference is where in the maternal pelvis the denominator lies (**A**nterior, **T**ransverse, **P**osterior). These designations serve as a shorthand description for describing the lie, presentation, and position of the denominator within the circle of the pelvis (**Figure 19-16**). For example, the designation LOA indicates that the lie is longitudinal, the presentation is cephalic, and the denominator, which is the occiput, is in the anterior portion of the left side of the pelvis. The possible fetal relationships to the maternal pelvis for each lie and presentation are summarized in **Table 19-7**.

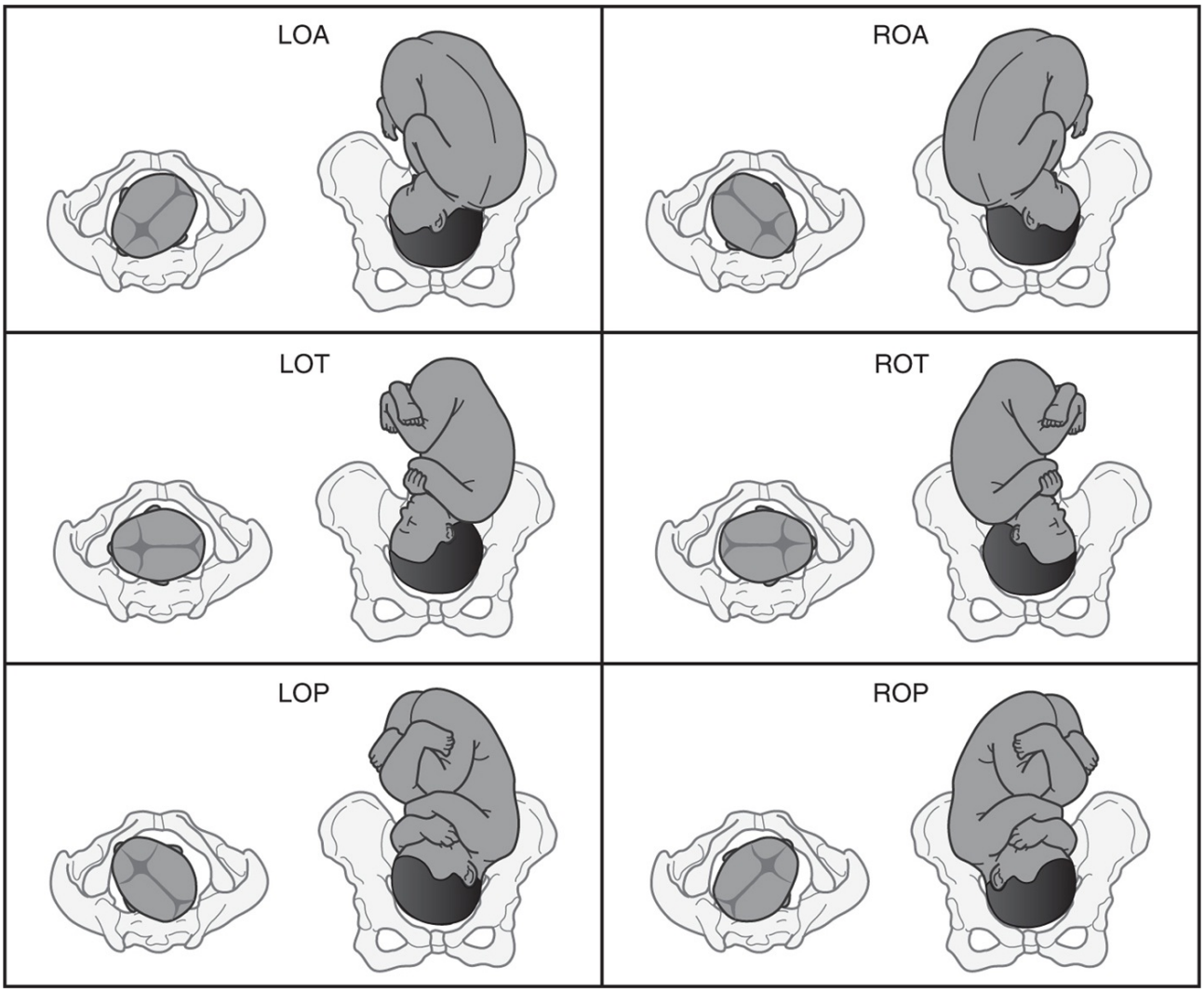


Figure 19-16 Fetal position for occiput presentations.

Abbreviations: LOA, left occiput anterior; LOP, left occiput posterior; LOT left occiput transverse; ROA right occiput anterior; ROP, right occiput posterior; ROT, right occiput transverse.

Table 19-7 **Possible Fetal Relationships to the Maternal Pelvis for Each Lie and Presentation**

Lie	Presentation	Denominator	Designation for Position
Longitudinal	Cephalic		
	Vertex	Occiput	ROA LOA
			ROT LOT
			ROP LOP
	Sinciput	Sinciput (bregma, anterior fontanel)	Sinciput and brow presentations usually convert to either a vertex or a face presentation.
	Brow	Brow	
	Face	Mentum (chin)	RMA LMA
			RMT LMT
			RMP LMP
	Breech		
	Frank	Sacrum	RSA LSA
			RST LST
			RSP LSP
	Full/complete	Sacrum	Same as frank presentation
	Footling	Sacrum	Same as frank presentation
Transverse	Shoulder	Acromion	RAA LAA
			RAP LAP
			A transverse variety is not possible.
Oblique	With an oblique lie, the midwife will feel nothing at the inlet. There is no presentation, position, or variety associated with an oblique lie, which is usually a transitory condition.		

Abbreviations: LMA, left mentum anterior; LMP, left mentum posterior; LMT, left mentum transverse; LOA, left occiput anterior; LOP, left occiput posterior; LOT left occiput transverse; LSA, left sacrum anterior; LST, left sacrum transverse; ROA, right occiput anterior; ROP, right occiput posterior; ROT, right occiput transverse; RSA, right sacrum anterior; RSP, right sacrum posterior; RST, right sacrum transverse; RMA, right mentum anterior; RMP, right mentum posterior; RMT, right mentum transverse.

Vertex is the presentation most often associated with a longitudinal lie; it has an incidence of approximately 95%. Approximately two-thirds of all fetuses will be positioned with the occiput in the left side of the maternal pelvis (LOA, LOT, LOP) by the last month of pregnancy; one-third will be positioned with the occiput in the right side of the woman's pelvis (ROA, ROT, ROP). Because the head usually enters the inlet with the occiput directed to the transverse portion of the maternal pelvis, the most common position of the fetus at the onset of labor is left occiput transverse (LOT).

In a vertex presentation, the fetal head usually enters the pelvis with the biparietal diameter parallel to the plane of the pelvis. If the fetal head is tilted laterally toward the fetal shoulder, the head will enter the pelvis at an oblique angle which is referred to as *asynclitism.*

Estimated Fetal Weight

When assessing fetopelvic relationships, a midwife is also able to estimate a fetal weight

(EFW). The average EFW at 30 gestational weeks is approximately 3 pounds or 1360 grams. A fetus usually reaches 4 pounds at approximately 33 weeks (1900 grams); and at 37 weeks it is 5 pounds 8 ounces or 2500 grams. The latter is important since it is the international definition for low birth weight. Fetal weight is important for fetopelvic relationships, since a small fetus (either constitutionally or due to disease) is more likely to present abnormally such as in a breech presentation. Conversely, a large fetus can have difficulty navigating even an average-sized pelvis.

Conclusion

Remarkable physiologic changes occur during pregnancy. Knowledge regarding physiologic pregnancy changes must inform every aspect of pregnancy care. A deep understanding of these changes will allow the midwife to interpret signs and symptoms accurately, enabling reassurance and guidance regarding managing common pregnancy discomforts or initiation of assessment for pathology. These are essential steps in the provision of quality maternity health care.

References

1. Buhimschi CS. Endocrinology of lactation. *Obstet Gynecol Clin North Am.* 2004;31:963-979.
2. Chen JZ, Sheehan PM, Brennecke SP, Keogh RJ. Vessel remodelling, pregnancy hormones and extravillous trophoblast function. *Mol Cell Endocrinol.* 2012;349(2):138-144.
3. Damjanov I. Vesalius and Hunter were right: decidua is a membrane [Editorial]. *Lab Invest.* 1985;53:597.
4. Strowitzki T, Germeyer A, Popovici R, Wolff M. The human endometrium as a fertility-determining factor. *Hum Reprod Update*. 2006;12(5):617-630.
5. Murphy CR. Understanding the apical surface markers of uterine receptivity: pinopods or uterodomes: *Hum Reprod*. 2000;15:2451-2454.
6. Gellersen B, Brosens IA, Brosens JJ. Decidualization of the human endometrium: mechanisms, functions and clinical perspectives. *Semin Reprod Med*. 2007;25(6):445-453.
7. Tingaker BK, Irestedt L. Changes in uterine innervation in pregnancy and during labour. *Curr Opin Anethesiol.* 2010;23:300-310.
8. Latini C, Frontini A, Morroni M, Marzioni D, Castellucci M, Smith PG. Remodeling of uterine innervation. *Cell Tissue Res*. 2008;334:1-6.
9. Brauer MM. Cellular and molecular mechanisms underlying plasticity in uterine sympathetic nerves. *Auton Neurosci.* 2008;140:1-16.
10. Ludmir J, Sehdev H. Anatomy and physiology of the uterine cervix. *Clin Obstet Gynecol.* 2000;43(3):433-439.
11. Timmons B, Akins M, Mahendroo M. Cervical remodeling during pregnancy and parturition. *Trends Endocrin Metab*. 2010;21(6):353-361.
12. Nott JP, Bonney EA, Pickering JD, Simpson NAB. Structure and function of the cervix during pregnancy. *Translational Res Anat.* 2016;2:1-7.
13. Vink J, Feltovich H. Cervical etiology of spontaneous preterm birth. *Semin Fetal Neonatal Med*. 2016;21(2):106-112.
14. Yao W, Gan Y, Myers KM, Vink JY, Wapner RJ, Hendon CP. Collagen fiber orientation and dispersion in the upper cervix of non-pregnant and pregnant women. *PLoS One*. 2016;11(11):e0166709. doi: 10.1371/journal.pone.0166709.
15. Fournier T. Human chorionic gonadotropin: different glycoforms and biological activity depending on its source of production. *Ann Endocrinol (Paris)*. 2016;77(2):75-81. doi: 10.1016/j.ando.2016.04.012.nier.
16. Cole LA. Biological functions of hCG and hCG-related molecules. *Reprod Biol Endocrinol*. 2010;8:102.
17. Cole LA. hCG, the wonder of today's science. *Reprod Biol Endocrinol.* 2012;10:24-28.
18. Barnhart KT, Sammel MD, Rinaudo PF, Zhou L, Hummel AC, Guao W. Symptomatic patients with an early viable intrauterine pregnancy: hCG curves redefined. *Obstet Gynecol.* 2004;104:50-55.
19. Barnhart KT, Guo W, Cary MS, et al. Differences in serum human chorionic gonadotropin rise in early pregnancy by race and value at presentation. *Obstet Gynecol.* 2016;128(3):504-511.
20. Bustos M, Venkataramanan R, Caritis S. Nausea and vomiting of pregnancy: what's new? *Auton Neurosci.* 2017;202:62-72.
21. Lager S, Powell TL. Regulation of nutrient transport across the placenta. *J Preg*. 2012;2012:179827. doi: 10.1155/2012/179827.
22. Wahabi HA, Fayed AA, Esmaeil SA, Al Zeidan RA. Progestogen for treating threatened miscarriage. *Cochrane Database Syst Rev.* 2011;12:CD005943. doi: 10.1002/14651858.CD005943.pub4.
23. Carp H. A systematic review of dydrogesterone for the treatment of threatened miscarriage. *Gynecol Endocrinol*. 2012;28(12):983-990. doi: 10.3109/09513590.2012.702875.
24. Society for Maternal-Fetal Medicine Publications Committee, Berghella V. Progesterone and preterm birth prevention: translating clinical trials data into clinical practice [erratum, *Am J Obstet Gynecol.* 2013;208:86]. *Am J Obstet Gynecol* 2012;206:376-386.
25. Brosens I, Pijnenborg R, Vercruysse L, Romero R. The "Great Obstetrical Syndromes" are associated with disorders of deep placentation. *Am J Obstet Gynecol.* 2011;204(3):193-201.
26. Costa MA. The endocrine function of human placenta: an overview. *Reprod BioMed Online*. 2016;32(1):14-43.
27. Moore KL, Persaud TVN, Torchia MG. *The Developing Human: Clinically Oriented Embryology*. 10th ed. Philadelphia, PA: Elsevier; 2015.
28. Degner K, Magness RR, Shah DM. Establishment of the human uteroplacental circulation: a historical perspective. *Reprod Sci.* 2017;24(5):753-761.
29. Fisher SJ. Why is placentation abnormal in preeclampsia? *Am J Obstet Gynecol.* 2015;213:S115-S122.
30. Pijnenborg R, Vercruysse L, Hanssens M. The uterine spiral arteries in human pregnancy: facts and controversies. *Placenta*. 2006;27:939-958.
31. Jain A. Endothelin-1: a key pathological factor in pre-eclampsia. *Reprod BioMed Online*. 2012;25:443-449.
32. Longo LD, Reynolds LP. Some historical aspects of understanding placental development, structure, and function. *Int J Dev Biol.* 2010;54:237-255.

33. Aherne W, Dunnill MS. Morphometry of the human placenta. *Br Med Bull*. 1966;22:5-8.
34. Schuler-Maloney D. Placental triage of the singleton placenta. *J Midwifery Womens Health*. 2000;45:104-113.
35. Maltepe E, Fisher SJ. Placenta: the forgotten organ. *Annu Rev Cell Dev Biol*. 2015;31:523-552.
36. Dawe GS, Tan XW, Xiao ZC. Cell migration from baby to mother. *Cell Adh Migr*. 2007;1(1):19-27.
37. Sferruzzi-Perri AN, Camm EJ. The programming power of the placenta. *Front Physiol*. 2016;7:33. doi: 10.3389/fphys.2016.00033.
38. Barker DJ. Developmental origins of chronic disease. *Public Health*. 2012;126:185-189.
39. Barker D. The midwife, the coincidence, and the hypothesis. *BMJ*. 2003;327(7429):1428-1430.
40. Barker DJ, Gluckman PD, Godfrey KM, Harding JE, Owens JA, Robinson JS. Fetal nutrition and cardiovascular disease in adult life. *Lancet*. 1993;341:938-941.
41. Gluckman PD, Hanson MA, Cooper C, Thornburg KL. Effect of in utero and early-life conditions on adult health and disease. *N Engl J Med*. 2008;359:61-73.
42. Dyer JS, Rosenfeld CR. Metabolic imprinting by prenatal, perinatal, and postnatal overnutrition: a review. *Semin Reprod Med*. 2011;29(3):266-276.
43. Lillycrop KA, Burdge GC. Epigenetic mechanisms linking early nutrition to long term health. *Best Pract Res Clin Endocrin Metab*. 2012;26:667-676.
44. Lau C, Rogers JM. Embryonic and fetal programming of physiological disorders in adulthood. *Birth Defects Res C Embryo Today*. 2004 Dec;72(4):300-312.
45. Roberts VHJ, Frias AE, Grove KL. Impact of maternal obesity on fetal programming of cardiovascular disease. *Physiology*. 2015;30(3):224-231. doi: 10.1152/physiol.00021.2014.
46. Alexander BT, Dasinger JH, Intapad S. Fetal programming and cardiovascular pathology. *Compr Physiol*. 2015;5(2):997-1025. doi: 10.1002/cphy.c140036.
47. Burlingham WJ. A lesson in tolerance—maternal instruction to fetal cells. *N Engl J Med*. 2009;360(13):1355-1357.
48. Latendresse G, Founds S. The fascinating and complex role of the placenta in pregnancy and fetal well-being. *J Midwifery Womens Health*. 2015;60(4):360-370.
49. Modena AB, Fieni S. Amniotic fluid dynamics. *Acta Biomed*. 2004;75(suppl 1):11-13.
50. Moore TR. Amniotic fluid dynamics reflect fetal and maternal health and disease. *Obstet Gynecol*. 2010;116(3):757-763.
51. Derbala Y, Grochal F, Jeanty P. Vasa previa. *J Perinat Med*. 2007;1(1):2-13.
52. Murphy VE, Smith R, Giles WB, Clifton VL. Endocrine regulation of human fetal growth: the role of the mother, placenta, and fetus. *Endocrine Rev*. 2006;27:141-169.
53. Lieberman E, Gremy I, Lang JM, Cohen AP. Low birthweight at term and the timing of fetal exposure to maternal smoking. *Am J Public Health*. 1994; 84:1127-1131.
54. de Vries JIP, Fong BF. Normal fetal motility: an overview. *Ultrasound Obstet Gynecol*. 2006;27:701-711.
55. Martin CB. Normal fetal physiology and behavior, and adaptive responses with hypoxemia. *Semin Perinatol*. 2008;32:239-242.
56. Borg-Stein J, Duagn S, Gruber J. Musculoskeletal aspects of pregnancy. *Am J Phys Med Rehabil*. 2005;84:180-192.
57. Geraghty LN, Pomeranz MK. Physiologic changes and dermatoses of pregnancy. *Int J Dermatol*. 2011;50:771-782.
58. Ozuounian JG, Elkayam U. Physiologic changes during normal pregnancy and delivery. *Cardiol Clin*. 2012;30:317-329.
59. Bieber AK, Martires KJ, Stein JA, Grant-Kels JM, Driscoll MS, Pomeranz MK. Pigmentation and pregnancy: knowing what is normal. *Obstet Gynecol*. 2017;129(1):168-173.
60. Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. *Circulation*. 2014;130:1003-1008.
61. Stangret A, Skoda M, Whuk A, Pzylak M, Szukiewicz D. Mild anemia during pregnancy upregulates placental vascularity development. *Med Hypoth*. 2017;102:37-40.
62. Barrett JF, Whittaker PG, Williams JG, Lind T. Absorption of non-haem iron from food during normal pregnancy. *BMJ*. 1994;309:79-82.
63. Wise RA, Polito AJ, Krishnan V. Respiratory physiologic changes in pregnancy. *Immunol Clin North Am*. 2006;26:1-12.
64. Elkus R, Popovich J. Respiratory physiology in pregnancy. *Clin Chest Med*. 1992;12:555-565.
65. Cornelis T, Odutayo A, Keunen J, Hladunewich M. The kidney in normal pregnancy and preeclampsia. *Semin Nephrol*. 2011;31:4-14.
66. Alemu A, Terefe B, Abebe M, Biadgo B. Thyroid hormone dysfunction during pregnancy: a review. *Int J Reprod Biomed*. 2016;14(11):677-686.
67. Casey MB, Leveno K. Thyroid disease in pregnancy. *Hil Obstet Gynecol*. 2006;108:1283-1292.
68. Brent GA. Maternal thyroid function: interpretation of thyroid function tests in pregnancy. *Clin Obstet Gynecol*. 1997;40:3-15.
69. Baeyens L, Hindi S, Sorenson RL, German MS. β-cell adaptation in pregnancy. *Diab Obes Metab*. 2016;18(suppl 1):63-70.
70. Herrera E. Lipid metabolism in pregnancy and its consequences in the fetus and newborn. *Endocrine*. 2002;19(1):43-55.

71. Zeng Z, Liu F, Li S. Metabolic adaptations in pregnancy. *Ann Nutr Metab*. 2017;70:59-65.
72. Munoz-Suano A, Hamilton AB, Betz AG. Gimme shelter: the immune system during pregnancy. *Immunol Rev*. 2011;241:20-38.
73. Morelli SS, Mandal M, Goldsmith LT, Kashani BN, Ponzio NM. The maternal immune system during pregnancy and its influence on fetal development. *Res Reports Biol*. October 2015. Available at: https://www.dovepress.com/the-maternal-immune-system-during-pregnancy-and-its-influence-on-fetal-peer-reviewed-fulltext-article-RRB#. Accessed June 14, 2017.

CHAPTER

20

Genetics

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Introduction

Essentially all health and disease conditions have a genetic component, and the provision of midwifery care increasingly will include genetics within the context of prevention, screening, diagnosis, and treatment selection. Furthermore, genetic disorders are not rare occurrences. Approximately 3% to 7% of the population will be identified with a genetic disorder at some point in life.[1] Chromosomal abnormalities occur in approximately 10% to 20% of pregnancies and are an important contributor to pregnancy loss; 95% of conceptions affected by chromosomal abnormalities are miscarried before term.[2] Approximately 6% of all newborns have a major congenital malformation; in these newborns, 15% of the anomalies are "familial," 10% are chromosomal, 3% to 4% are related to single gene mutations, 23% are multifactorial (i.e., involve multiple genetic and environmental components), and 3% are due to teratogens.[2,3] Thus, genetic effects are a substantial contributor to newborn/infant mortality.

A solid understanding of genetics and available technology, as well as essential skills necessary for genetic risk assessment, screening, diagnostic testing, basic counseling, and appropriate referral, are needed for clinical midwifery practice. However, because genetic science and technology is evolving rapidly, it can be challenging to stay current with the knowledge necessary for clinical practice.[4] This chapter reviews basic genetics and contemporary approaches to genetic testing for women contemplating pregnancy and during pregnancy.

Foundations in Genetics

The foundations of molecular genetics and heredity provide the basis for understanding perinatal genetics specifically. This section reviews basic principles in molecular genetics, as well as inheritance patterns and the meaning of genetic mutations, gene expression, and chromosomal structure. **Table 20-1** provides a glossary of commonly used genetics-related terms.[5-7] Readers are also encouraged to access the many excellent genetics resources currently available in print as well as online for more in-depth information. A select list of online resources can be found at the end of this chapter.

Table 20-1 Genetics Glossary

Term	Definition/Description
Allele	One of the two or more versions of a genetic sequence that encodes for the same protein or function at a specific location on a chromosome. An individual normally inherits one allele from each parent and these matching alleles contain the genes that code for the same characteristic.
Aneuploidy	A chromosomal condition in which there is an abnormal number of chromosomes in the complement of 23 pairs, secondary to either a deletion or an addition of a chromosome.
Autosome	A chromosome that is not a sex chromosome. Autosomes appear in pairs.
Base pair	Two nitrogenous bases paired together in double-stranded DNA (i.e., adenine paired with thymine, and guanine paired with cytosine). Sequences of various lengths of base pairs make up the various genes.
Cell-free fetal DNA	Fetal genetic material (DNA) that is not contained within cells and that circulates freely in the blood of a pregnant woman. Cell-free fetal DNA is derived primarily from the placenta and can currently be used to test for aneuploidy in high-risk women.
CFTR	The gene that encodes for chloride channel transmembrane regulation. Several variations of mutations in this gene are known, including those that directly cause cystic fibrosis.
Chromosomal abnormality	An alteration in the number or structure of a chromosome. Chromosomal abnormalities can be inherited or occur de novo. Most common chromosomal abnormalities, such as trisomy 21, are not heritable.
Chromosomal microarray analysis (CMA)	A technique for identifying chromosomal abnormalities, including those that are too small (i.e., microdeletions) to be detected by conventional karyotyping. CMA requires direct testing of fetal tissue, so it can be offered only when chorionic villus sampling (CVS) or amniocentesis is performed.
Chromosomal microdeletion	A very small deletion of a part of a chromosome or sequence of DNA, which results in a loss of genetic material. These microdeletions are usually not fatal, but they frequently result in physical and mental abnormalities depending on the genetic material that is lost. Conventional karyotyping is not able to identify microdeletions. Cri-du-chat syndrome is caused by a microdeletion on the short arm of chromosome 5.
Chromosomal translocation	A rearrangement of chromosomal segments between different chromosomes. These can be *balanced* (even exchange of material) or *unbalanced* (unequal exchange of material, resulting in extra or missing genetic material). Although there is frequently no effect on phenotype, individuals with chromosomal translocations may have an increased risk of nonviable conception and trisomy 21.
Deoxyribonucleic acid (DNA)	The double-stranded helix of nitrogenous base pairs within cells that provides instructions for all cell activity, and is passed on from generation to generation. The DNA strand comprises the collection of genes within a chromosome.
Dominant allele	An allele that, if present, leads to expression of the phenotype associated with that allele.

	Dominant alleles can mask the presence of a recessive allele.
Epigenetics	Changes in the regulation of gene expression that occur due to modifications in DNA, rather than changes in DNA sequence. Methylation—the attachment of methyl groups to DNA at cytosine bases—is one example of a DNA modification that alters gene expression.
Exon	The segment of the gene that codes for a specific amino acid. By comparison, introns are generally considered noncoding segments of DNA and are normally spliced out of the sequence prior to formation of amino acids.
Expressivity	Phenotypic variation (severity) among persons who have a specific genotype.
Gene	The fundamental unit of heredity. An ordered sequence of DNA constitutes a gene; it is found on a specific location on a specific chromosome. Each gene encodes for a specific functional protein.
Gene expression	The process by which information from a gene is used to produce a functional gene product (usually a protein); it is how a genotype results in a particular phenotype. Not all genes are expressed.
Genetic disorder	Alterations in genes that code for a particular protein, which results in a heritable disorder.
Genome	The entire DNA sequence and set of genetic instructions found within each cell. The genome is different for each organism. The Human Genome Project documented the entire DNA sequence of the approximately 25,000 genes held within the 23 pairs of chromosomes in humans.
Genotype	A person's complete collection of specific genes. A person's genotype is largely responsible for determining the phenotype.
Heterozygous	Referring to an individual who has a mutant allele on only one of a pair of specific genes. A specific phenotype in a heterozygous individual is the result of a dominant gene mutation, requiring only one mutant allele. Conversely, a heterozygous individual with a recessive gene mutation is referred to as a "carrier" and does not exhibit the phenotype.
Homozygous	Referring to an individual who has a mutant allele on both genes in a specific gene pair (one from each parent). Autosomal recessive inheritance of a trait, condition, or disease occurs only when an individual is homozygous for the specific gene. Cystic fibrosis and sickle cell anemia, for example, occur only in individuals who are homozygous for the specific disease gene.
Incomplete penetrance	Genetic mutation that does not always result in the phenotype associated with that mutation. Often the expression of a gene with incomplete penetrance depends on the presence of other environmental factors.
Karyotype	An individual's full set of chromosomes. Also, the standardized laboratory result or visual display (i.e., a photograph) of an individual's set of chromosomes.
Meiosis	Cell division that results in sperm or ova. Each daughter cell contains only half the original number of chromosomes.
Monosomy	A chromosomal condition in which a single chromosome is found when there should be a chromosome pair. These mutations are almost always lethal when found in autosomal chromosomes.
Multifactorial disease	A condition caused by the interactions between several genes and several environmental factors. Cancer, type 2 diabetes mellitus, and heart disease are all multifactorial conditions.
Mutation	A change in DNA sequence that may or may not result in a change in the protein product. Mutations may affect only one base pair or a larger segment in the DNA sequence via deletion, insertion, repetition, or duplication. Genetic mutations cause such disorders as cystic fibrosis, Tay–Sachs disease, and sickle cell anemia.
Nondisjunction	Failure of chromosome pairs to separate properly during meiosis. Meiosis is the type of cell reproduction that results in gametes (eggs and sperm), in which each gamete has only one set of 23 chromosomes rather than a full complement of 46 chromosomes in 23 paired sets.
Penetrance	The proportion of persons with a specific genotype who also express the expected phenotype. For example, if a particular gene mutation has 95% penetrance, then 95% of persons with that mutation will have the associated phenotype.
Phenotype	The observable traits or physical expression of an individual's genotype, which is also influenced

	by environmental factors.
Recessive allele	An allele that expresses a particular phenotype only if both alleles in the pair are recessive.
Ribonucleic acid (RNA)	A single-stranded nitrogenous molecule that mirrors the DNA sequence. RNA is essential for moving DNA instructions out of the nucleus (via transcription and translation) and into the cell cytoplasm, and for subsequent construction of amino acids into final protein products.
Sex-linked	A gene that is located on an X or Y sex chromosome.
Single-nucleotide polymorphism (SNP)	Pronounced “snip”; any of the common variations found in a single nucleotide of genomic DNA sequence between individuals, as well as between different populations.
Transcription	The synthesis of an RNA strand from a sequence of DNA; the first step in gene expression.
Translation	During protein synthesis, the process through which the sequence of bases in a molecule of messenger RNA is read to create a sequence of amino acids.
Trisomy	A chromosomal condition in which three chromosomes occur where there should be only a pair. Trisomy is a form of aneuploidy. The occurrence of trisomy increases in infants born to older mothers. A common trisomy condition is trisomy 21 (Down syndrome).

Based on Feero WG, Guttmacher AE, Collins FS. Genomic medicine: an updated primer. *N Engl J Med.* 2010;362:2001-2011[5]; National Cancer Institute. NCI dictionary of genetics terms. Available at: https://www.cancer.gov/publications/dictionaries/genetics-dictionary. Accessed June 25, 2017[6]; Elston R, Satagopan J, Sun S. Genetic terminology. *Meth Molec Biol.* 2012;850:1-9.[7]

Genes, Genome, DNA, and Chromosomes

Genes—the basic unit of inheritance—consist of *deoxyribonucleic acid (DNA)* that is found within the cell nucleus; genes are contained within the 46 chromosomes found in every cell in the human body, except ova and sperm cells, which contain 23 chromosomes. *Chromosomes* consist of long segments of DNA that are tightly wrapped around proteins (**Figure 20-1**).[8] The human cell normally contains 46 chromosomes that are organized in 23 paired sets. In each of these sets, one chromosome is contributed by the individual’s biological mother and the other is contributed by the individual’s biological father. Twenty-two of those pairs are called *autosomes* and are the same in males and females. The last pair consists of the sex chromosomes: Females have two X chromosomes and males have an X and a Y chromosome.

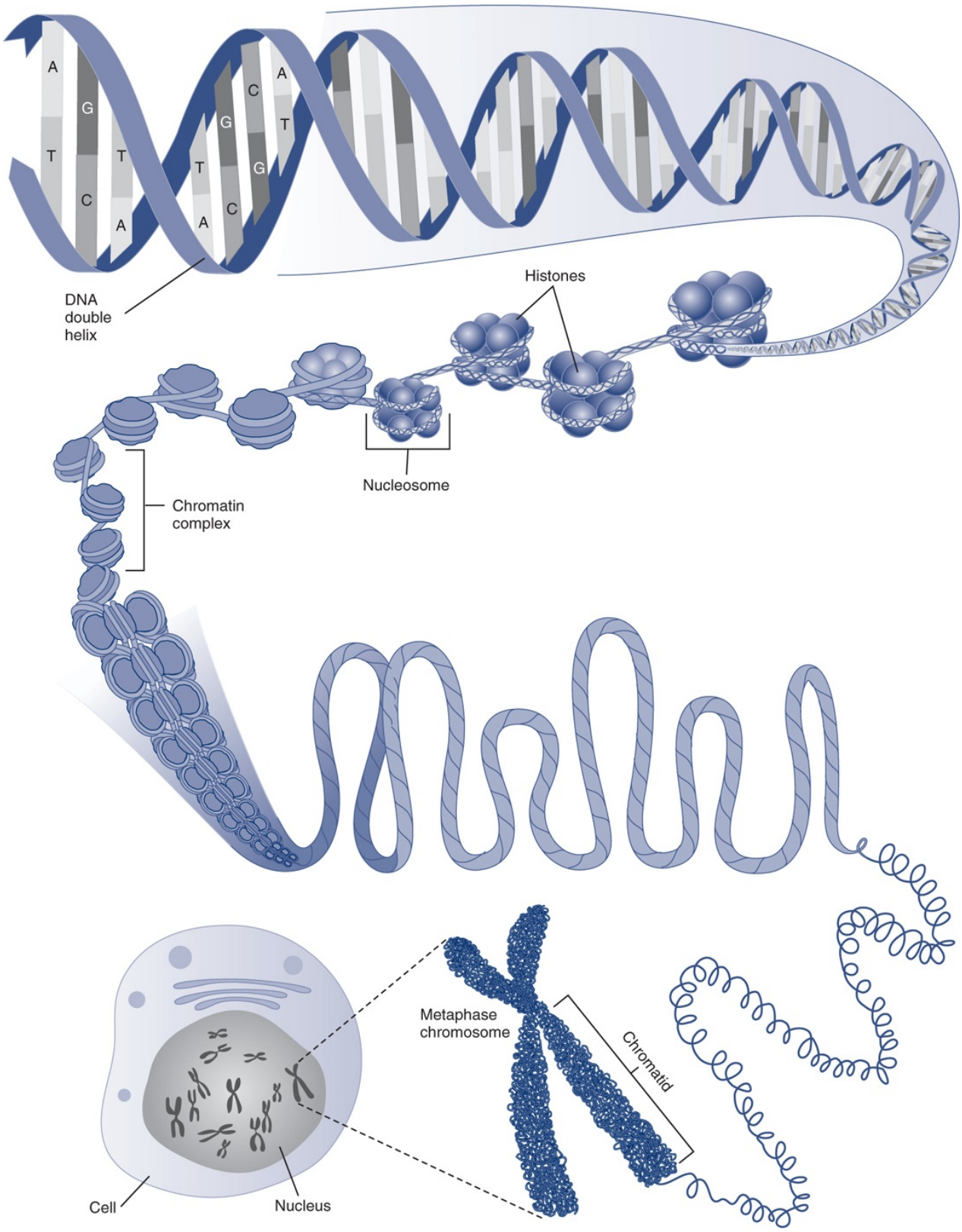


Figure 20-1 Patterns of DNA coiling. DNA is wound around eight histone cores (proteins) to form nucleosomes. Nucleosomes make up the chromatin complex, which is tightly coiled into a chromatid. The chromosome is made up of chromatids, which are held together by a centromere.

The entire collection of genes in an individual, referred to as the *genome*, provides a complete set of "instructions" for directing all biologic functions within the living organism.

The end product of gene instruction is the production of a protein, via the processes of *transcription* and *translation* of the DNA that occur as the instructions are used to construct actual proteins.[9] Every gene comprises a sequence of nitrogenous base pairs—molecules of cytosine, thymine, adenine, and guanine—that form the double-stranded DNA molecule, and each genetic sequence encodes for a specific protein with a specific function. The single-stranded *ribonucleic acid (RNA*) contributes to this process by providing a template for the eventual assembly of amino acids into proteins within the cell's cytoplasm.

The human genome has approximately 20,000 genes in chromosomes and 37 genes in the mitochondria of each nucleated cell. Approximately 1% of these genes are translated into proteins. The rest are involved in regulation or repression of the protein-coding genes.[10]

Gene expression refers to the eventual production of a gene product, although not all genes are expressed in every cell or in precisely the same way. For example, an epithelial cell (a skin cell) does not look or function the same way as a myocyte (a muscle cell). Although both cell types contain the exact same set of genetic instructions, only those genes that are expressed will contribute to the unique structure and function of the specific cell type.[11] Gene expression is regulated by a complex set of signals within an organism. This allows undifferentiated cells (i.e., fetal stem cells) to evolve into a wide variety of cells with differing structures and functions. The term *epigenetic* refers to the regulation of gene expression via modifications of the DNA structure (i.e., histones or chromatin, as noted in **Figure 20-1**), but not modification of the DNA sequence itself.[12] Environmental signals, both internal and external to an organism, are now known to make tremendous contributions to the regulation of gene expression.[10-12]

Genetic Mutations

Mutation refers to a permanent alteration in the DNA sequence within a gene, either by deletion, insertion, repetition, or duplication of a single base pair or larger segments of the DNA strand, entire genes, or pieces of chromosomes. Mutations occur rather frequently, and are often repaired by the body; a large number of them do not result in a clinical effect or disease.[1,10] However, a genetic mutation that causes a change in the protein product can subsequently alter the function of the cell. This change results in the disease *phenotype*, which refers to clinically or physically observed signs or symptoms that are reflective of the *genotype* within the individual. Genetic mutations can occur spontaneously or can be inherited from parent to child.

Alleles and Penetrance

All of the genes on a given maternally inherited chromosome correspond to the same gene in the same location (*allele*) on the paternal chromosome counterpart. Individuals with a mutant allele on only one of the pair of genes are referred to as "heterozygous," while those with a mutant allele on both genes in the pair are referred to as "homozygous."

Penetrance refers to the percentage of individuals with a specific disease genotype who also express the expected phenotype (the observable characteristics of the genotype). For example, a genetic mutation that is not associated with an expected phenotype 100% of the time

has “incomplete penetrance,” whereas a penetrance of 50% indicates that only half of the individuals with a specific disease genotype will exhibit the phenotype. The variation in phenotype (i.e., severity) of a particular genetic disorder, which is called *expressivity*, can be influenced by modifier genes, and perhaps aging or the environment. Expressivity contributes to the wide range of abilities that is observed among individuals with Down syndrome, for example. Knowledge about penetrance and expressivity helps to answer the question of why all individuals with the same genotype do not have the same phenotypic characteristics or severity of the genetic disorder.

Inheritance Patterns of Genetic Mutations

Genes, as the basic unit of inheritance, contribute significantly to an individual's characteristics, including disposition for health and disease in addition to aspects of physical appearance, such as height, eye color, and hair color. Mutations that occur in somatic cells are called acquired mutations and are significant when they lead to malignant transformation of the cell such as occurs in cancer. Genetic mutations that occur in sperm or eggs can be inherited, and there are several different patterns of inheritance.

Patterns of inheritance are either *autosomal*, which means the inherited genes are located on the first 22 pairs of chromosomes, or *sex-linked*, which means the inherited genes are located on the X or Y chromosome.[13] In addition, they are either dominant or recessive. Many well-known heritable genetic disorders are caused by a mutation in a single gene. These conditions are referred to as classic *Mendelian* disorders, after the early geneticist Gregor Mendel. Mendel was an Austrian monk who is usually called the "father of genetics" because he was one of the first scientists to clearly establish that physical traits are heritable from one generation to the next.[1,13] The classic Mendelian patterns of inheritance are autosomal dominant, autosomal recessive, X-linked dominant, X-linked recessive, and Y-linked.

In contrast, most chromosomal abnormalities are not heritable. This clinical distinction is important because tests for chromosomal abnormalities are often inaccurately referred to as "genetic tests." Use of the word "genetics" when referring to tests for both genetic disorders and chromosomal abnormalities can mistakenly convey the idea that chromosomal abnormalities are inherited. Thus, when a midwife discusses genetic testing with individuals and their families, inheritance is an important concept to include in the discussion.

Autosomal Dominant Inheritance

Disease phenotypes expressed only in *heterozygous* individuals (i.e., those persons with only one mutant allele in a specific gene pair) reflect an autosomal dominant inheritance pattern.[1,13] Only one copy of the mutant allele is required for expression of such a disorder. Examples of autosomal dominant disorders include achondroplasia (commonly known as dwarfism), neurofibromatosis, Marfan's syndrome, and Huntington disease. An individual who has an autosomal dominant condition has a 50% probability of having a child with the same disease-causing mutation. This is true for both males and females. The existence of an autosomal dominant condition is usually well known to the affected family because it is commonly observed within all generations of the family tree, unlike autosomal recessive disorders.

The occurrence of spontaneous mutations in either the egg or the sperm explains why autosomal dominant disorders can occur in individuals with no family history of the condition. Since the mutation has not been inherited from the parents, the affected individual is the first in the family to experience the disorder. Although the disorder has not been inherited from the individual's parents, the affected individual will now have a 50% probability of transmitting the mutation to offspring. The parents of an individual who has an autosomal dominant disorder resulting from a spontaneous mutation have a low risk of having another child with the mutation.[2]

A diagram referred to as a Punnett square is often used to demonstrate the probability of inheritance in offspring of affected and unaffected parents, as shown in **Figure 20-2A**. A typical corresponding pedigree reflective of autosomal dominant inheritance is shown in **Figure 20-2B**.

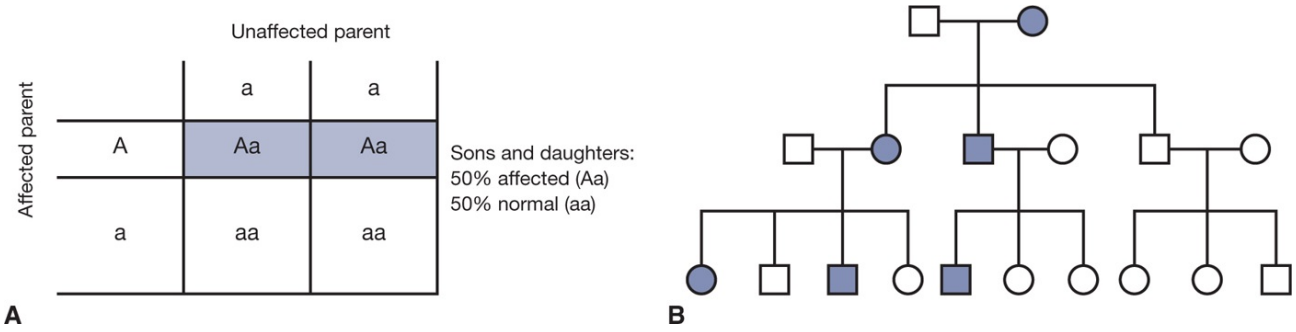


Figure 20-2 A. Probability of offspring inheriting an autosomal dominant disorder. This diagram demonstrates the probability of offspring inheriting an autosomal dominant disorder from a heterozygous (affected) parent and an unaffected parent. Shading indicates affected individuals. **B.** The corresponding dominant pedigree pattern. Pedigree showing inheritance pattern of an autosomal dominant disorder. Both genders are equally affected. Solid shapes represent affected individuals and open shapes represent unaffected individuals. Circles represent females and squares represent males.

Abbreviations: A, chromosome with a dominant disease allele; a, chromosome with a normal allele.

Autosomal Recessive Inheritance

Disease phenotypes expressed only in homozygous individuals (i.e., those persons with two mutant alleles in the specific gene pair) indicate an autosomal recessive inheritance pattern.[13] Individuals who are heterozygous for an autosomal recessive inheritance pattern are considered “carriers” and are largely unaffected by the disease. However, these carriers can pass the genetic mutation to their offspring. A carrier’s single working gene is sufficient for normal function. Cystic fibrosis (CF), Tay–Sachs disease, and sickle cell anemia are examples of autosomal recessive disorders.

A man and a woman who have the same autosomal recessive mutation will have a 25% probability that any pregnancy achieved from their union will result in a child who is affected with the disorder—that is, a child who is homozygous for the mutation (two mutant alleles). Additionally, any child from this same union has a 25% probability of being an unaffected noncarrier (no mutant alleles) of the mutation and a 50% probability of being an unaffected carrier (one mutant allele). Since autosomal recessive mutations have an inheritance pattern that results in less frequent occurrence of the disorder (frequently skipping generations in which the condition is observed), families are often unaware that the disease mutation exists. No one in the immediate family may have the disorder, and therefore, families are not aware of the possibility that a family member will have the disorder until an affected child is born.

The number of persons who are carriers for an autosomal recessive disorder varies greatly by race and ethnicity. Although carrier status for some autosomal recessive disorders is quite common among some populations, the incidence of newborns being affected with the disorder actually is much lower as noted in **Table 20-2**. **Figure 20-3A** demonstrates the probability of

inheritance in the offspring of parents who are carriers of an autosomal recessive disorder, and **Figure 20-3B** shows a typical corresponding pedigree.

Table 20-2 Autosomal Recessive Carrier Frequency in Specific Populations and Associated Incidence in Newborns

Genetic Disease	Ethnic Group	Carrier Frequency	Newborn Incidence
Tay–Sachs disease	Ashkenazi Jews	1/30	1/3600
Sickle cell disease	African Americans	1/12	1/600
Cystic fibrosis	Northern Europeans	1/25	1/2500
β-Thalassemia	Greeks/ Italians	1/30	1/3600
α-Thalassemia	Southeast Asians, Chinese	1/25	1/2500

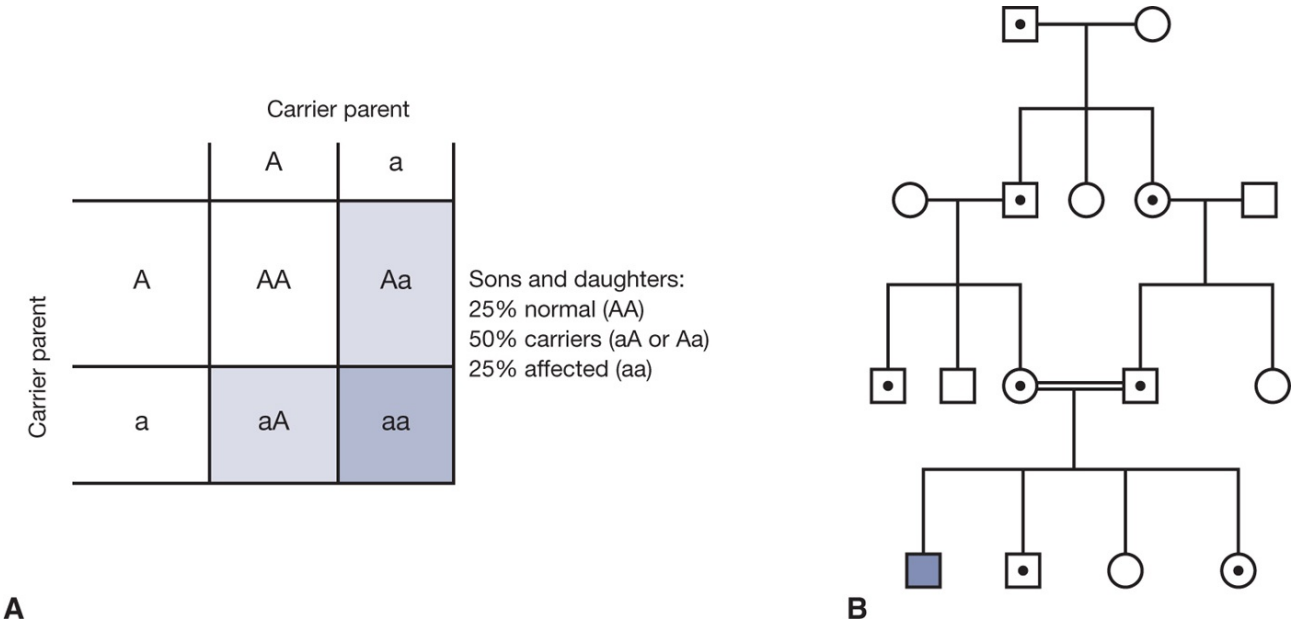


Figure 20-3 A. Probability of offspring inheriting an autosomal recessive disorder. Diagram

demonstrating the probability of offspring inheriting an autosomal recessive disorder from two heterozygous (carrier) parents. Light shading indicates carrier status and heavy shading indicates affected individuals. **B.** The corresponding autosomal recessive pedigree pattern. Pedigree showing the inheritance pattern of an autosomal recessive disorder. Consanguinity is denoted as a double line in this figure, but should not be interpreted as a requirement for inheritance. Solid shapes represent affected individuals and open shapes represent unaffected individuals. Circles represent females and squares represent males. Shapes containing a dot represent carriers of an autosomal recessive mutation.

Abbreviations: A, chromosome with a normal allele; a, chromosome with the disease allele.

Sex-Linked Inheritance

Disorders caused by mutations on the sex chromosomes are inherited differently than disorders inherited via autosomal mutations. Since females do not have a Y chromosome, Y-linked disorders do not occur in biological females. The Y chromosome also contains far fewer genes than the X chromosome, creating far fewer opportunities for mutation among biological males. Therefore, Y-linked disorders are extremely rare. In contrast, X-linked disorders are more common and predominantly affect males who do not have the paired normal X chromosome that females have. A paired X chromosome, as occurs in females, could contribute a normally functioning allele to compensate for a mutant allele. The inheritance pattern of X-linked mutations results in unaffected female carriers of the mutation and affected males with a Y chromosome that cannot compensate for the mutation occurring on the X chromosome. In the male, the single mutation on the X chromosome is sufficient to cause the disorder.

A female who is a carrier for an X-linked recessive mutation has four possible pregnancy outcomes (assuming a male partner without an X-linked disorder): a 25% chance for an unaffected carrier female (one mutant X allele and one normal X allele), a 25% chance for an unaffected noncarrier female (two normal X alleles), a 25% chance for an affected male (one mutant X allele, one normal Y allele unable to compensate), and a 25% chance for a healthy male (normal X and Y alleles).[13] An affected male has two possible outcomes for his offspring (assuming a female partner who is not a carrier for an X-linked disorder): a carrier female (one mutant X allele and one normal X allele) and an unaffected, noncarrier male (males contribute only a Y chromosome to their male offspring). **Figure 20-4A** illustrates the inheritance probabilities for X-linked disorders among offspring of a carrier mother and an unaffected father, by far the most common occurrence. **Figure 20-4B** shows a typical corresponding pedigree.

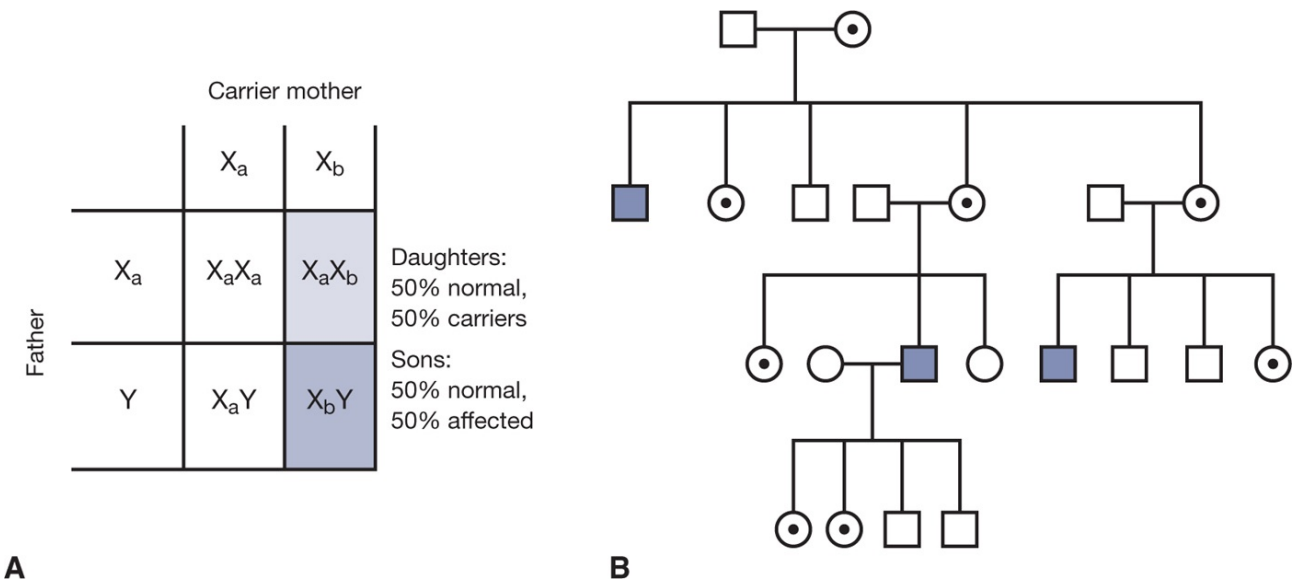


Figure 20-4 A. Probability of offspring inheriting an X-linked recessive disorder. Diagram demonstrating the probability of offspring inheriting an X-linked recessive disorder from a heterozygous mother (carrier) and an unaffected, noncarrier father. Light shading indicates carrier status. Heavy shading indicates an affected individual. **B.** The corresponding X-linked recessive pedigree pattern. Pedigree showing inheritance pattern of an X-linked recessive disorder. Only males are affected. Only females are carriers. For offspring of a carrier mother and a normal father, in each pregnancy there is an independent chance that for males there is a 50% chance the male will be affected and a 50% chance the male will be unaffected. For females there is a 50% the daughter will be a carrier, and a 50% chance the daughter will be unaffected. For offspring of affected males, 100% of daughters are carriers and 100% of sons are normal. Solid shapes represent affected individuals and open shapes represent unaffected individuals. Circles represent females and squares represent males. Shapes containing a dot represent carriers of an autosomal recessive mutation.

Abbreviations: Xa, chromosome with a normal allele; Xb, chromosome with the disease allele.

Hemophilia A and Duchenne muscular dystrophy are among the more common X-linked disorders. One way to distinguish X-linked from autosomal dominant conditions when analyzing a pedigree is to determine whether affected males have affected sons. Male-to-male transmission is observed with autosomal dominant conditions but not with X-linked recessive traits.

X-linked dominant disorders exist, but appear rarely in the population. These conditions also affect heterozygous females. Upon pedigree analysis, none of the sons but all of the daughters of affected males will be affected.

Multifactorial Inheritance

Many common conditions and disorders occur due to multifactorial inheritance, meaning that they are caused by the combined effects of several genes (genetic predisposition) and several environmental factors.[1] It is also generally accepted that a “threshold” must be reached for the disease or condition to become manifest. In other words, an additive effect of genetic predisposition and increasing environmental “load” will cause an individual to reach a certain “tipping point” when the disease phenotype will occur. Gender, age, socioeconomic status, lifestyle, nutrition status, geographic area, and ethnic background frequently influence the

occurrence of a multifactorial condition or disease. Neural tube defects (spina bifida and anencephaly) and congenital pyloric stenosis are common examples of multifactorial conditions that are present at birth. Cancer, type 2 diabetes mellitus, and heart disease are multifactorial conditions that usually manifest in adulthood.

Neural Tube Defects

Neural tube defects (NTDs) are examples of multifactorial conditions that contribute significantly to adverse birth outcomes. NTDs occur when the neural tube fails to develop and close properly during very early embryonic life (2–6 weeks' gestational age).[11] Multiple genes —particularly those associated with folate metabolism—contribute to a genetic predisposition for NTDs; environmental factors, such as maternal age, diet, geographic area, drug exposure, and socioeconomic status, are associated with these defects as well.[11] Spina bifida (protrusion of the spinal tissue through the vertebral column) and anencephaly (partial or complete absence of the cranial vault and partial or complete absence of the cerebral hemispheres) are the most common NTDs observed.[14] These two conditions are also referred to as open neural tube defects to differentiate them from the rarer closed neural tube defects, in which skin covers the spinal abnormality.

The prevalence of NTDs varies widely among different populations, from 1 to 2 persons per 1000 live births, to as high as 6 persons per 1000 live births in northern Chinese populations. While the pathophysiologic basis for NTDs is complex and not well understood, it is known that folic acid supplementation can reduce the occurrence of NTDs by 60% to 70%. This recognition has led to the widespread recommendation for folic acid fortification of enriched cereal products and supplementation (400–800 mcg per day) prior to conception, as well as during the first 8 weeks of pregnancy.[13] Although women who have had an affected child have a risk of reoccurrence in subsequent children of approximately 2% to 5%, 95% of all NTDs occur in previously unaffected families.[15]

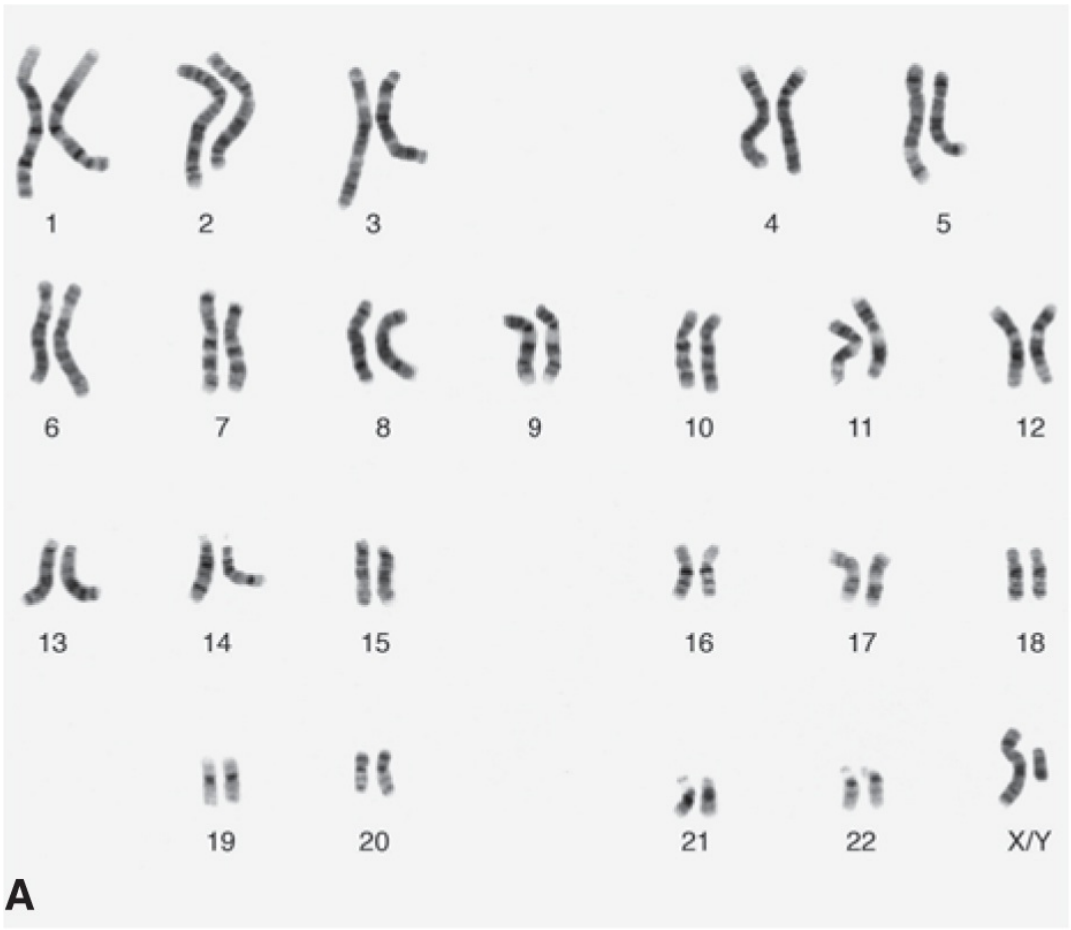
Individuals with spina bifida—the most common NTD—have varying degrees of physical and mental challenges, including hydrocephalus (which is independently associated with increased risk for physical and intellectual disability). The severity of disability increases with the severity of the defect. Approximately 35% of infants born with spina bifida do not survive to 10 years. Surgical repairs of the defect are the usual approach for improving prognosis.

Anencephaly has an extremely high level of mortality. The majority of infants with anencephaly are stillborn, and those who are born alive die soon after birth.[16]

Chromosomal Disorders

Chromosomal disorders are not the result of single-gene mutations, such as the autosomal dominant and recessive disorders, but rather reflect changes in the number or structure of the chromosomes.[2] Chromosomal abnormalities are relatively common, occurring in an estimated 1 in 150 live births and approximately 50% of first-trimester spontaneous abortions.[2] Such abnormalities frequently are associated with common phenotypic and physical characteristics, thereby aiding in their early identification, including during fetal ultrasound examination. However, chromosomal evaluation via a *karyotype* (a visual display of the full complement of an individual's chromosomes) or *chromosomal microarray analysis* provides the definitive diagnosis.

The most common numeric chromosomal aberrations are caused by *aneuploidy*—an addition or deletion of an entire chromosome in a normal set of 23 pairs of chromosomes. Aneuploidy most often occurs due to nondisjunction during cell division in the egg or sperm.[17] The resulting absence of one chromosome (monosomy) in a set is almost always lethal, so such a mutation is rarely observed in live-born children. In contrast, the addition of a chromosome (trisomy) leads to one of several disorders depending on which chromosome pair receives the extra genetic material. The most common trisomy is trisomy 21 (Down syndrome), which is caused by an extra chromosome added to the 21st pair of chromosomes. **Figures 20-5A** and **20-5B** contrast the 21st chromosomes in a normal female karyotype and one with trisomy 21. Phenotypic characteristics of individuals with trisomy 21 include specific facial features, such as small upturned eyes, small flat nose, small mouth with large tongue, and small ears (**Figure 20-6**). Intellectual and physical disabilities of varying severity are also observed. Congenital heart defects, obstructions of the gastrointestinal tract, and frequent respiratory infections are associated with trisomy 21. Trisomy 18 (Edwards syndrome) and trisomy 13 (Patau syndrome) are less frequently encountered aneuploidies, but both result in severe physical and intellectual disability in offspring, and are usually lethal during gestation or shortly after birth.[2,18]



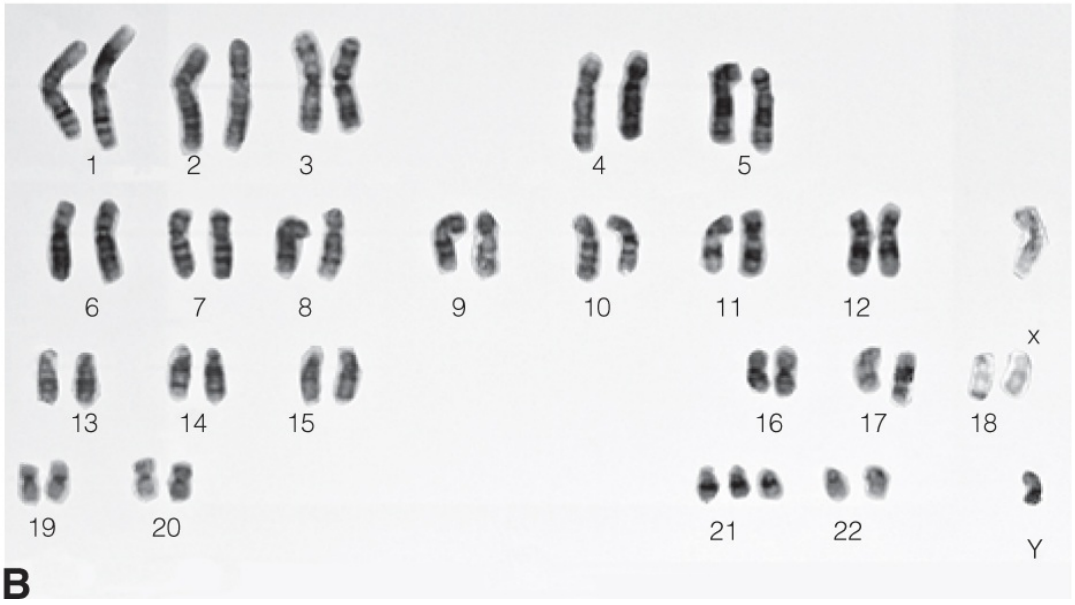


Figure 20-5 Karyotype. **A.** A full complement of 23 pairs of chromosomes in a female individual. **B.** An extra chromosome is observed in this female individual with trisomy 21 (Down syndrome).

A. Courtesy of Darryl Leja/National Human Genome Research Institute. B. © Jens Goepfert/Shutterstock



Figure 20-6 Infant with Down syndrome (trisomy 21) chromosomal disorder.

© iStockphoto/Thinkstock

Other common aneuploid disorders include sex chromosomal aberrations, such as Turner syndrome (XO), Klinefelter syndrome (XXY), trisomy X (XXX), and 47, XYY syndrome. Aneuploidy of sex chromosomes frequently results in less severe consequences than does autosomal aneuploidy, but the resulting conditions are associated with sex characteristic effects (including sterility, feminization, or virilization), and frequent minor physical changes specific to the disorder.[18] Many individuals who are mildly affected are not identified with a

chromosome abnormality for much of their lives unless health care is sought for a seemingly unrelated condition (e.g., infertility) and a karyotype is performed to identify the potential genetic etiology of the condition.[2,19,20] The gain of an entire haploid set of chromosome (e.g., 69, XXY) also occurs in a partial hydatiform mole.

Rearrangements, inversions, small deletions, or microdeletions of portions of a chromosome are considered structural abnormalities. They contribute to infrequently occurring genetic conditions, such as cri-du-chat syndrome, the result of a deletion of a portion of chromosome 5; Wolf-Hirschhorn syndrome, the result of a deletion of a portion of chromosome 4; Prader-Willi syndrome, a microdeletion of a portion of chromosome 15; and Williams syndrome, a microdeletion of a portion of chromosome 7.[21] Each abnormality has phenotypic characteristics specific to the condition.

Birth Defects, Incidence, and Risk Identification

Genetic disorders and chromosomal disorders are not rare occurrences. An individual's risk for a genetic disorder is based on several factors, including age, gender, and ethnic background. For example, cystic fibrosis has a prevalence of 1 case per 2500 persons among whites of Northern European descent, while sickle cell disease has a prevalence of 1 case per 400 to 600 persons among blacks of African descent. Another example of a factor affecting the risk for chromosomal disorders is maternal age: Women have an increased risk of having a child with trisomy 21 as the woman ages.[22] **Table 20-3** and **Table 20-4** list several conditions and diseases with a genetic component, along with their prevalence in specific populations. Understanding particular risks among specific populations and groups can prompt the midwife to probe further when obtaining a medical and family history, provide accurate patient education, recommend specific testing to women and families, or make a referral to a genetic counselor, as appropriate.

Table 20-3	Selected Chromosomal and Genetic Disorders with Approximate Incidence Rates
Condition/Disease	**Approximate Incidence**
Chromosome Abnormalities	
Klinefelter syndrome	1/1000 males
Trisomy 21 (Down syndrome)	1/700 to 1/1000
Trisomy 18 (Edwards syndrome)	1/6000
Trisomy 13 (Patau syndrome)	1/10,000
Turner syndrome	1/2500 to 1/10,000 females
Single-Gene (Mendelian) Disorders	
Achondroplasia (dwarfism)	1/25,000
Cystic fibrosis	1/2000 to 1/4000 (persons of European descent)
Duchenne muscular dystrophy	1/3500 males
Hemophilia A	1/5000 to 1/10,000 males
Huntington disease	1/20,000 (persons of European descent)
Neurofibromatosis	1/3000 to 1/5000
Sickle cell disease	1/400 to 1/600 African Americans; as many as 1/50 in persons from Central Africa
Tay–Sachs disease	1/3000 in persons of Ashkenazi Jewish descent
Thalassemia	1/50 to 1/100 (South Asian and circum-Mediterranean populations)

Modified with permission from Jorde L, Carey J, Bamshad M. Background and history. In: Jorde L, Carey J, Bamshad M, eds. *Medical Genetics*. 5th ed. Philadelphia, PA: Elsevier; 2016:1-5.[1]

Table 20-4	Selected Multifactorial Disorders with Approximate Incidence Rates

Condition/Disease	Approximate Incidence
Congenital Malformations	
Cleft lip with or without cleft palate	1/500 to 1/1000
Club foot (congenital talipes equinovarus)	1/1000
Congenital heart defects	1/200 to 1/500
Neural tube defects (spina bifida, anencephaly)	1/200 to 1/1000
Pyloric stenosis	1/300
Adult Diseases	
Alcoholism	1/10 to 1/20
Alzheimer disease	1/10 persons in the United States older than 65
Bipolar affective disorder	1/100 to 1/200
Cancer (all types)	1/3
Diabetes (types 1 and 2)	1/10
Heart disease or stroke	1/3 to 1/5
Schizophrenia	1/100

Modified with permission from Jorde L, Carey J, Bamshad M. Background and history. In: Jorde L, Carey J, Bamshad M, eds. *Medical Genetics*. 5th ed. Philadelphia, PA: Elsevier; 2016:1-5.[1]

Age

Women who are older than 35 years have a well-documented increase in the risk for having a fetus with a chromosomal abnormality, particularly for aneuploidy such as trisomy 21 (Down syndrome).[23] The graph in **Figure 20-7** demonstrates the escalating risk of a pregnancy being affected by a chromosomal anomaly as maternal age increases. By the maternal age of 45 years, the risk for any chromosomal disorder is approximately 5.3% (the majority being trisomy 21), compared to approximately 0.25% for women who are younger than age 30 years. Presented another way, **Table 20-5** shows that 1 in 30 infants born to women age 45 years will have trisomy 21, compared to 1 in 1250 infants born to women age 25 years.[22] The risk for any chromosomal disorder for infants born to women age 45 years is 1 in 20.

Table 20-5 **Maternal Age and Risk of Chromosomal Disorders (Live Births)**

Maternal Age (years)	Risk for Trisomy 21	Risk for Any Chromosomal Disorder
20	1 in 1667	1 in 526
25	1 in 1250	1 in 476
30	1 in 952	1 in 384
35	1 in 385	1 in 204
40	1 in 106	1 in 65
45	1 in 30	1 in 20
49	1 in 11	1 in 7

Data from Arnold KM, Self ZB. Genetic screening and counseling: family medicine obstetrics. *Prim Care.* 2012;39:55-70.[22]

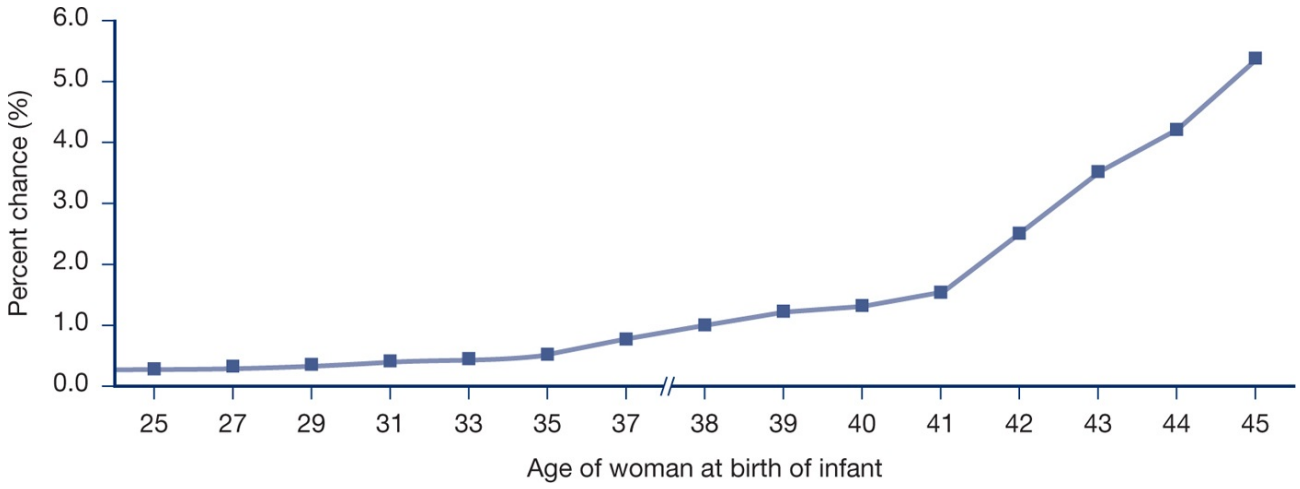


Figure 20-7 Maternal age and risk of chromosomal abnormality.

Data from Hook EB. Rates of chromosomal abnormalities. *Obstet Gynecol*. 1981;58(3):282-285.[23]

Although a few studies have shown a very slight increase in the risk of genetic disorders for children born to older men (primarily autosomal dominant disorders such as achondroplasia and neurofibromatosis), most research has not revealed that older fatherhood is associated with an increased risk for chromosomal anomalies.[24,25] Furthermore, unlike the risk associated with advanced maternal age, no clear consensus exists for when a man is considered to be of advanced paternal age. A commonly used criterion is age 40 years, but there are no specific recommendations for couples wherein the biologic father is of advanced paternal age.

Maternal Disorders

Exposure to teratogens may be a risk factor for birth defects depending on the level and type of exposure (i.e., duration, strength, potency).[22] A *teratogen* is a drug, virus, chemical, or other substance that can interfere with normal embryonic or fetal development and cause malformations. A list of medications that are known or suspected to be teratogens can be found in the *Pharmacotherapeutics* chapter. Selected maternal conditions that are known or highly suspected contributors to birth defects are listed in **Table 20-6**.[26]

Table 20-6 Selected Maternal Conditions That Are Teratogenic

Teratogen	Associated Defects	Percentage of Pregnancies Affected
Maternal Infections		
Cytomegalovirus	Mental retardation, microcephaly	10–15%
Rubella	Deafness, cataracts, heart defects, mental retardation	Up to 85%

Syphilis (untreated)	Abnormal teeth and bones, mental retardation	Not established
Toxoplasmosis	Hydrocephaly, blindness, mental retardation	5–6%
Varicella	Limb reduction defects, skin scarring, muscle atrophy, chorioretinitis	1%
Zika	Microcephaly, decreased brain tissue, joints with limited range of motion, restrictive muscle tone, damage to back of the eye	6% overall 11% for exposure during first trimester
Maternal Conditions		
Active alcoholism	Miscarriage, fetal alcohol syndrome (minor facial changes, heart defects), IUGR, developmental delay	Up to 50%
Diabetes mellitus	Heart defects, microcephaly, neural tube defects, skeletal defects, and defects in the urinary, reproductive, and digestive systems	HbA1c < 7.9%: 3.2% HbA1c 8.0–9.9%: 8.1% HbA1c > 10%: 23.5%
Hyperthermia (fever); early pregnancy only	Neural tube defects, heart and abdominal wall defects, oral cleft	Not established
Seizure disorder (treated)	Cleft lip with or without cleft palate, heart defects	6–8%
Systemic lupus erythematosus (uncontrolled)	Miscarriage, stillbirth, congenital heart block, IUGR, prematurity	Not established

Abbreviations: HbA1c, hemoglobin A1c; IUGR, intrauterine growth restriction.

Based on Teratology Society: Organization of Teratology Information Specialists (OTIS). MotherToBaby fact sheets. 2017. Available at: https://www.teratology.org/OTIS_fact_Sheets.asp. Accessed February 21, 2017.[26]

Environmental Teratogens

A number of environmental chemicals such as mercury, polychlorinated biphenyls [PCBs], some paints, and phthalates have been linked to teratogenic or fetotoxic effects.[27] Most of these agents have suspected adverse effects on fertility, spontaneous abortion, and impaired neurodevelopment. Although evidence that firmly establishes some of these compounds as teratogens is varied, these associations are the subject of current research. Adverse reproductive outcomes associated with specific environmental toxins and pollutants are discussed in more detail in the *Preconception Care Visit* appendix of the *Health Promotion Across the Lifespan* chapter.

Teratogens may cause genetic mutations, but it is thought that an alteration in gene expression during critical embryonic and fetal developmental periods is the more likely mechanism contributing to genetic disorders that are secondary to teratogen exposure.[28] This is an important distinction because epigenetic regulation of gene expression is an increasingly documented and plausible mechanism that may explain some of the link between environmental exposures and birth.[28] For example, epigenetic regulation of gene expression may be the mechanism underlying the link between chronic hyperglycemia, found among women with poorly managed diabetes (gestational or otherwise), and an increased risk of birth defects, such as heart defects and NTDs.[29,30] Moreover, epigenetic modifications may endure and

confer risk for more than one generation.[31]

The major concern is for exposures to women during pregnancy, particularly during the first trimester. As yet, little to no evidence has been published suggesting that paternal exposure is associated with increased occurrence of birth defects, although there may be some association with adverse health outcomes during childhood.[32]

Family History and Pedigree Evaluation as Risk Assessment Tools

One of the most effective, low-cost approaches to genetics risk assessment is to obtain a thorough personal health and family history that includes evaluation of a three-generation pedigree. A personal and family history is a "free, well-proven, personalized genomic tool that . . . can serve as the cornerstone for individualized disease prevention."[33] Many clues that indicate the need for further evaluation, testing, or referral to a genetic counselor can be found by obtaining a thorough family history (**Table 20-7**).[34] Furthermore, a number of consumer-completed family genetics history tools (see the Resources section at the end of this chapter) are available, both in print and online, to facilitate collection of personal and family histories. Such participation tools can be completed by an individual prior to a prenatal care appointment with the midwife and can be an effective and efficient way for identifying genetic risk.

Table 20-7 Personal and Family History That May Warrant Genetic Testing and/or Referral to Genetic Counseling

Maternal age
High-risk ethnic heritage
Consanguinity (blood relationship of parents)
Family history (maternal and/or paternal) of a known or suspected genetic condition
Multiple affected family members (maternal and/or paternal) with the same or related disorders
Any major malformations (e.g., heart, kidney, brain) or other birth defects occurring in mother, father, grandparents, offspring, or close relatives (brothers/sisters)
Congenital blindness or deafness in family members
Extremely tall or short stature of mother, father, or their relatives
Developmental delays or intellectual disability occurring in mother, father, offspring, or close relatives
Recurrent pregnancy losses (two or more) in the woman or first-degree family members
Environmental exposure to known or suspected teratogens
Infertility or premature ovarian failure

Based on The New York – Mid-Atlantic Guide for Patients and Health Professionals. Indications for a genetic referral. In: *Understanding Genetics: A New York, Mid-Atlantic Guide for Patients and Health Professionals*. Washington, DC: Genetic Alliance; 2009. Available at: https://www.ncbi.nlm.nih.gov/books/NBK115563/pdf/Bookshelf_NBK115563.pdf. Accessed June 26, 2017.[34]

Constructing and evaluating a three-generation pedigree for genetic risk is a basic skill for all healthcare professionals, including midwives.[35] Instructions for how to obtain a three-generation pedigree can be found in **Appendix 20-A**.

Screening Tests Versus Diagnostic Tests

Diagnostic tests provide a definitive answer to whether an individual has a particular disorder. In contrast, screening tests simply separate those persons who "might" from those persons who "probably don't" have the specific condition being tested. Screening tests are *not* diagnostic. Screening results usually aid in determining the level of risk for a specific disorder, and they identify individuals who might be advised to undergo further, definitive diagnostic testing.

For example, the results of a maternal serum screening test for trisomy 21 during the first trimester are not simply reported as "normal" or "abnormal." Instead, the results will indicate the chance (e.g., 1 in 40, 1 in 400, 1 in 4000) of having a fetus with trisomy 21. This chance is based on the level of pregnancy-associated plasma protein A (PAPP-A) and human chorionic gonadotropin (hCG) in maternal serum in conjunction with nuchal translucency measurement identified on ultrasound performed at a specific gestational age. Such screening test results give the woman the information necessary to help decide whether diagnostic testing will be chosen as the next step.

Screening tests have six critical components: (1) sensitivity, (2) specificity, (3) false-positive rate, (4) false-negative rate, (5) positive predictive value, and (6) negative predictive value (**Table 20-8**). The *sensitivity* refers to the number of individuals with the disorder who have a screening test that is positive for that disorder. *Specificity* is the number of individuals without the disorder who have a negative screening test. Again, using first-trimester maternal serum screening (without adding nuchal translucency ultrasound) as an example, such screening has the demonstrated ability to detect approximately 70% of the fetuses who have trisomy 21.[36] Thus, approximately 30% of fetuses with trisomy 21 will not be detected with this screening test; this is the *false-negative rate*. In contrast, the *false-positive rate* reflects the chance of a positive test result indicating a high likelihood for trisomy 21 when a healthy fetus is actually present. The false-positive rate for first-trimester screening for trisomy 21 is approximately 5%,[37] and is a significant contributor to unintended parental anxiety during the prenatal period and potentially unnecessary interventions.

Table 20-8 Sensitivity, Specificity, Positive Predictive Value, and Negative Predictive Value

Test Results	Disease Present	No Disease	
Screen test positive	A (true positive)	B (false positive)	A ÷ A and B Positive predictive value
Screen test negative	C (false negative)	D (true negative)	D ÷ C and D Negative predictive value
	A ÷ A and C Sensitivity	D ÷ B and D Specificity	

Many factors affect test results, including gestational age, maternal age, presence of multifetal gestation, certain medical conditions such as diabetes, and other coexisting congenital or genetic disorders.[38] In addition, the prevalence of a disorder varies for different

groups of women (e.g., based on ethnicity or geographical location). These differences must be taken into account to accurately interpret test results and contribute to determining the positive predictive value and negative predictive value, as illustrated in **Figure 20-8**. Continuing the example of first-trimester screening for Down syndrome, the *positive predictive value* is the proportion of all positive tests that truly indicate a baby *with* trisomy 21; it is calculated as the number of women with a positive test divided by the total number of women in the population tested who had a positive screening test result. In contrast, the *negative predictive value* is the proportion of all negative tests that truly indicate a baby without trisomy 21, calculated as the number of persons without the disease who had negative results divided by the total number of women tested who had negative results.

Chart A: Sensitivity (87%), specificity (95%), and predictive value of first-trimester screening (nuchal translucency and maternal serum) for trisomy 21 when the prevalence rate is 1 per 1250, as it is among women age 25 years[a]

	Trisomy 21 Present	Trisomy 21 Absent	Interpretation (Predictive Value)
Positive test result	87 will have a true-positive test	6235 will have a false-positive test	A 25-year-old woman with a positive test for trisomy 21 has a 1.4% chance of actually having a fetus with trisomy 21 (out of 6322 positive tests, 87 fetus had trisomy 21 and 6235 tests were false alarms). This is the positive predictive value.
Negative test result	13 will have a false-negative test	118,665 will have a true-negative test	A woman with a negative test result has a 99.9% chance of *not* having a fetus with trisomy 21 (out of 75,918 negative tests, 13 fetuses actually had trisomy 21). This is the negative predictive value.
	100 fetuses affected	124,900 not affected	Out of 125,000 fetuses in women who are 25 years old, 100 will have trisomy 21 and 124,900 will not have a fetus with trisomy 21.

Chart B: Sensitivity (87%), specificity (95%), and predictive value of first-trimester screening (nuchal translucency and maternal serum) for trisomy 21 when the prevalence rate is 1 per 30, as it is among women age 45 years[a]

	Trisomy 21 Present	Trisomy 21 Absent	Interpretation (Predictive Value)
Positive test result	87 will have a true-positive test	145 will have a false-positive test	A 45-year-old woman with a positive test for trisomy 21 has a 37.5% chance of actually having a baby with trisomy 21 (out of 232 positive tests, 87 babies had trisomy 21 and 145 tests were false alarms). This is the positive predictive value.
Negative test result	13 will have a false-negative test	2755 will have a true-negative test	A woman with a negative test result has a 99.3% chance of *not* having a fetus with trisomy 21 (out of 2768 negative tests, 13 babies actually had trisomy 21). This is the negative predictive value.
	100 babies affected	2900 babies not affected	Out of 3000 babies born to 45-year-old mothers, 100 will have trisomy 21 and 2900 will not have a fetus with this chromosomal abnormality.

Figure 20-8 The impact of prevalence on the predictive value and interpretation of first-trimester

screening for trisomy 21 among women of differing age.[a]

[a] Test performance (specificity and sensitivity) is the same in both charts. However, the positive predictive value and the negative predictive value are different in the two charts because the prevalence rate for trisomy 21 is different for women who are 25 years old (chart A) and women who are 45 years old (chart B). The interpretation is therefore different for women of different ages as represented in each of these charts.

This type of multifactorial input into test results is also illustrated with the use of the maternal serum alpha-fetoprotein (MSAFP) or alpha-fetoprotein (AFP) test. AFP is a protein made by the fetal liver and found in the amniotic fluid. It passes through the placenta and circulates into the maternal blood, thereby enabling assessment of AFP through maternal serum sampling. MSAFP is a screening test for NTDs, but also is used to identify risk for trisomy 21. High levels of MSAFP are associated with increased risk of NTDs, whereas lower levels are associated with elevated risk for trisomy 21.[39] In this case, one of the additional factors influencing the interpretation of results is an accurate estimation of gestational age. The MSAFP test has different normative values at different gestational ages. Thus, if a woman has the test done at 15 weeks' gestation, it will be interpreted based on 15-week norms. However, if the gestational age has not been accurately determined and the pregnancy is actually at 13 gestational weeks, the test results will appear abnormal because they have been interpreted incorrectly. Similarly, there are different normative values for women who have a multifetal gestation. This situation has prompted clinicians to use multiple biochemical markers as screening tests, rather than relying on a single marker, such as MSAFP.

Factors That Influence Women's Decision Making

Women are faced with many difficult decisions in healthcare settings, and prenatal genetic screening and testing are not exceptions. Many factors, such as individual, family, religious and cultural values, will play a role in how a woman perceives screening and diagnostic testing for genetic defects. Midwives can ease this process by engaging in evidence-based, shared decision making when offering prenatal testing, interpreting test results, and discussing options. Women and their families should have their questions answered adequately and candidly, but with compassion and respect for individual values and wishes.

Prenatal tests can screen for some structural abnormalities, genetic disorders, and chromosomal abnormalities. The aim of prenatal screening and diagnostic testing is to provide families with the information necessary to make well-informed reproductive health decisions. The clinical implications of test results can be complicated, and sharing information with women both before and after tests are performed is a necessary skill.

Pre- and Post-Test Counseling When Screening for Genetic and Chromosomal Disorders

Genetic and genomic health care is changing rapidly as new technologies are incorporated into clinical practice. The number of tests and algorithms promoted as part of prenatal testing continues to increase, and best practices for incorporating new tests are being studied. A standard algorithm for prenatal testing based on professional guidelines and the tests commonly available today is presented in the *Prenatal Care* chapter. This chapter reviews the process of counseling women about these tests and the characteristics of the screening and diagnostic tests that are currently in use.

Pre- and post-test counseling is recommended for all women who have prenatal screening tests for genetic disorders. Women who might have an elevated risk for a genetic or chromosomal disorder (as indicated in **Table 20-7**) and any women who want more detailed information should be referred for genetic counseling. Most healthcare settings offer genetic counseling to any woman who has a positive screening test.

Table 20-9 provides an overview of the questions that women most often ask and key topics to address in pretest counseling.[40-47] The most important question for a woman is, "What will I do with the information?" This question will need to be answered by the woman for herself with support from her significant others and her midwife. Answering this question can assist her to make the ultimate decision as to whether she will undergo screening tests, diagnostic tests, or both.[45] A woman can often arrive at an answer by addressing additional, very personal questions: "Would I terminate this pregnancy if I know my baby is affected with (fill in the blank)?" "Do I want to know that my baby is affected before the baby is born?" "Would testing simply make me more anxious, rather than less anxious?" "Am I willing to accept the increased risk of miscarriage associated with a diagnostic test such as chorionic villus sampling or amniocentesis?" "Am I strongly opposed to pregnancy termination even if I have a baby affected with (fill in the blank)?" "Why should I have screening or diagnostic tests if I am fine with any outcome the baby might have?" It often helps women to self-identify their stance when

the midwife offers statements that begin with “Some women choose to have these tests because . . .” and “Some women choose not to have these tests because . . . .”

Table 20-9 Key Topics for Pretest Counseling

Questions Women Ask	Essential Information for Pretest Counseling
Why is this test offered?	Review the conditions being tested for, including common phenotype and variability in phenotype
	Type of testing offered (screening, carrier screening, diagnostic test)
	Alternative testing options, including the risks, benefits, and limitations of each alternative
How are the results communicated?	Review how test results are shared with the woman and timing for getting results
What will I do with the information?	Possible results of testing: positive, negative, unclear (inconclusive), unexpected
What if there is a positive result?	Availability of genetic counseling to provide additional information and assist decision making
	Review that genetic counseling will address diagnosis of the condition, options for the woman if a disorder is diagnosed, and treatments for the condition
How much does the test cost and will insurance cover the cost?	Discuss cost of testing and insurance coverage
	Address the effect on health insurance if a disorder is diagnosed
When does the test have to be done?	Review timing for performing the test
Who will know about the results?	Review patient confidentiality and discuss implications of sharing information with a partner and family

Based on Fonda Allen J, Stoll K, Bernhardt BA. Pre- and post-test genetic counseling for chromosomal and Mendelian disorders. *Semin Perinatol.* 2016;40(1):44-55.[45]

Tests for Genetic and Chromosomal Disorders

The number and types of tests that screen or diagnose genetic or chromosomal disorders have increased rapidly in the last several years. These tests can be applied to carrier screening, preimplantation testing, prenatal screening, diagnostic testing, or newborn screening. In general, these tests can be classified into one of three categories:

- *Biochemical tests* evalute the proteins or enzymes produced by the genetic code, but not the genes themselves. Biochemical tests can be performed on maternal or fetal cells and may be used for screening or diagnosis purposes.
- *Cytogenic testing* examines the chromosomes to identify structrual abnormalities. The traditional karyotype cultures cells and stains the chromosomes to detect changes in structure. Fluorescent in situ hybridization (FISH) targets specific chromosomes to detect aneuploidy or smaller abnormalities such as deletions.
- *Molecular testing* is a direct analysis of DNA that detects small DNA mutations. Molecular DNA testing may use polymerase chain reaction (PCR)–based assays to amplify the DNA present. Chromosomal microarray analysis is a molecular DNA test applied to fetal cells to detect aberrations in chromosomal structure that may not be visible on a normal karyotype.

Carrier Screening

The purpose of carrier screening is to detect couples at risk for having a child with a genetic condition that is not evident in either of the parents. Carrier screening for autosomal dominant or autosomal recessive disorders is ideally performed preconceptionally, but often is first addressed during pregnancy.

Carrier screening can be performed for one disorder or multiple disorders at one time.[47] Specific carrier screening tests have traditionally been offered to individuals from racial and ethnic populations that have an increased prevalence of the disorder, a form of screening referred to as *ethnic-specific screening*. An ethnic-based screening strategy has multiple problems. For example, in today's multiracial society, it may be difficult to determine ethnicity. Furthermore, ethnicity does not identify all persons with an elevated risk and therefore, this screening strategy can miss many persons who would benefit from such testing. *Panethnic screening* refers to a strategy of offering carrier screening to all individuals regardless or race or ethnicity. Panethnic screening is an acceptable strategy.

New sequencing technologies have created the ability to screen for large numbers of conditions simultaneously. Nevertheless, such *expanded carrier screening* also has several unresolved problems. Many of the conditions covered by expanded carrier screening panels vary greatly in the expressed phenotype or disability, so counseling women about results can be quite complex. In addition, some of the expanded carrier screening panels screen for conditions that are rare and for which diagnostic tests are not well established. Furthermore, different commercially marketed panels screen for different disorders. Current professional association guidelines recommend ethnic-specific, panethnic, or expanded carrier screening. Factors such as severity of condition, prevalence carrier frequency, detection rates, and residual risk following screening results as described in **Table 20-10** contribute to the choice of screening strategy.[47]

Table 20-10 Carrier Screening

Disorder	Indications	Clinical Considerations
Ashkenazi Jewish population, prevalent conditions: Tay–Sachs disease, cystic fibrosis, Canavan disease, familial dysautonomia, Niemann–Pick (type A), Bloom syndrome, Fanconi anemia group C, mucolipidosis IV, Gaucher disease	Ashkenazi Jewish population	
Cystic fibrosis	Offer screening to all women of reproductive age	
Hemoglobinopathies	African ancestry Mediterranean ancestry Southeast Asian ancestry	African ancestry: Perform hemoglobin electrophoresis. Mediterranean or Southeast Asian ancestry: If anemic and mean corpuscular volume < 80 fL, evaluate for iron deficiency. Perform hemoglobin electrophoresis if iron studies normal.

		Southeast Asian ancestry: If hemoglobin electrophoresis is normal, perform molecular testing for alpha-thalassemia.
Fragile X syndrome	Individuals with a family history of intellectual disability or unexplained developmental delay, autism, or primary ovarian insufficiency	Offer genetic counseling for persons with a family history.
Spinal muscular atrophy	Offer screening to persons with a family history of spinal muscular atrophy[a]	Offer genetic counseling for persons with a family history.
Tay–Sachs disease	Ashkenazi Jewish ancestry French Canadian ancestry Cajun ancestry	

Based on Edwards JG, Feldman G, Goldberg J, et al. Expanded carrier screening in reproductive medicine: points to consider: a joint statement of the American College of Medical Genetics and Genomics, American College of Obstetricians and Gynecologists, National Society of Genetic Counselors, Perinatal Quality Foundation, and Society for Maternal–Fetal Medicine. *Obstet Gynecol*. 2015;125(3):653-662.[47]

Screening Tests

There are several time points during pregnancy at which specific screening and diagnostic tests can be performed so that the most accurate results can be obtained. The decision whether to engage in any testing is always the individual woman's choice. The midwife's role is to provide as much information as needed to facilitate an individual's decision making about what is personally best, as well as best for the family. **Table 20-11** provides a comprehensive list of both screening and diagnostic tests for the most common congenital disorders, including the appropriate timing for tests that are regularly offered in the United States, as well as some that are expected to be adopted soon.[48]

Table 20-11 **Prenatal Screening and Diagnostic Tests Currently Available**

Gestational Age When Test Is Performed	Test	Testing Available for These Conditions			
		Aneuploidy	NTDs	Genetic Mutation	Congenital Anomalies
First-Trimester Screening					
10–14 weeks	Ultrasound: nuchal translucency (thickness)	✓			✓
10–14 weeks	Maternal serum: PAPP-A and hCG	✓			
10+ weeks	cfDNA (maternal serum)[a]	✓			
Second-Trimester Screening					
15–23 weeks (optimal at 18 weeks)	Ultrasound: anatomic examination	✓	✓		✓
15–18 weeks	Maternal serum: multiple marker (quad): AFP, hCG, uE3, and inhibin-A	✓	✓		
Integrated screening	Combines first- and second-trimester screening as a comprehensive risk assessment	✓	✓		✓
Sequential	First-trimester screening, followed by second-trimester screening, as desired, based on first-trimester results	✓	✓		✓
Anytime	cfDNA (maternal serum)[a]	✓			
First-Trimester Diagnostic Tests					
10–14 weeks	CVS For karyotype and/or direct DNA[b] and cytogenetic (chromosomal)[c] testing	✓		✓	
Second-Trimester Diagnostic Tests					
15–22 weeks	Amniocentesis for karyotype and/or direct DNA[b] and cytogenetic[c] testing, and amniotic fluid AFP level	✓	✓	✓	
Anytime Prepregnancy or During Pregnancy (for parents), Newborn, and Children					
Direct diagnostic testing for an individual (i.e., DNA[b] for autosomal recessive carrier status, cytogenetic[c] testing)	Blood, saliva, and skin cells from parents prior to or during pregnancy, or in the newborn or siblings	✓		✓	

Abbreviations: AFP, alpha-fetoprotein; cfDNA, cell-free DNA; CVS, chorionic villus sampling; DNA, deoxyribonucleic acid; hCG, human chorionic gonadotropin; NTDs, neural tube defects; PAPP-A, pregnancy-associated plasma protein A; uE3, unconjugated estriol.

[a] cfDNA, often referred to as noninvasive prenatal testing (NIPT), is the most recently developed screening test for trisomy 21, 18, and 13, sex chromosome aneuploidy, and some microdeletions.

[b] DNA testing for genetic deletions, insertions, rearrangements, duplications, and other mutations can be accomplished using laboratory techniques, such as polymerase chain reaction (PCR) DNA amplification, hybridization of labeled probes to DNA, and microarray analysis, among others.

[c] Cytogenetic (chromosomal) structural aberrations, including microdeletions and aneuploidy, can be identified by karyotyping, chromosomal banding, fluorescence in situ hybridization (FISH), chromosomal microarray, and comparative genomic hybridization (CGH).

Historically, women who were younger than 35 years were offered screening tests and women older than 35 years were offered screening or diagnostic tests based on their higher baseline risk for trisomy 21. Currently, it is recommended that both screening and diagnostic testing be offered to all pregnant women, regardless of age or risk factors present.[49] This recommendation was made in light of the low-risk, reliable, noninvasive methods currently available, as well as the belief that all women should have a choice in testing—not just women with risk factors.

First-Trimester Screening for Aneuploidy

Screening for aneuploidy—particularly trisomy 21, 18, and 13—can be done in the first trimester and is best performed between 10 and 14 weeks' gestation. **Table 20-12** reviews the detection and false-positive rates of currently available screening and diagnostic tests for aneuploidy.[48] Risk for aneuploidy can be determined by measuring maternal serum levels of PAPP-A and hCG (expressed as multiples of the median) in relation to maternal weight and age, and gestational age. Generally, an increased risk of trisomy 21 is associated with lower PAPP-A levels and higher hCG levels.[39]

Table 20-12 **Detection and False-Positive Rates for Currently Available Prenatal Tests for Aneuploidy**[a]

Test	Detection Rate[b] % (trisomy type)	False-Positive Rate[c] % (trisomy type)
First-Trimester Screening		
NT alone	68 (trisomy 21)	5 (trisomy 21)
	68 (trisomy 18)	0.5 (trisomy 18)
	72 (trisomy 13)	
Maternal serum markers alone	67 (trisomy 21)	5 (trisomy 21)
	80 (trisomy 18)	0.5 (trisomy 13 and 18)
	59 (trisomy 13)	
Combined NT measurement and maternal serum markers	90 (trisomy 21)	5 (trisomy 21)
	97 (trisomy 18)	0.5 (trisomy 13 and 18)
	84 (trisomy 13)	
Second-Trimester Screening		
Maternal serum markers alone (quad)	80 (trisomy 21)	5 (trisomy 21)
	70 (trisomy 18)	0.2 (trisomy 13 and 18)
	19 (trisomy 13)	2–5 (ONTD)
	75–90 (ONTD)	
	95 (anencephaly)	
Anatomic US alone	73 (trisomy 21)	4 (trisomy 21)
	93-100 (trisomy 18)	4 (trisomy 18)
	90-100 (trisomy 13)	0.5 (trisomy 13)
Maternal quad screen and US	83 (trisomy 21)	5 (trisomy 21)
	100 (trisomy 18)	0.4 (trisomy 18)
	100 (trisomy 13)	0.5 (trisomy 13)
Integrated Screening		
First- and second-trimester maternal serum *without* NT	86 (trisomy 21)	2 (trisomy 21)
	86 (trisomy 18)	0.5 (trisomy 13 and 18)
	49 (trisomy 13)	
First- and second-trimester maternal serum *with* NT	95 (trisomy 21)	4 (trisomy 21)
	92 (trisomy 18)	0.5 (trisomy 13 and 18)
	72 (trisomy 13)	
CVS	98 (aneuploidy)	< 0.04
Amniocentesis	99.5 (aneuploidy)	< 0.04
	96–99 (ONTD)	2 (ONTD)
Fetal Cell-Free DNA[d]	99 (trisomy 18 and 21)	0.3–1 (overall)
	92 (trisomy 13)	
Direct DNA Testing	99.9	< 0.04
Saliva, blood, placental, or skin cell sample for identification of genetic mutation and carrier status		

Abbreviations: CVS, chorionic villus sampling; DNA, deoxyribonucleic acid; NT, nuchal translucency (thickness); ONTD, open neural tube defects; US, ultrasound.

[a] Detection and false-positive rates can vary depending on population risk and the risk cut-off used in testing.
[b] Detection rate reflects test sensitivity—that is, the "accuracy" or ability of a test to correctly identify those who have a disorder.
[c] False-positive rate reflects test specificity—that is, the ability of a test to correctly identify those who do not have a disorder.
[d] Fetal cell-free DNA, frequently referred to as noninvasive prenatal testing (NIPT).

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Increased risk for aneuploidy can also be calculated during ultrasound measurement of nuchal translucency between 10 and 14 weeks' gestation. More than 3 mm of fluid retention in the nuchal fold is associated with an increased risk of trisomy 21, other aneuploidies, and perhaps other defects, such as heart defects, diaphragmatic hernias, skeletal dysplasia, and a variety of genetic syndromes.[38,50] Absence of the fetal nasal bone is also associated with trisomy 21; thus, some facilities include ultrasound assessment of the nasal bone.[39]

Fetal cell-free DNA (cfDNA) testing, often referred to as noninvasive prenatal testing

(NIPT), is a recent screening option for identification of trisomies 13, 18, and 21, and sex chromosome aneuploidy. This test has become widely available since its introduction in 2011.[51,52] Testing of cfDNA involves collection of maternal blood any time after 10 weeks' gestation; this blood contains small fragments of circulating cell-free fetal DNA. The test amplifies the fetal DNA and makes it possible to identify the extra genetic material that exists when aneuploidy is present in the fetus.[45] Some laboratories include testing for a few chromosomal microdeletions, but the performance of these tests is of concern.[53] Because cfDNA is a screening test rather than a diagnostic test, it is recommended that any positive result be followed with CVS or amniocentesis to confirm diagnosis.[53]

Although the trisomy 21 detection rate with cfDNA (99%) nearly matches that of CVS (98%) and amniocentesis (99.5%), the positive predictive value (PPV) is important to note. For example, a 25-year-old woman with a positive cfDNA test has a 33% chance of having a fetus with trisomy 21, whereas a 40-year old woman with a positive cfDNA test has an 87% chance of having a fetus with trisomy 21. The PPV is much lower in cfDNA testing for trisomy 13 and 18. Because of this wide variance in PPV, the American College of Obstetricians and Gynecologists (ACOG) does not currently recommend routinely offering cfDNA testing to pregnant women considered at low risk for aneuploidy.[53] Nevertheless, many other sources advocate that all women, regardless of risk status, should have this option among those already available to them, such as first- and second-trimester maternal serum markers and ultrasound, as well as invasive chorionic villus sampling (CVS) and amniocentesis.[54] Some predict that, given the rapidly advancing technology, cfDNA will be used as a noninvasive diagnostic test in the not too distant future, and will likely include full genomic testing of the embryo/fetus for genetic mutations and all chromosomal abnormalities.[54]

Second-Trimester Testing

Screening for aneuploidy and NTDs in the second trimester currently includes measurement of several markers in maternal serum. Commonly referred to as a maternal serum, multiple marker, or quad screen, this test includes the measurement of maternal serum levels of alpha-fetoprotein, uE3 (unconjugated estriol), human chorionic gonadotropin, and inhibin-A. The last three of these markers are hormones produced by the placenta and secreted into the maternal circulation. In contrast, AFP is a protein made by the fetus. The MSAFP test has been in use longer than the other tests included in the quad screen. Fetal cell-free DNA testing also can be conducted at any time during the second trimester.

Screening options over the past 20 years have moved rapidly from a single serum marker test (MSAFP) to the addition of other markers as multiple markers were recognized as more effective in detecting abnormalities; they also decrease the risk of false-negative results compared to a single marker.[55]

Integrated, Combined, or Sequential Prenatal Testing for Aneuploidy

First- and second-trimester screening can be combined (ultrasound and maternal serum markers) in an effort to improve the ability to detect trisomy 21 and other trisomies.[33,48]

Results can be calculated and returned to the pregnant woman after all testing has been completed (i.e., integrated/combined), or after each trimester's results are known (i.e., sequentially). Calculation of the risk that the fetus has trisomy 21 after all test results are known, however, would preclude the woman's choice to have a CVS, if diagnostic testing is indicated. In contrast, the sequential screen calculates risk based on first-trimester screening tests and returns the results to the woman before proceeding with further screening tests. One advantage of a sequential screen is that a woman and her prenatal care provider can decide whether to continue with the second-trimester screening or to proceed with diagnostic testing (CVS or amniocentesis) if the first-trimester results indicate high risk for chromosomal abnormalities.[39,56]

Diagnostic Tests

Diagnostic procedures for aneuploidy and direct DNA genetic testing include chorionic villus sampling and amniocentesis. The screening and diagnostic tests performed on fetal cells following CVS or amniocentesis are briefly described in **Table 20-13**.

Table 20-13 Screening and Diagnostic Tests Performed on Fetal Cells

Test	Description	Results	Clinical Considerations
Karyotype	Cytogenic test Metaphase analysis of cultured cells	All aneuploidies and chromosome abnormalities that are larger than 5–10 megabases	Diagnostic test Relies on culture so results are not available for 7–14 days May not detect mosaicism
Fluorescence in situ hybridization (FISH)	Cytogenic test Fluorescent-labeled probes identify specific chromosomes Performed on uncultured cells	Tests chromosomes 13, 18, 21, X, and Y May detect deletions if a probe for a specific deletion is requested	Considered a screening test because the test can be falsely positive or falsely negative Results are available in 2–3 days
Chromosomal microarray (CMA)	Molecular DNA Performed on cultured or uncultured cells to detect copy number variants	Detects aneuploidy, unbalanced translocations, microdeletions, and microduplications	Results available in 3–5 days Primary test for women who have amniocentesis following detection of structural anomaly on ultrasound; approximately 6% of fetuses that have a normal karyotype will have a chromosomal abnormality detected by CMA

First-Trimester Diagnostic Testing

Chorionic villus sampling entails collection of placental tissue between 10 and 14 weeks' gestation, transcervically or transabdominally, and completion of karyotyping for detection of aneuploidy using the placental tissue. Other technologies besides the karyotype may be required to detect smaller chromosomal abnormalities, such as microdeletions and translocations in the fetal cells obtained via CVS. Techniques such as fluorescent in situ hybridization (FISH), spectral karyotyping, and comparative genomic hybridization (CGH) can be used to examine chromosomal structure more thoroughly.

One advantage of CVS over amniocentesis is that it can be performed earlier in the pregnancy. This early testing allows more time to consider options and to make decisions including pregnancy termination, particularly if a woman desires to make those decisions before the pregnancy becomes known to others. The disadvantage of CVS is that it does not obtain amniotic fluid, which can be tested to detect the presence of a neural tube defect. The best screening for NTDs consists of MSAFP testing followed by measurement of AFP in amniotic fluid collected during an amniocentesis. The best diagnostic test for NTDs is

anatomic ultrasound examination of the fetus.

Figure 20-9A depicts collection of placental tissue for diagnostic testing, which should be performed by an obstetrician or maternal–fetal medicine specialist. A separate direct DNA test can be completed to identify genetic mutations (e.g., Tay–Sachs disease, cystic fibrosis, hemophilia A, Huntington disease).

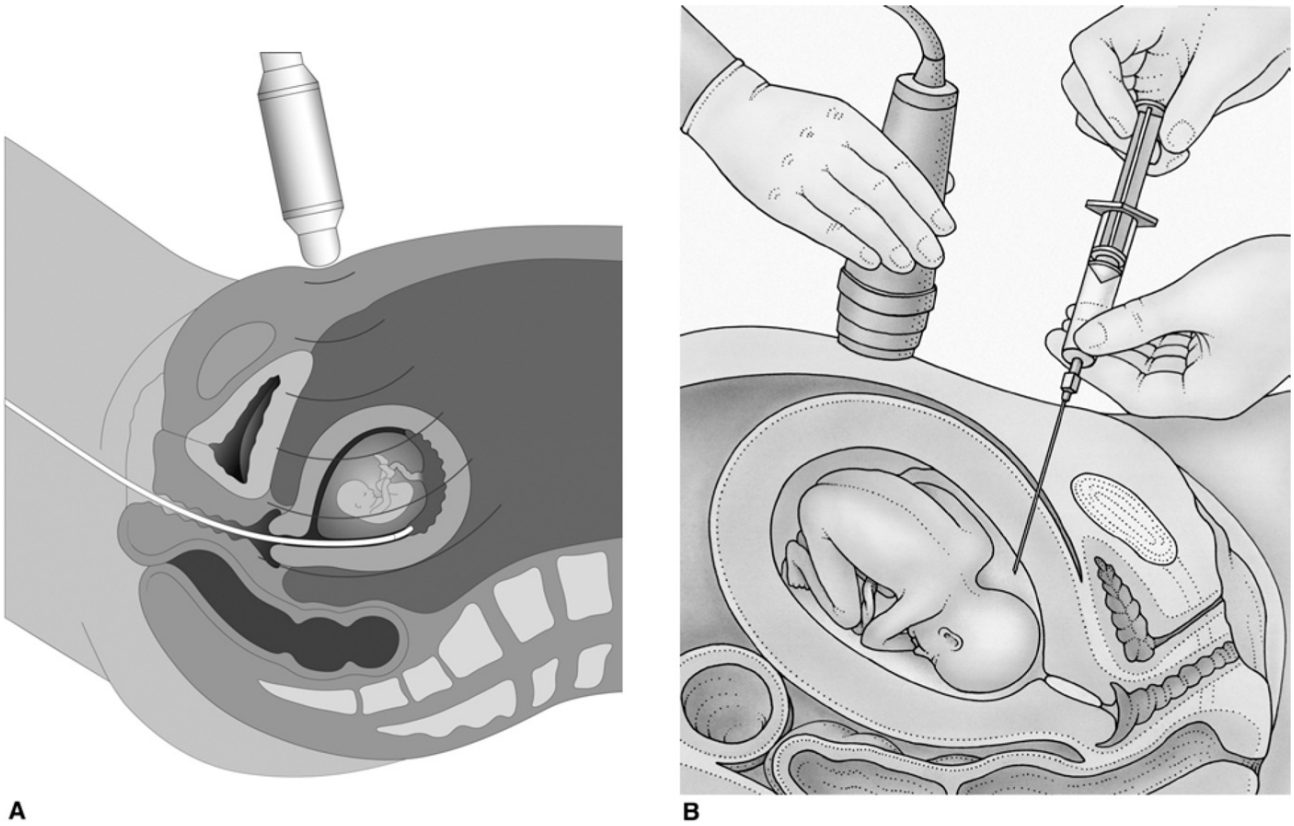


Figure 20-9 A. Transcervical chorionic villus sampling. **B.** Amniocentesis.

A. © Dorling Kindersley/Getty **B.** © Halli Verrinder/Getty

CVS is an invasive procedure that is associated with an additional 0.2% risk for miscarriage above the baseline risk of approximately 15% for an average pregnant woman at a similar gestation.[57] Furthermore, CVS is not performed prior to 10 weeks' gestation due to the documented risk of limb reduction birth defects among infants born to women who had CVS testing at an earlier gestational age. Transabdominal CVS is considered safer, technically less difficult, and more accurate than transcervical CVS.

Second-Trimester Diagnostic Testing

Diagnostic testing in the second trimester can be accomplished via amniocentesis, which entails inserting a fine needle through the woman's abdomen and into the uterus to collect a small sample of amniotic fluid (**Figure 20-9B**). Fetal cells are found in the fluid, separated, and usually grown in a culture medium until the chromosomes can be examined under a microscope. The final result is a fetal "G-banded" karyotype that is used to identify aneuploidies and other chromosomal derangements (**Figure 20-5A** provides an example of a

karyotype display.) As for CVS diagnostic testing, other technologies besides the G-banded karyotype may be required to detect smaller chromosomal abnormalities, such as microdeletions and translocations in the fetal cells obtained via amniocentesis.

Amniocentesis is usually performed at 16 to 18 gestational weeks of pregnancy and has an associated risk of pregnancy loss of approximately 1 in 1000 above the background risk for pregnancy loss less than 24 weeks' gestation.[57] Early amniocentesis (14–15 weeks' gestation) can be performed, but may be associated with a higher risk of pregnancy loss (approximately 1%).[58,59] Early amniocentesis may also be associated with a higher incidence of talipes (club foot) compared to CVS testing, although research has not confirmed this relationship. [58,59]

During a CVS or amniocentesis, direct DNA testing can be completed if warranted to identify genetic mutations in the fetus (e.g., Tay–Sachs disease, cystic fibrosis, hemophilia A, Huntington disease). If an individual chooses to have a diagnostic amniocentesis, an amniotic fluid AFP test can be completed at the same time to screen for NTDs.

Ultrasound

Ultrasound examination of the fetus during the second trimester is performed to establish gestational age and detect anatomic abnormalities such as NTDs and structural defects in the heart, kidneys, cranium/brain, and/or limbs. This examination is targeted to identify physical characteristics associated with aneuploidy phenotypes.[60,61] For example, ultrasound findings in a fetus that include an absent nasal bone, palmar creases, short femur, and echogenic bowel correspond to characteristics commonly found in the trisomy 21 phenotype.[60] Such findings would arouse suspicion for aneuploidy and prompt recommendations for further testing, such as diagnostic amniocentesis. Second-trimester anatomic ultrasounds are usually performed at approximately 20 weeks' gestation for best results.

Genetic Disorder Prevention and Risk Reduction

There are currently no cures for genetic disorders. However, there are some options and preventive approaches that can improve the outcomes for many women and their infants, of which midwives will want to be aware.

Excellent nutrition, including folic acid supplementation,[15] and avoidance of exposures to teratogens are two long-standing preventive strategies that are well known in most geographic areas of the world. Furthermore, many countries can offer genetic risk assessment and genetic testing to women and couples who are considering childbearing so that options can be considered before a pregnancy occurs. Indeed, some groups have taken it upon themselves to offer screening for carrier status of a genetic disorder specific to the population. For example, some Ashkenazi Jewish groups strongly encourage carrier status testing for Tay–Sachs disease prior to conception.

Unfortunately, many couples learn of hereditary disorders only after the birth of an affected child. In this situation, the midwife provides the family with accurate information and compassionate care. Preconception and postpartum testing for genetic disorders and carrier status can be obtained by families who have a child with a birth defect. This testing often provides answers to the questions most families have. Testing may be able to identify the specific disorder, as well as predict the risk for reoccurrence in subsequent children.

Moreover, options can be considered once a definitive answer is given. Options include the choice not to bear biological children, addition of future children via adoption, sperm and/or egg donation, and, in some countries, pre-implantation genetic testing. Pre-implantation testing involves in vitro fertilization (often with the sperm and/or egg from the prospective parents), genetic testing of a cell collected from the eight-cell blastocyst, followed by selection of only normal embryos for transfer into the uterus of the woman.[62] This method assures that only normal embryos without genetic disease mutations will be implanted. Some couples are delighted with this choice, whereas others are strongly opposed to this approach owing to religious or personal reasons; still others find the use of such technology too complex, too expensive, or too reminiscent of eugenics to consider it.[63]

When Genetic Disorders Are Identified

When genetic disorders are identified during pregnancy, parents have many questions and are in need of resources to help them deal with the situation. Helpful resources will vary depending on the setting and geographic area, and cultural, societal, family, and personal beliefs. Women may choose to enlist their families, religious leaders, or mental health counselors for direction and support, and may desire access to medical resources and options, where available. Midwives can be a valuable resource for women who need additional information and discussion of options, and for referrals to other healthcare providers such as geneticists and genetics counselors. If available, women may need and want additional information obtained from a referral to a maternal–fetal medicine physician or neonatologist. If the woman has an established relationship with a midwife, it may also be helpful for her to meet again with the midwife after she has met with and received input from other providers. Making decisions about how to proceed can be emotionally wrenching for women and their families.[45]

Pregnancy termination may be an option, depending on a woman's feelings, the gestational age, and local regulations and laws. Midwives can provide substantial assistance by offering nonjudgmental support and education about options and anticipatory planning.

Many women can continue with midwifery care during pregnancy and birth, in consultation or collaboration with other members of the healthcare team. Frequently, affected newborns will need immediate attention after birth, and arrangements for this care will need to be made in advance, including the site for the birth. Many proponents of prenatal testing believe that the biggest value in testing lies in the ability for parents to access resources and make arrangements for the care of a special-needs child well in advance of the birth. In addition, many support networks exist to help families who have a child with a specific genetic disorder or birth defect, such as the National Down Syndrome Society. Whatever final decision is made, the midwife can be instrumental in supporting a woman and directing her and her family to the best resources.

Financial, Ethical, Legal, and Social Issues in Genetics

Along with the rapid increase in genetic knowledge and technology came the need to address a myriad of financial, ethical, legal, and social issues, collectively referred to as FELSI. The World Health Organization (WHO) has proposed international guidelines regarding ethics in genetic testing, including in developed countries.[64] These guidelines include respect for the autonomy of individuals, beneficence, nonmalfeasance, and justice in provision of genetics-related services. The WHO guidelines are clear that genetic testing can have a profound impact on women and their families. Thus, confidentiality and privacy, the right for persons to receive sufficient information about testing and treatment, and individual autonomy to make decisions about testing and treatment, regardless of geographic area, socioeconomic status, ethnic background, religion, or belief system, are emphasized. Midwives have an obligation to understand and act on the principles related to FELSI, including a duty to fully inform women, offer appropriate testing when available, and refer women and families to additional resources such as genetic counselors or geneticists when their own professional or personal limitations have been reached. Some of the more common FELSI concerns and questions are listed in **Table 20-14**.[65]

Table 20-14 Common Financial, Ethical, Legal, and Social Concerns in Genetic Testing

Disparities in access to genetic resources due to lack of health insurance coverage, inability to pay, low-income economy, other competing priorities in healthcare needs, or geographic location
Distributive injustice (some populations benefit but not others)
Religious and cultural beliefs and perceptions about genetic risk, decision making, health and disease, family, privacy, authority, invasive procedures, childbearing, influence over life events, and use of alternatives to Western medicine
Racial and ethnic discrimination
Cultural factors that may impact accuracy or interpretation of family history information
Privacy and confidentiality
Discrimination or stigmatization by others based on genetic information
Judgment about family genetic history (Is it good or bad?)
Sensitive issues encountered, such as previously undisclosed adoption, rape, incest, misattributed paternity, substance abuse, mental illness, and ethnic origins
Stigma and legality of consanguinity
When or if relatives (who may not want to know) should be informed about an individual's genetic testing results
Implications of family history information and genetic testing results
The negative impact of false-positive test results
The ethical question of testing for disorders that have no cure or treatment
Eugenics
Pregnancy termination
Cost-effectiveness: the high cost of genetic testing versus the possibility of improved health outcomes
Denial of health insurance or employment based on genetic test results[a]

[a] The Genetic Information Nondiscrimination Act of 2008 (GINA) is a federal law that prohibits discrimination in health coverage and employment on the basis of genetic information including genetic test results.

Based on The New York – Mid-Atlantic Guide for Patients and Health Professionals. Ethical, legal, and social issues. In: *Understanding Genetics: A New York, Mid-Atlantic Guide for Patients and Health Professionals.* Washington, DC: Genetic Alliance; July 8, 2009. Available at: https://www.ncbi.nlm.nih.gov/books/NBK115574/. Accessed February 21, 2017.[65]

Future Trends in Genetic Testing

Expanded carrier testing is an approach that can identify a greater number of diseases, and is applied across all ethnic groups, regardless of risk status.[66,67] The availability of expanded carrier testing is rapidly expanding, with such testing increasingly being offered to women and couples in the preconception and early pregnancy periods. Testing for more than 100 autosomal recessive disorders is now readily available in the United States. Nevertheless, expanded carrier screening is not yet recommended for all women.

Direct-to-consumer (DTC) genetic testing is also widely available.[68] The harms and benefits of DTC genetic tests are the subject of debate. Proponents argue that provision of such testing options empowers people by increasing their knowledge about their own health and the genetic basis of their health. Others are concerned that many people have low health literacy, particularly regarding genetic information, and may not be able to interpret or emotionally process these tests without the assistance of trained healthcare personnel.[56] Although some countries and locations have adopted an over-the-counter approach in making the tests freely available, others have banned DTC genetic testing.[69-71]

Where available, individuals can purchase noninvasive DTC tests, including expanded carrier testing from commercial enterprises if they would like to determine, for example, the presence of genetic mutations for heritable disorders, including those that indicate carrier status and susceptibility to multifactorial diseases. Some tests are targeted to pregnant women and couples who would like to determine carrier status for specific genetic and chromosomal disorders, paternity, or the sex of the fetus prenatally. Samples (usually saliva/cheek swab or blood spot via finger stick) are collected in the privacy of one's home and returned to the commercial testing facility. This step may not require the services of a healthcare intermediary, such as the midwife, obstetrician, or family practice provider, although some tests do. One advantage of DTC testing is that it enables potential parents to test for carrier status prior to making the decision to attempt pregnancy. This approach raises the prospect of increasing use of preventive options for couples who want to avoid having a child with specific heritable disorders.[70,71]

Conclusion

In 1990, a collaborative project was begun among several public and private entities in the United States as well as governments and research centers in Europe and Japan with the goal of exploring genetics in more detail. The Human Genome Project focused on sequencing the DNA and mapping the more than 25,000 genes found in the entire genome of *Homo sapiens*. Although the Human Genome Project was completed in the early twenty-first century, applications of its findings and further investigation will continue for the foreseeable future.

In addition to knowledge being gleaned from the Human Genome Project, the body of information about non-Mendelian inheritance patterns has been growing for several years. For the midwife receiving a report of a CVS, amniocentesis, or direct DNA testing, the results usually are phrased in Mendelian terms (e.g., "autosomal recessive"). However, non-Mendelian inheritance patterns are emerging as potential important etiologies for some human genetic diseases. Among these are mitochondrial inheritance, which is a pattern exclusively from the maternal line; mosaicism, in which cells within the same person have different genetic makeup; and genomic imprinting, which occurs when a child receives only one "working" copy from one parent (instead of both) due to gene "silencing" by epigenetic mechanisms. Research continues into the variety of inheritance patterns because they may be of significant clinical relevance in the future.

This chapter has provided a foundation in genetics for midwives and has included several approaches for the provision of genetics-related healthcare services to women and families. The intention is to increase the confidence that midwives have when addressing prenatal genetic risk assessment, testing options, and decision making. Although genetics is considered a high-tech science, midwives have a talent for using low-tech tools to communicate potential risk, and screening and test results, to assist women and their families in shared decision making. Although most genetic disorders do not have a cure, prenatal genetic risk assessment, counseling, screening, and diagnosis have many potential benefits for individuals, including the ability to consider options, make well-informed decisions, access valuable resources, prepare for outcomes in advance, and ultimately optimize the health of women and newborns. Well-informed midwives can make valuable contributions to applications of genetic science to women's health care.

There are many excellent resources that can assist midwives and individuals for whom they care in obtaining additional information related to genetics. A selected list is included in the Resources section.

Resources

Organization	Description	Webpage
Genetics Competencies for Midwives		
Genetic Alliance	A nonprofit health advocacy organization. The website has multiple resources. Includes current recommended genomics competencies for nurses, genetic counselors, and physicians.	http://www.geneticalliance.org
	Understanding Genetics: A New York, Mid-Atlantic Guide for Patients and Health Professionals. This e-book includes a basic review of genetic disorders and tests. The appendix lists the National Coalition for Health Professional Education in Genetics (NCHPEG)'s Core Competencies in Genetics for Health Professionals.	https://www.ncbi.nlm.nih.gov/pubmed/23304754
American Nurses Association (ANA) and International Society of Nurses in Genetics (ISONG)	Essential Genetic and Genomic Competencies for Nurses with Graduate Degrees	https://www.genome.gov/pages/health/healthcareprovidersinfo/grad_gen_comp.pdf
Online	Comprehensive	http://www.ncbi.nlm.nih.gov/Omim/

Mendelian Inheritance in Man (OMIM)	description of single-gene disorders	
Online Gene Tests	Comprehensive list of all gene tests with a description of their characteristics	https://www.genetests.org/tests/
Genetics Learning Resources		
American Society of Human Genetics (ASHG)	Health provider genetics resources	http://www.ashg.org/press/healthprofessional.shtml
National Institutes of Health (NIH), National Human Genome Research Institute (NHGRI)	Information about the Human Genome Project and multiple online educational resources. Includes fact sheets on genetic testing techniques and a glossary of genetic terms.	https://www.genome.gov
Self-Completed Family History Tools		
National Cancer Institute's Center for Biomedical Informatics and Information Technolog	My Family Health Portrait: A tool from the Surgeon General	https://familyhistory.hhs.gov/FHH/html/index.html
Public Organizations[a]		
March of Dimes	The mission of the March of Dimes is to improve the health of babies by preventing birth defects, premature birth and infant mortality. The website has resources for providers and families.	http://www.marchofdimes.com

References

1. Jorde L, Carey J, Bamshad M. Background and history. In: Jorde L, Carey J, Bamshad M, eds. *Medical Genetics*. 5th ed. Philadelphia, PA: Elsevier; 2016:1-5.
2. Nelson K , Holmes LB. Malformations due to presumed spontaneous mutations in newborn infants. *N Engl J Med*. 1989;32(1):19-23.
3. Jorde L, Carey J, Bamshad M. Multifactorial inheritance and common diseases. In: Jorde L, Carey J, Bamshad M, eds. *Medical Genetics*. 5th ed. Philadelphia, PA: Elsevier; 2016:239-264.
4. Crane MJ, Quinn Griffin MT, Andrews CM, Fitzpatrick JJ. The level of importance and level of confidence that midwives in the United States attach to using genetics in practice. *J Midwifery Womens Health*. 2012;57(2):114-119.
5. Feero WG, Guttmacher AE, Collins FS. Genomic medicine: an updated primer. *N Engl J Med*. 2010;362(21):2001-2011.
6. National Cancer Institute. NCI dictionary of genetics terms. Available at: https://www.cancer.gov/publications/dictionaries/genetics-dictionary. Accessed June 25, 2017.
7. Elston R, Satagopan J, Sun S. Genetic terminology. *Meth Molec Biol*. 2012;850:1-9.
8. Dorman JS, Schmella MJ, Wesmiller SW. Primer in genetics and genomics, Article 1: DNA, genes and chromosomes. *Biol Res Nurs*. 2017;19(1):7-17.
9. Sakatani Y, Ischihashi N, Kazuta Y, Yomo T. A transcription and translation-coupled DNA replication system using rolling-circle replication. *Sci Rep*. 2015;5:10404.
10. Read CY. Primer in genetics and genomics. Article 3: explaining human diversity: the role of DNA. *Biol Res Nurs*. 2017;19(3):350-356.
11. Krauss RS, Hong M. Gene–environment interactions and the etiology of birth defects. *Curr Topics Develop Biol*. 2016;116:569-580.
12. Epigenetics. Learn.Genetics: Genetic Science Learning Center. 2015. Available at: http://learn.genetics.utah.edu/content/epigenetics/. Accessed February 21, 2017.
13. Aiello LB, Chiatti BD. Primer in genetics and genomics. Article 4: inheritance patterns. *Biol Res Nurs*. 2017;19(4):465-472.
14. Viswanathan M, Treiman KA, Doto JK, Middleton JC, Coker-Schwimmer EJL, Nicholson WK. *Folic Acid Supplementation: An Evidence Review for the U.S. Preventive Services Task Force*. Rockville, MD: Agency for Healthcare Research and Quality; 2017.
15. Agopian AJ, Tinker SC, Lupo PJ, Canfield MA, Mitchell LE. Proportion of neural tube defects attributable to known risk factors. *Birth Defects Res Part A Clin Molec Teratol*. 2013;97(1):42-46.
16. U.S. Department of Health and Human Services, National Institutes of Health. Neural tube defects (NTDs): condition information. Available at: https://www.nichd.nih.gov/health/topics/ntds/conditioninfo/Pages/default.aspx. Accessed February 21, 2017.
17. Webster A, Schuh M. Mechanisms of aneuploidy in human eggs. *Trends Cell Biol*. 2017;27(1):55-68.
18. Hutaff-Lee C, Cordeiro L, Tartaglia N. Cognitive and medical features of chromosomal aneuploidy. *Handb Clin Neurol*. 2013;111:273-279.
19. Nieschlag E. Klinefelter syndrome: the commonest form of hypogonadism, but often overlooked or untreated. *Deutsches Arzteblatt Int*. 2013;110(20):347-353.
20. Levitsky LL, Luria AH, Hayes FJ, Lin AE. Turner syndrome: update on biology and management across the life span. *Curr Opin Endocrin Diabet Obesity*. 2015;22(1):65-72.
21. Morin SJ, Eccles J, Iturriaga A, Zimmerman RS. Translocations, inversions, and other chromosomal rearrangements. *Fertil Steril*. 2017;107:19-26.
22. Arnold KM, Self ZB. Genetic screening and counseling: family medicine obstetrics. *Primary Care*. 2012;39:55-70.
23. Hook EB. Rates of chromosomal abnormalities. *Obstet Gynecol*. 1981;58(3):282-285.
24. Allen EG, Freeman SB, Druschel C, et al. Maternal age and risk for trisomy 21 assessed by the origin of chromosome nondisjunction: a report from the Atlanta and National Down Syndrome Projects. *Hum Genetics*. 2009;125(1):41-52.
25. Donate A, Estop AM, Giraldo J, Templado C. Paternal age and numerical chromosome abnormalities in human spermatozoa. *Cytogenet Genome Res*. 2016;148(4):241-248.
26. Teratology Society: Organization of Teratology Information Specialists (OTIS). MotherToBaby fact sheets. 2017. Available at: https://www.teratology.org/OTIS_fact_Sheets.asp. Accessed February 21, 2017.
27. Di Renzo GC, Conry JA, Blake J, et al. International Federation of Gynecology and Obstetrics opinion on reproductive health impacts of exposure to toxic environmental chemicals. *Int J Gynaecol Obstet*. 2015;131(3):219-225.
28. Honein MA, Dawson AL, Petersen EE, et al. Birth defects among fetuses and infants of US women with evidence of possible Zika virus infection during pregnancy. *JAMA*. 2017;317(1):59-68.
29. Ornoy A, Reece EA, Pavlinkova G, Kappen C, Miller RK. Effect of maternal diabetes on the embryo, fetus, and children: congenital anomalies, genetic and epigenetic changes and developmental outcomes. *Birth Defects Res C Embryo Today*. 2015;105(1):53-72.

30. Balsells M, Garcia-Patterson A, Gich I, Corcoy R. Major congenital malformations in women with gestational diabetes mellitus: a systematic review and meta-analysis. *Diab Metab Res Rev.* 2012;28(3):252-257.
31. Schaefer S, Nadeau JH. The genetics of epigenetic inheritance: modes, molecules, and mechanisms. *Q Rev Biol.* 2015;90(4):381-415.
32. Lassi ZS, Imam AM, Dean SV, Bhutta ZA. Preconception care: caffeine, smoking, alcohol, drugs and other environmental chemical/radiation exposure. *Reprod Health*. 2014;11(suppl 3):S6.
33. Guttmacher AE, Collins FS, Carmona RH. The family history: more important than ever. *N Engl J Med*. 2004;351(22):2333-2336.
34. The New York – Mid-Atlantic Guide for Patients and Health Professionals. Indications for a genetic referral. In: *Understanding Genetics: A New York, Mid-Atlantic Guide for Patients and Health Professionals*. Washington, DC: Genetic Alliance; 2009. Available at: https://www.ncbi.nlm.nih.gov/books/NBK115563/pdf/Bookshelf_NBK115563.pdf. Accessed June 26, 2017.
35. Korf BR, Berry AB, Limson M, et al. Framework for development of physician competencies in genomic medicine: report of the Competencies Working Group of the Inter-Society Coordinating Committee for Physician Education in Genomics. *Genet Med*. 2014;16(11):804-809.
36. Alldred SK, Takwoingi Y, Guo B, et al. First trimester serum tests for Down's syndrome screening. *Cochrane Database Syst Rev.* 2015;11:CD011975.
37. Dashe JS. Aneuploidy screening in pregnancy. *Obstet Gynecol*. 2016;128:181-194.
38. Lithner CU, Kublickas M, Ek S. Pregnancy outcome for fetuses with increased nuchal translucency but normal karyotype. *J Med Screen*. 2016;23(1):1-6.
39. Chitayat D, Langlois S, Wilson RD, Genetics Committee of the SOGC, Prenatal Diagnosis Committee of the Canadian College of Medical Geneticists. Prenatal screening for fetal aneuploidy in singleton pregnancies. *JOGC*. 2011;33(7):736-750.
40. Allum N, Sibley E, Sturgis P, Stoneman P. Religious beliefs, knowledge about science and attitudes towards medical genetics. *Public Underst Sci.* 2014;23(7):833-849.
41. Farrell RM, Nutter B, Agatisa PK. Meeting patients' education and decision-making needs for first trimester prenatal aneuploidy screening. *Prenat Diag*. 2011;31(13):1222-1228.
42. Hill M, Lewis C, Chitty LS. Stakeholder attitudes and needs regarding cell-free fetal DNA testing. *Curr Opin Obstest Gynecol.* 2016;28(2):125-131.
43. Minear MA, Alessi S, Allyse M, Michie M, Chandrasekharan S. Noninvasive prenatal genetic testing: current and emerging ethical, legal, and social issues. *Ann Rev Genomics Hum Genetics.* 2015;16:369-398.
44. Tekola-Ayele F, Rotimi CN. Translational genomics in low- and middle-income countries: opportunities and challenges. *Public Health Genomics*. 2015;18(4):242-247.
45. Fonda Allen J, Stoll K, Bernhardt BA. Pre- and post-test genetic counseling for chromosomal and Mendelian disorders. *Semin Perinatol.* 2016;40(1):44-55.
46. Gates EA. Communicating risk in prenatal genetic testing. *J Midwifery Womens Health*. 2004;49:220-227.
47. Edwards JG, Feldman G, Goldberg J, et al. Expanded carrier screening in reproductive medicine: points to consider: a joint statement of the American College of Medical Genetics and Genomics, American College of Obstetricians and Gynecologists, National Society of Genetic Counselors, Perinatal Quality Foundation, and Society for Maternal–Fetal Medicine. *Obstet Gynecol*. 2015;125(3):653-662.
48. Latendresse G, Deneris A. An update on current prenatal testing options: first trimester and noninvasive prenatal testing. *J Midwifery Womens Health*. 2015;60(4):360-371.
49. Benn P, Chapman AR. Ethical and practical challenges in providing noninvasive prenatal testing for chromosome abnormalities: an update. *Curr Opin Obstest Gynecol.* 2016;28(2):119-124.
50. Christiansen M, Ekelund CK, Petersen OB, et al. Nuchal translucency distributions for different chromosomal anomalies in a large unselected population cohort. *Prenat Diag*. 2016;36(1):49-55.
51. Norton ME, Wapner RJ. Cell-free DNA analysis for noninvasive examination of trisomy. *N Engl J Med*. 2015;373:2582.
52. Norton ME, Baer RJ, Wapner RJ, Kuppermann M, Jelliffe-Pawlowski LL, Currier RJ. Cell-free DNA vs sequential screening for the detection of fetal chromosomal abnormalities. *Am J Obstet Gynecol.* 2016;214(6):727.e1-727.e6.
53. American College of Obstetricians and Gynecologists. Committee Opinion No. 640: cell-free DNA screening for fetal aneuploidy. *Obstet Gynecol* 2015;126:e31-e37.
54. Tamminga S, van Maarle M, Henneman L, Oudejans CB, Cornel MC, Sistermans EA. Maternal plasma DNA and RNA sequencing for prenatal testing. *Ad Clin CHem.* 2016;74:63-102.
55. Alldred SK, Deeks JJ, Guo B, Neilson JP, Alfirevic Z. Second trimester serum tests for Down's syndrome screening. *Cochrane Database Syst Rev.* 2012;6:CD009925.
56. Fisher J. Supporting patients after disclosure of abnormal first trimester screening results. *Curr Opin Obstest Gynecol.* 2012;24(2):109-113.
57. Akolekar R, Beta J, Picciarelli G, Ogilvie C, D'Antonio F. Procedure-related risk of miscarriage following amniocentesis

and chorionic villus sampling: a systematic review and meta-analysis. *Ultrasound Obstet Gynecol.* 2015;45(1):16-26.
58. Ciach K, Preis K, Swiatkowska-Freund M, Wydra D. [Early or late amniocentesis: which method is safer?]. *Ginekol Pol.* 2007;78(5):400-404.
59. Kornacki J, Gozdziewicz T, Kwinecka B, Skrzypczak J. [Complications rate and pregnancy outcome in women who underwent early and mid trimester amniocentesis]. *Ginekol Pol.* 2007;78(6):443-448.
60. Rao R, Platt LD. Ultrasound screening: status of markers and efficacy of screening for structural abnormalities. *Semin Perinatol.* 2016;40(1):67-78.
61. Rao RR, Valderramos SG, Silverman NS, Han CS, Platt LD. The value of the first trimester ultrasound in the era of cell free DNA screening. *Prenat Diag.* 2016;36(13):1192-1198.
62. Dahdouh EM, Balayla J, Audibert F, et al. Technical update: preimplantation genetic diagnosis and screening. *JOGC.* 2015;37(5):451-463.
63. Londra L, Wallach E, Zhao Y. Assisted reproduction: ethical and legal issues. *Semin Fetal Neonatal Med.* 2014;19(5):264-271.
64. World Health Organization. *Medical genetic services in developing countries: the ethical, legal and social implications of genetic testing and screening*. Geneva, Switzerland: World Health Organization; 2006.
65. The New York – Mid-Atlantic Guide for Patients and Health Professionals. Ethical, legal, and social issues. In: *Understanding Genetics: A New York, Mid-Atlantic Guide for Patients and Health Professionals*. Washington, DC: Genetic Alliance; July 8, 2009. Available at: https://www.ncbi.nlm.nih.gov/books/NBK115574/. Accessed February 21, 2017.
66. Haque IS, Lazarin GA, Kang HP, Evans EA, Goldberg JD, Wapner RJ. Modeled fetal risk of genetic diseases identified by expanded carrier screening. *JAMA.* 2016;316(7):734-742.
67. Lazarin GA, Haque IS. Expanded carrier screening: a review of early implementation and literature. *Semin Perinatol.* 2016;40(1):29-34.
68. Direct-to-consumer genetic testing. *Lancet.* 2012;380(9837):76.
69. Borry P, van Hellemondt RE, Sprumont D, et al. Legislation on direct-to-consumer genetic testing in seven European countries. *Eur J Hum Genet.* 2012;20(7):715-721.
70. Howard HC, Borry P. To ban or not to ban? Clinical geneticists' views on the regulation of direct-to-consumer genetic testing. *EMBO Rep.* 2012;13(9):791-794.
71. Valles SA. Should direct-to-consumer personalized genomic medicine remain unregulated? A rebuttal of the defenses. *Perspect Biol Med.* 2012;55(2):250-265.

APPENDIX

20A

Steps in Constructing a Three-Generation Pedigree

GWEN A. LATENDRESSE

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1. Use standard symbols and notation for building the pedigree (**Figure 20A-1**).

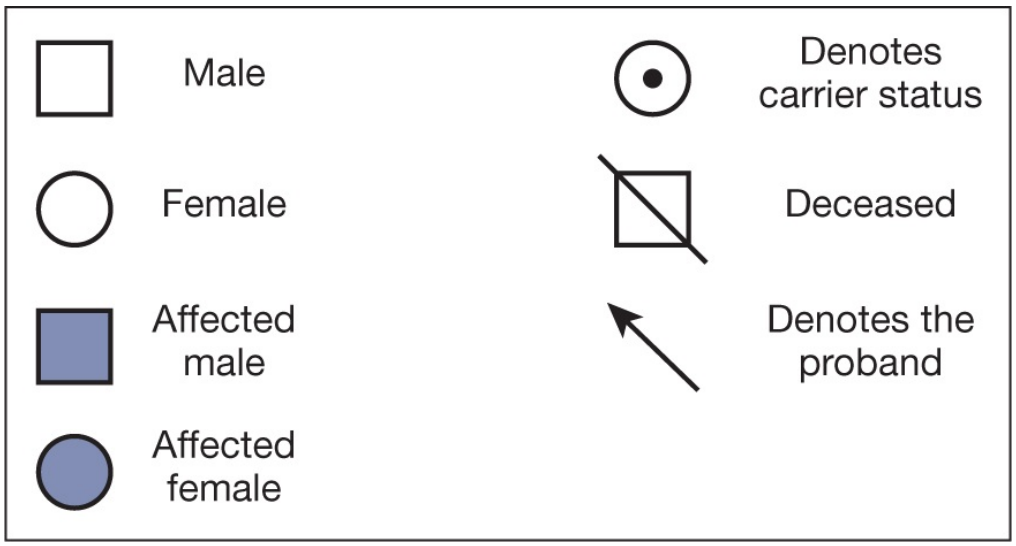


Figure 20A-1 Symbols used in constructing a three-generation pedigree.

2. Computer-generated pedigrees can be constructed, but a simple piece of paper works well.
3. For greater convenience, women can complete a genetic checklist or questionnaire or fill out a computer-generated (i.e., online) pedigree prior to the prenatal visit.
4. Collect information about the individual woman (often called the "proband") and her family members, including grandparents, parents, aunts and uncles, brothers and sisters, and any previous offspring.
5. Collect information about the father of the baby and his family members, including

grandparents, parents, aunts and uncles, brothers and sisters, and any previous offspring.

6. Identify any genetic "red flags" and mark affected relatives (i.e., use shading of symbols as appropriate).
7. List any health or medical conditions, or cause of death (if known), for each relative, along with the current age or age at time of death (if known) next to each corresponding relative on the pedigree.

An example of a three-generation pedigree is shown in **Figure 20A-2**. In this example, the 34-year-old woman is the proband. She has a 2-year-old daughter. Both she and her partner, who is the father of her daughter, are healthy. She has a sister who is healthy, one brother who has type 2 diabetes, and one brother who has a congenital heart defect. Her aunt died of cystic fibrosis at the age of 12.

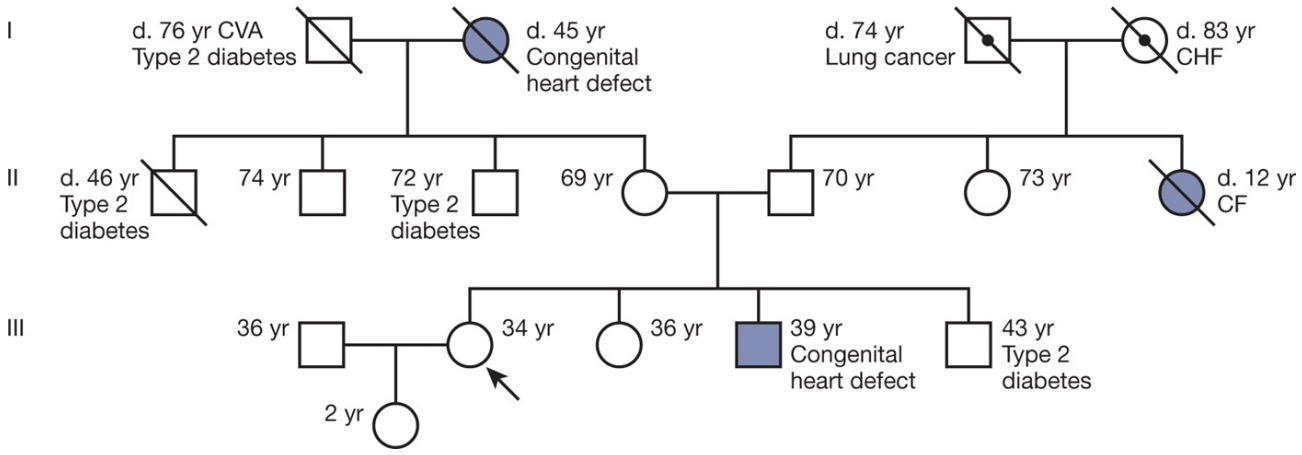


Figure 20A-2 Example of a three-generation pedigree.

Abbreviations: CF, cystic fibrosis; CHF, congestive heart failure; CVA, cardiovascular accident.

CHAPTER

21

Prenatal Care

MARIA OPENSHAW, CECILIA M. JEVITT, AND TEKOA L. KING

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Introduction

Pregnancy is a time of role transition and identity change. Many aspects of primary care are integrated into prenatal care, along with evaluation of social support and referral to resources when needed. Guiding a person through the childbearing year with care that is safe, individualized, and ultimately empowering is one of the great joys of midwifery practice.

The goal of prenatal care is to optimize the health of the woman and developing fetus. The five basic components of prenatal visits, as established in a 1989 report by the U.S. Public Health Service, are (1) early and accurate identification of gestational age; (2) identification of the woman at risk for complications and ongoing risk assessment; (3) ongoing assessment of growth and well-being of the embryo/fetus; (4) health promotion in the form of counseling, education, and provision of resources; and (5) interventions and follow-up for existing physical and/or psychosocial illness and/or consultation with specialized providers as needed.[1]

This chapter reviews the essential components of routine prenatal care for women who have not been diagnosed with medical or pregnancy-related complications. The chapter is divided into six sections. The first section provides an overview of prenatal care, women's experiences of pregnancy, midwifery provision of prenatal care, and definitions of terms used. The second and third sections review the components of the first and return prenatal visits, including diagnosis and dating of pregnancy, prenatal history, physical examination, routine laboratory tests, and ultrasound evaluations. The fourth section discusses nutrition during pregnancy. The fifth section focuses on risk reduction topics that are covered over the course of prenatal care. The final section reviews preparation for birth. Detailed information about genetic counseling and screening tests can be found in the *Genetics* chapter. Management of the common discomforts of pregnancy and obstetric complications are reviewed in the *Pregnancy-Related Conditions* chapter. Medical complications of pregnancy are reviewed in the *Medical Complications in Pregnancy* chapter.

Overview of Prenatal Care

Organized prenatal care in the United States was introduced in the early twentieth century, initially as home visits conducted by public health nurses. The purpose of these visits, which occurred when the woman was 7 to 8 months pregnant, was to detect and prevent eclampsia using the newly invented sphygmometer.[2] By the 1960s, prenatal care had become a ritualized series of 9 to 12 individual visits conducted over the course of the pregnancy. Nevertheless, evidence of effectiveness for the individual components of prenatal care had not been established.

In 1989 and 2005, a U.S. Public Health Service Expert Panel conducted an analysis of the structure and content of prenatal care for the first time.[1,3] These reports recommended a total of 8 visits for nulliparous women and 6 visits for multiparous women, with an additional visit at 41 weeks' gestation for women with post-dates pregnancies. More recently, a 2015 Cochrane review of seven randomized controlled trials (RCTs) of more than 60,000 women evaluated standard prenatal care schedules versus a reduced number of visits and found that maternal and neonatal clinical outcomes were not different for selected low-risk populations, with the exception of a higher rate of perinatal mortality in women with a reduced visit schedule in low-resource countries.[4]

Most prenatal care providers in the United States have not adopted a decreased visit schedule, and most models of prenatal care include between 6 and 12 individual or group visits, depending on the timing of a woman's entry into prenatal care. A woman who is progressing normally through her pregnancy is typically seen every 4 weeks until 28 weeks' gestation, then every 2 weeks until 36 weeks' gestation, and then weekly until she gives birth.[5] When planning a schedule of care for an individual woman, her specific risk factors and her needs and concerns are the first consideration.

Effectiveness and Quality Measures in Prenatal Care

While prenatal care participation is medically and culturally accepted as important, limited evidence supports the effectiveness of prenatal care in the United States. Although women who access prenatal care have lower rates of preterm and low-birth-weight infants, it is not clear if prenatal care is responsible for these outcomes or whether healthier women are more likely to participate in prenatal care. This conundrum has haunted the research on the effectiveness of prenatal care, and the question remains unresolved. Despite significant expansion of prenatal care content over the last several decades, rates of negative birth outcomes such as low birth weight and preterm birth have remained elevated—especially among African American women.[6,7] Nonetheless, some authors argue that prenatal care should not be judged on the basis of specific birth outcomes per se, as longitudinal contact with the healthcare system provides the opportunity for numerous primary and secondary prevention strategies to be initiated. The body of prenatal care research has generally found that early entry into care and higher numbers of prenatal visits are positively correlated with better perinatal outcomes.[8] Furthermore, some medical interventions—in particular, those for preterm birth prevention and the detection of fetal anomalies—cannot be offered to women who first access prenatal care

after the first trimester. Racial and ethnic disparities with regard to the percentage of women who access prenatal care in the first trimester mirrors health disparities in maternal and neonatal outcomes.[7]

In summary, research to determine the effectiveness of individual components of prenatal care is lacking.[1,9] Innovations in the structure and content of prenatal care have been slow to emerge, although research related to nontraditional models of prenatal care—for example, group prenatal care, home visits, telemedicine, and reduced numbers of visits for low-risk women—suggests that these approaches hold promise to improve outcomes.

A Woman's Experience of Pregnancy

Pregnancy is a time of both biologic and social transformation. Just as each individual is different, so is each pregnancy. Prior to offering health education specific to pregnancy, it is important to understand the concerns identified by the woman. Most of the factors that influence the health and outcome of a woman's pregnancy are found outside the hospital or clinic walls. This section highlights a few of these factors that affect an individual's experience of pregnancy.

Social Determinants of Health

Social factors affecting pregnancy include housing, unemployment, food insecurity, exposure to environmental toxins, as well as racism, poverty, and discrimination of all kinds. The term *social determinants of health* (SDOH) refers the growing body of knowledge about how structural conditions in the places where people live and work affect health risks and health outcomes.[10] SDOH theory is increasingly being used in public health to help identify and reduce health disparities. It is also an excellent framework for exploring an individual's concerns in preparation for prenatal care education, counseling, and anticipatory guidance.

The Effects of Stress

The precise influence of stress on fetal development and pregnancy duration is poorly understood, although theories have linked chronic stress to poor health outcomes including preterm birth.[11,12] For example, African American adolescents and persons with a low income are at the greatest risk for poor perinatal outcomes; in addition, these individuals may experience a number of other conditions that independently adversely affect perinatal outcomes.[13-15] Moreover, an increasing body of evidence suggests that epigenetic mechanisms increasingly may mediate changes in fetal developmental programming through the hypothalamic–pituitary–adrenal axis, which controls the body's response to both acute and chronic stress.[14]

Stress during pregnancy may also exacerbate preexisting mental health conditions. The strongest risk factor for postpartum depression or postpartum mood disorder is a history of anxiety, depression, post-traumatic stress disorder, or other psychological illness during pregnancy.[15] Identification of prenatal mood disorders or severe acute or chronic stress during

prenatal care, followed by referral to mental health and social work resources as appropriate, is an important component of mental health support.

Individual Factors That May Adversely Affect Pregnancy Outcomes

Both younger maternal age and older maternal age affect the course of pregnancy. Young women, for example, are more likely have lower levels of education, finances, and social support. They are at higher risk for living in poverty, having depression, and being exposed to intimate-partner violence (IPV) than their peers who defer childbearing. Although adolescent pregnancy is associated with an increased risk for preterm birth and low birth weight, it is difficult to ascertain whether these outcomes occur secondary to a biologic age effect, socioeconomic status, or a combination of factors.[16]

Pregnancy can also be a more complicated and stressful experience for older women. As women age, fertility declines and the incidence of chronic illnesses increases. In addition, some chromosomal abnormalities are more likely to occur at the time of fertilization when a woman's eggs are older.

Prenatal care is unique with regard to the periodic pelvic examinations performed and the frequent reference to topics that impinge on sexual and gender identity. Women who have experienced sexual abuse or assault are at particular risk for feelings of vulnerability, stress, and loss of control during pregnancy.[17] Lesbian and bisexual women, transgender men, and gender-diverse individuals may all experience exacerbations of gender dysphoria during the physical changes of pregnancy or simply experience pregnancy in ways that are unique to that individual.[18],[19] To date, however, little research has been published regarding parental identity formation in transgender men and gender-diverse individuals.

Midwifery Provision of Prenatal Care

Midwifery has a long history and central role in the provision of prenatal care in the United States. Although studies have not demonstrated the effectiveness of prenatal care overall, several seminal studies have documented the effectiveness of midwifery-led prenatal care for specific populations.[20],[21] Midwifery care has shown demonstrable benefits in improving several pregnancy outcomes, including lower rates of preterm labor, decreased rates of low-birth-weight infants, increased access to care, and lower rates of morbidity and mortality.[20,21]

The benefits of midwifery care have been demonstrated internationally as well. For instance, in a review of 11 studies in four developed nations, which sought to compare midwife-led care with physician-led and shared care models of prenatal care, midwife-led care for low-risk women showed benefits in outcomes such as decreased prenatal hospitalization, decreased need for analgesia/anesthesia in labor, decreased number of operative deliveries, fewer episiotomies, and more breastfeeding initiation.[21] Midwifery care in underdeveloped nations has a well-recognized essential role in preventing adverse pregnancy outcomes.

Definitions

The prenatal period includes the time period that extends from the first day of the last normal menstrual period to the start of true labor, which marks the beginning of the intrapartum period. **Table 21-1** summarizes the nomenclature used to describe how many pregnancies (*gravida*, from the Latin word *gravidus*, which means "pregnant") and births (*para*, from the Latin word *parous*, which means "bringing forth or producing") a woman has experienced. It is important that the terms are used correctly with an accepted set of definitions.[22,23] The definitions listed in this chapter are those recommended in most texts. Because parity does not reflect all the possible outcomes of birth, a four-digit system, which encompasses term/premature/abortion/living (TPAL) outcomes, is used to better describe a woman's birth history.

Table 21-1 Nomenclature for Gravida and Para

Term	Definitions
Gravida	The total number of times a woman has been pregnant, regardless of whether the pregnancy resulted in a termination or if multiple infants were born from a pregnancy. A current pregnancy is included in the gravida count.
Nulligravida	A woman who has never been pregnant.
Primigravida	A woman who is pregnant for the first time.
Secundigravida	A woman who is pregnant for the second time.
Multigravida	A woman who has been pregnant more than twice.
Para	The number of times a woman has given birth to a fetus of at least 20 weeks' gestation (viable or not viable), counting multiple births as one birth "event."
Nullipara	A woman who has not remained pregnant beyond 20 weeks' gestation.
Primipara	A woman who has had one pregnancy in which the fetus or fetuses reached 20 weeks' gestation. This woman has given birth once.
Multipara	A woman who has had two or more pregnancies in which the fetus or fetuses reached 20 weeks' gestation. This woman has given birth more than once (counting multiple births as one birth "event").
Gravida/para 2-digit nomenclature	Notation of gravida and para status is essential to describe a woman's reproductive history and status. G2P0: A woman who has been pregnant twice and no births. She may have had abortions or miscarriages. G2P1: A woman who is currently pregnant. She has had one prior pregnancy that resulted in an infant born at 38 weeks' gestation.
Gravida/para TPAL 4-digit nomenclature	**Term (T):** The number of term births the woman has experienced. Term refers to any gestation of 37 completed weeks or more. **Preterm (P):** The number of preterm births the woman has experienced. Premature in this system refers to any infant born between 20 and 37 completed weeks' gestation. **Abortions (A):** The number of pregnancies ending in abortion (either spontaneous or induced). Abortion refers to any fetus delivered before 20 weeks' gestation. **Living (L):** The number of children currently living. Examples: G3 P2002: A woman who is currently pregnant with her third pregnancy. She gave birth to a full-term baby with each prior pregnancy, both of whom are living. G2 P0101: A woman who is currently pregnant. She gave birth to one preterm infant and has

one live child.

G3 P1103: A woman who is currently pregnant with her third pregnancy. She gave birth to one full-term infant and preterm twins. All three offspring are alive.

Gestational Age Versus Embryonic Age

Fertilization takes place during or shortly after ovulation, which is usually about 14 days before the next menstrual period, assuming a 28-day menstrual cycle. Therefore, gestation is approximately 266 days—that is, 38 weeks—in length after fertilization.[24] Women are not usually aware of the date of fertilization, but they typically do know the date their last menstrual period started. Thus, historically, a woman's due date has been calculated as 40 weeks (280 days) after the first day of her last menstrual period. The terms "gestational age" and "weeks' gestation" are used to refer to the number of weeks subsequent to the last menstrual period. The mean gestational age (i.e., menstrual age) for spontaneous labor in primigravid women is 41 weeks from the last menstrual period (287 days), or 40 weeks 3 days (283 days) for the multiparous woman.[24] Estimates based on pooled data are consistent with this finding and suggest that the mean menstrual age for pregnancy is 283 to 284 days.[24]

The study of embryology prefers language referring to "embryonic age," "postconceptional age," or "ovulation age," which more precisely reflects the age of the embryo or fetus. A postconceptional age differs from a gestational age by 2 weeks. When reviewing information about embryonic development or exposure to teratogenic agents, the type of calendar weeks being used—gestational or postconceptional—needs to be confirmed to avoid confusion.

Trimesters

The prenatal period is divided into three trimesters that each consist of 12–13 weeks or 3 calendar months. In practice, the first trimester is considered to be weeks 1 to 12 (12 weeks) because organogenesis is completed at the end of 12 weeks and the risk for spontaneous abortion is significantly reduced at this time. Historically, the second trimester was considered to be weeks 13 to 28 because prior to the introduction of modern neonatal intensive care techniques, 28 weeks' gestation was the limit of viability. The third trimester extends from weeks 28 to 40. The term "post dates" or "post term" is typically used to describe a pregnancy that continues beyond 40 weeks.[24]

Estimated Due Date

For many years, the expected date of the end of pregnancy by birth of a full-term newborn was called the estimated date of confinement (EDC). While this term is still used in many settings, a number of healthcare professionals have changed it to estimated date of delivery or estimated due date (EDD). This change was introduced because of the perception that the word "confinement" connotes illness, limitation, and a passive role based on an archaic practice of isolating women in late pregnancy, rather than recognizing pregnancy as a normal event, a healthy process, with active participation. Although there is no national consensus regarding

the use of EDC versus EDD, the term EDD is used throughout this text.

Initial Prenatal Visit

The primary goal for an initial prenatal visit is to establish the diagnosis of pregnancy and gestational age. If a pregnancy is confirmed, the next step is to determine the woman's goals. Approximately half of all pregnancies are unintended, and a woman may present for prenatal care prior to making a decision about the pregnancy. A pregnant women has three possible choices: (1) to continue the pregnancy and parent the child, (2) to continue the pregnancy and give the infant to an adoptive family, or (3) to terminate the pregnancy, provided that the gestational age is within an allowable time frame for an abortion procedure to be performed.

A good starting point during any initial prenatal visit is to ask the woman open-ended questions to ascertain her own questions, concerns, and plans for the current pregnancy, and to listen carefully to the responses. Options counseling is reviewed in detail in the *Family Planning* chapter. If the woman is planning an ongoing pregnancy, the focus of an initial prenatal visit shifts to establishing the viability of the pregnancy, determining the gestational age of the fetus, detecting significant maternal conditions that could adversely affect the pregnancy, and planning the course of care.

Diagnosis of Pregnancy and Calculation of Gestational Age

The evaluation of pregnancy has three components: (1) confirm that the woman is pregnant, (2) establish a gestational age, and (3) determine whether the pregnancy is viable. Signs and symptoms of pregnancy have historically been categorized into presumptive, probable, and positive signs (**Table 21-2**). While differentiation between these categories is not typically clinically significant today, the importance of these categories remains conceptually important because presumptive and probable signs of pregnancy can be manifestations of other conditions. The two positive signs of pregnancy are ultrasound visualization of a pregnancy and auscultation of fetal heart tones.

Table 21-2	Signs and Symptoms of Pregnancy
Presumptive Signs of Pregnancy (Reported by a Woman in the History)	
Amenorrhea in a woman who previously experienced regular cycles	
Breast or nipple tingling, tenderness, and/or enlargement	
Fatigue	
Nausea and vomiting	
Quickening (perception of fetal movement)	
Pigmentation changes (e.g., chloasma, linea nigra)	
Sustained elevated basal body temperature (in the absence of infection)	
Urinary frequency	
Probable Signs of Pregnancy (Detected by Physical Examination or Laboratory Testing)	
Breast changes (e.g., expression of colostrum, enlargement of breasts and nipples)	
Enlargement of the abdomen	
Enlargement of the uterus	

Changes noted on the pelvic examination:
Ballottement: detection of the fetus bouncing against the uterine wall after the midwife sharply pushes on the uterus with an examining hand in the vagina; palpation technique for detecting a floating object in the body
Chadwick's sign: bluish discoloration of cervix, vagina, and labia due to vascularization
Goodell's sign: softening of the cervix
Hegar's sign: softening of the lower uterine segment
Piskacek's sign: asymmetric lateral bulge in the uterus
Palpable fetal outline or fetal movement
Palpation of uterine contractions
Positive pregnancy test
Positive Signs of Pregnancy (Confirms Pregnancy)
Sonographic evidence of pregnancy
Audible fetal heart tones

Hormonal Pregnancy Tests

Hormonal pregnancy tests are based on the presence of the beta subunit of human chorionic gonadotropin (beta-hCG) in serum or in urine. Beta-hCG is produced by the placenta in increasing amounts in early pregnancy. Levels of this hormone are higher in serum than in urine, so a serum test performed very early in pregnancy may detect a pregnancy when the urine test is still negative.

Urine Pregnancy Tests

Different commercially available urine pregnancy tests have varying sensitivity thresholds, but are usually intended to test positive when the beta-hCG level is approximately 20 to 50 mIU/L. This corresponds to approximately the date of the missed menses, at which time beta-hCG is detectable in urine. A great deal of discrepancy exists in the actual detection rates for specific commercial products.[25] Furthermore, the concentration of beta-hCG in urine can vary within the same woman throughout the day depending on fluctuations in urine concentration. For some women, the first urine of the day tests positive while subsequent voids will test negative.

Although there is little difference between commercially available home urine pregnancy tests and those performed in the clinical setting, there is considerable room for user error. As a consequence, the theoretical accuracy of home pregnancy tests is superior to the accuracy found in typical-use testing. Some reports indicate the typical-use sensitivity is as low as 75%. False-negative results are more likely if the test is performed prior to the first day of the expected menstrual period.[26] As many as 10% of viable pregnancies are not implanted in the uterus by the first day of the missed menstrual period and, therefore, would register as a false-negative pregnancy test if testing is done at that time.[26]

A positive pregnancy test does not confirm the viability of a pregnancy. The percentage of pregnancies that are detected very early and result in a spontaneous abortion has been found to range between 20% and 62%.[27] Women who carefully track their menses and use highly sensitive home pregnancy tests may detect very early "chemical pregnancies" that otherwise

would be noted as a late or missed menses.

Serum Pregnancy Tests

Serum pregnancy tests can detect the presence of beta-hCG either qualitatively or quantitatively. However, as reviewed in the *Anatomy and Physiology of Pregnancy* chapter, individual blood values at specific gestational ages are highly variable, such that a single quantitative serum beta-hCG value does not provide reliable information about the gestational age or viability of the pregnancy. Instead, the clinical utility of quantitative beta-hCG measurement is based on following trends in these measurements in early pregnancy. When the serum beta-hCG is at least 1500 to 2000 mIU/L, the value is within the clinical "discriminatory zone." Approximately 91% of women with a beta-hCG level higher than this range will have an intrauterine gestational sac visible if transvaginal ultrasound is performed. Once the beta-hCG level reaches 3500 mIU/L, 99% of pregnancies are visible on ultrasound.[28]

Serum values of beta-hCG increase exponentially in early pregnancy, doubling approximately every 1.5 to 3 days in the first 5 to 6 weeks, then doubling every 3 to 3.5 days in weeks 7 to 8, peaking at about 8 to 10 weeks' gestation, and then plateauing (**Table 21-3**).[29,30] However, there is a great deal of inter-individual variability in the rate at which hCG rises in maternal plasma. In viable embryos, the minimal rise over 2 days can as little as 53% rather than doubling.[31]

Table 21-3 **Discriminatory Levels of Human Chorionic Gonadotropin Levels and Ultrasound Findings by Gestational Age**[a]

Gestational Age in Weeks from Last Menstrual Period	Approximate beta-hCG Level (mIU/mL)	Ultrasound Findings
3–4 weeks	150–1000	Decidual thickening
4–5 weeks	> 1000–2000	Gestational sac visible with TVUS when beta-hCG is approximately 1000 mIU/mL, and detectable on abdominal ultrasound when beta-hCG is approximately 1800 mIU/mL
5–6 weeks	1000–7200	Yolk sac present when gestational sac is > 10 mm
		Embryo present when gestational sac is > 18 mm
		Cardiac activity present when crown–rump length is > 5 mm
6–7 weeks	> 10,800	Crown–rump length is 4–9 mm

Abbreviations: beta-hCG, beta subunit of human chorionic gonadotropin; TVUS, transvaginal ultrasound.

[a] Beta-hCG values are approximate. Each ultrasound department will have institutionally specific values that can be used to guide management.

Modified with permission from Snell BJ. Assessment and management of bleeding in the first trimester of pregnancy. *J Midwifery Womens Health.* 2009;54:483-491.[30] © 2009, with permission from Wiley.

Serial measurements of quantitative serum beta-hCG values repeated every 48 to 72 hours are often used in conjunction with an early ultrasound and other clinical data to help determine the presence and viability of an intrauterine pregnancy, rule out ectopic pregnancy, or follow

the decline when a spontaneous abortion has occurred. The *Pregnancy-Related Conditions* chapter reviews management of first-trimester spontaneous abortion, ectopic pregnancy, and *pregnancy of unknown origin* (the diagnostic term for the clinical scenario in which the serum beta-hCG is positive but the ultrasound does not detect a pregnancy in the uterus).

Establishing Gestational Age and Estimated Due Date

Accurate assessment of gestational age is one of the most important aspects of caring for a pregnant woman. Gestational age at the time for birth is the single most valuable predictor of newborn health. An accurate calculation of gestational age is necessary to appropriately select interventions for threatened preterm labor, to determine the adequacy of fetal growth, and to time the initiation of fetal surveillance or induction of labor. Conversely, incorrect estimation of the gestational age can lead to inappropriate timing for screening, misinterpretation of data, unnecessary intervention, or a failure to intervene when indicated. The opportunity for accurate assessment of gestational age decreases as the pregnancy progresses, and estimated due dates should not be recalculated after the second trimester. Several methods can be used to calculate the EDD, all of which have some variability.

Estimated Due Date by Last Menstrual Period

The date of the first day of the last menstrual period (LMP) is used as the baseline for an initial determination of gestational age and the EDD. However, use of LMP to determine gestational age has several limitations. The normal menstrual cycle is 21 to 35 days, with the mean being 28.4 days. Many women with longer or shorter cycles, however, may not ovulate 14 days after their last menstrual period. Similarly, there is variation in the timing of implantation of the embryo, which can occur 5 to 14 days after the estimated time of ovulation.[32]

While many women can accurately recall the date of LMP within 1 to 2 days,[29] the reported LMP can also be inaccurate. Some women do not keep a record of their menstrual periods, and others have a history of irregular cycles or amenorrhea despite having an ovulatory cycle. In addition, various medications, recent changes in weight, breastfeeding, and shift work are examples of factors that can change menses frequency and timing of ovulation.

Many women experience spotting in early pregnancy that may be perceived as a menstrual period. It is theorized that this bleeding, or spotting at the time of implantation of the blastocyst, is a result of the invasive activity of the chorionic villi in the endometrial lining.[33] Some recent studies, however, suggest that bleeding that occurs near the time of the expected menses is common but not directly connected to implantation.[34] In one large study, 27% of pregnant women experienced bleeding in the first trimester, typically between 5 and 8 weeks' gestation, which corresponded to the shift from luteal to placental hormonal dominance.[35]

Given the inexact nature of menstrual frequency, it can help to establish the date of the last *normal* menstrual period (LNMP) before estimating gestational age. The woman's description of her last menstrual period is compared to her description of her regular menstrual periods with regard to timing, duration, and flow. Supplemental information that can be used in conjunction with LNMP to improve the accuracy of the estimated due date includes coital

timing (inclusion or exclusion dates for exposure to conception), use (or not) of contraception (i.e., type, timing, consistency), and date(s), type, and result(s) of any home ovulation timing or pregnancy test that the woman has done prior to seeking prenatal care.

Naegele's Rule

Traditionally the EDD is initially calculated using Naegele's rule, which is based on the assumption of a 28-day menstrual cycle. Franz Karl Naegele, a German physician, authored a text in 1812 that contained the advice to estimate that a pregnancy would terminate approximately 40 weeks (280 days) after the first day of the last menstrual period.[36] Although this theory is not based on any modern concept of research and its accuracy has been questioned,[37,38] it remains widely used today and is the basis of many pregnancy calculators.

To use Naegele's rule, 7 days are added to the LMP and then 3 months are subtracted to obtain the EDD. For LMPs that occur after the first 3 months of the year, an additional year is added. Thus, the formula is the first day of LMP + 7 days – 3 months = EDD.

Use the actual number of days in the month of the LMP if adding 7 days crosses over to the next month. This is illustrated in the following example of calculating the EDD by Naegele's rule: If the LMP is May 28, then 5/28 + 7 days = 6/4 (May has 31 days) – 3 months = EDD is March 4 of the next year. Note that Naegele's rule does not account for the additional day in a leap year.

Estimated Due Date by Pregnancy Wheels and Apps

The pocket gestational wheel, which is traditionally used to calculate EDD and determine gestational age, calculates an EDD based on 280 days for the duration of gestation. Such gestational wheels are easy to use, but their calculated EDDs vary by 1 to 7 days.[38]

In many settings, the pocket wheel has largely been replaced by computer-based software. Most electronic health record systems used for prenatal care and obstetric ultrasonography include dating calculators, which allow the automatic calculation of an EDD. The electronic calculators that can be found on the Internet or as an app remove most of the margin of error associated with pregnancy wheels and arithmetic calculations.[38]

Artificial Reproductive Technology

When a pregnancy is conceived through artificial reproductive technology (ART), the date of insemination or embryo transfer is used instead of the LMP. A precise due date can be calculated using the date of insemination or and the date of embryo transfer.

First-Trimester Ultrasound

Calculation of gestational age and EDD based on ultrasound performed before 13 6/7 weeks is the most accurate method for dating pregnancy.[39] In maternity care, an ultrasound may be requested to provide a general or targeted assessment. The usual application of ultrasound in pregnancy is classified as one of three types of evaluation as described in **Table 21-4**.

Table 21-4 Ultrasound in Pregnancy

Type of Ultrasound	Description
Standard ultrasound	Often referred to as an anatomy ultrasound, this examination provides fetal biometry needed to confirm gestational age and is simultaneously able to detect most major morphologic malformations prior to the time that the fetus would be viable if born. A standard ultrasound identifies gestational age, fetal number, fetal biometry, amniotic fluid volume, cardiac activity, placental position, placental localization, and complete assessment of fetal anatomy. Maternal adnexa and cervix including cervical length are usually included. Cervical length may be an included component of the standard examination. The standard ultrasound is offered to all pregnant women between 18 and 20 weeks' gestation.
Limited ultrasound	Performed for a specific indication and is not considered a replacement for the routine standard ultrasound. Components of a limited examination may include assessment of gestational age, embryonic or fetal viability, confirmation of fetal heart activity, identification of number of fetuses, confirmation of fetal presentation and position, determination of placental location, determination of amniotic fluid volume, fetal well-being and cervical length. A limited ultrasound may be done in any trimester and may be done in the office setting. Midwives may acquire advanced skills in performing limited ultrasounds in accordance with American College of Nurse-Midwives guidelines.
Specialized or targeted ultrasound	A specialized ultrasound is ordered when an anomaly is suspected based on a woman's history, laboratory results, or abnormal findings on a standard ultrasound. Additional technology can be utilized for specific purposes. For example, fetal echocardiogram can be used to accurately diagnose cardiac malformations. Fetal Doppler velocimetry is used to evaluate maternal-fetal blood flow particularly in cases of fetal growth restriction, multifetal gestations, severe maternal hypertension and placental abnormalities. A specialized ultrasound is performed/reviewed by physicians trained in the technology—typically maternal fetal medicine or specialist radiologists.

First-trimester ultrasound is used to confirm LMP dating or to establish the gestational age for pregnant women without a known LMP. The most accurate first trimester ultrasound measurement for determining gestational age is the crown–rump length measurement. This dating method can reduce the number of inductions of labor for post-dates pregnancies and has been shown to be cost-effective in most developed nations.[39,40] If the difference between the ultrasonographic and LMP due dates is more than 5 to 7 days, the ultrasound-calculated EDD will be used. The recommendations for changing due dates based on LMP and ultrasound dating discrepancies are presented in **Table 21-5**.

Table 21-5 Guidelines for Establishing Estimated Due Date Based on Last Menstrual Period and Ultrasound Discrepancies

Gestational Age Range Based on LMP	Ultrasound Measurements Obtained	Number of Days Difference Between LMP and Ultrasound Dates That Supports Use of Ultrasound Dating
≤ 8 6/7	CRL	> 5 days

9 0/7 to 13 6/7	CRL	> 7 days
14 0/7 to 15 6/7	BPD, HC, AC, FL	> 7 days
16 0/7 to 21 6/7	BPD, HC, AC, FL	> 10 days
22 0/7 to 27 6/7	BPD, HC, AC, FL	> 14 days
28 0/7 and beyond	BPD, HC, AC, FL	> 21 days

Abbreviations: AC, abdominal circumference; BPD, biparietal diameter; CRL, crown–rump length; FL, femur length; HC, head circumference; LMP, last menstrual period.

Modified with permission from American College of Obstetricians and Gynecologists. Committee Opinion No. 700: methods for estimating the due date. *Obstet Gynecol*. 2017;129:e150-e154.[37]

Second-Trimester Ultrasound

Second-trimester ultrasound estimates of fetal age are based on composite measurements of the fetal head circumference, biparietal diameter, and femur length, as well as the woman's abdominal circumference.[37] These estimates are slightly less accurate than first-trimester ultrasound gestational age determinations because there is more individual variation in fetal growth and development as pregnancy progresses.

Less Reliable Methods of Dating Pregnancy

Although uterine size correlates positively with gestational age, fundal measurement is a less reliable predictor of EDD than the LMP.[41,42] Clinical assessment of uterine size may be affected by distension of the bladder (up to 7 cm), maternal adipose deposition,[41] retroversion or retroflexion of the uterus, presence of fibroids, uterine anomaly, position and presentation of the fetus, amniotic fluid volume, and clinician experience.[42] Measurement of the uterine fundal height above the symphysis to monitor fetal growth is a standard component of prenatal care. The use of fundal height measurements is reviewed in detail in **Appendix 21A**.

Other indicators such as the onset of quickening, along with the clinician's notation of first detection of fetal heart tones by ultrasound or fetoscope or the client's signs and symptoms of pregnancy, are poor predictors of gestational age but may be supportive data if reliable dating is not available. In women in whom the fetal heart tones have been detectable via a Pinard stethoscope for at least 21 weeks, approximately 90% will be at least 38 weeks' gestation. Similarly, when fetal movement has been detected for at least 25 weeks, it is 90% certain that the woman is at least 38 weeks' gestation. Although these measures are helpful in predicting the presence of fetal maturity, they are not helpful in ruling out post-maturity. Nonetheless, these observations may be of value in low-resource or disaster settings, and it is traditional to record the initial detection of the fetal heart tones and fetal movement in the woman's health record.

Prenatal History

After the pregnancy is diagnosed and the woman's choice to pursue prenatal care has been confirmed, a complete history is obtained. This includes past medical and surgical history,

along with family, genetic, social, menstrual, obstetric, gynecologic, sexual, and contraceptive history. All current medications and allergies are noted. The components of the woman's health history that may adversely affect the course of pregnancy are detailed in **Table 21-6**.

Table 21-6 Components of the Medical History That May Affect the Course of Pregnancy

History Element	Clinical Implications
Present pregnancy	Signs of pregnancy complications such as cramping, bleeding, or excessive vomiting in the first trimester; signs of preterm labor or preeclampsia in the late second and early third trimesters
	Signs of medical complications such as fever, dysuria, or rash
	Extreme discomforts of pregnancy
	Exposures to teratogens such as drugs or medications, street drugs, alcohol, tobacco, X rays, environmental exposures, or occupational exposures
Oral health	Periodontal disease is common among pregnant women and has in some studies been associated with preterm birth. Women with poor oral health can transmit the bacteria that cause dental caries to their newborn and studies have shown that their children are more likely to develop dental caries. Dental care during pregnancy can prevent this transmission. It is recommended that the woman be asked about bleeding gums or tooth pain and when she last had dental care.
Medical history	Medical conditions that are associated with increased risks for adverse maternal or fetal outcomes, such as diabetes or hypertension; they indicate a need for consultation, collaboration, co-management, or referral to a physician
Surgical history	Uterine surgery such as cesarean section or myomectomy, which may indicate a need for a physician consultation to evaluate the ability of the uterine scar to withstand a subsequent labor
	Significant surgeries on vital organs such as the heart, lungs, or kidneys
Family history	Evidence of genetic disorders such as hemophilia, cystic fibrosis, or intellectual disability, which may suggest the need for specific genetic counseling or tests
Social history	Risks that indicate a possible need for additional visits, social services, psychology consultation, or referral:
	Living situation: barriers to accessing care such as transportation, work schedule, unemployment, homelessness, food insecurity
	Adolescent pregnancy
	History of substance use, alcohol use disorder
	Personal stressors: history of sexual or physical abuse, or emotional abuse
Menstrual history	Irregular or regular cycles
	Cycle length
	Number of days between menses
Reproductive history	Gravidity, parity (TPAL)
	Dates of previous births, as well as weights of newborns
	Pregnancy complications such as mid-trimester loss, gestational diabetes, preterm labor, preterm premature rupture of membranes, fetal growth restriction, or preeclampsia[a]
	Induction or augmentation of labor, prolonged stages of labor
	Previous birth complications: mode of birth, shoulder dystocia, vaginal lacerations, or episiotomies
	Postpartum complications
	Newborn complications such as injury, infection, or congenital defects
Gynecologic history	Past or current STIs, especially herpes simplex virus and HIV
	Diagnosed malformations of the reproductive tract

	History of female genital cutting or female circumcision
	Procedures for treatment of abnormal cervical cytology
	Vulvovaginal disorders
Sexual history	Sexual activity: number of current partners and number of partners in the last year; gender of partners (male, female, or both)
	Risks for STIs and use of barrier methods for prevention of STIs
Contraceptive history	What was the last form of contraception used and when was it discontinued?
Current medications	Current medications, dose, and duration of treatment
	Vitamins, supplements, or herbal remedies
Allergies	Allergies to medications
	Allergies to foods or environmental exposures

Abbreviations: HIV, human immunodeficiency virus; STI, sexually transmitted infection; TPAL, term birth, premature birth, abortion, living children.

[a] Each of these conditions is an indication for physician consultation or referral: (1) History of midtrimester loss: may indicate a need for referral and consideration for cerclage placement; (2) prior gestational diabetes: early pregnancy screening for glucose intolerance is recommended; (3) history of preterm birth or preterm premature rupture of membranes: physician consultation to consider or plan intramuscular progesterone beginning in the second trimester; and (4) previous preeclampsia: treatment with low-dose aspirin (81 mg) is recommended for women with a history of early-onset preeclampsia and preterm delivery at < 34 0/7 weeks' gestation or women who had preeclampsia in more than one prior pregnancy.

While extra time is typically allotted for the initial prenatal visit, the content of the visit may need to be divided between multiple visits. The scope of questions asked at the initial prenatal visit may seem intrusive. Sensitive subjects such as intimate-partner violence, sexual abuse history, and substance use can be readdressed at subsequent visits when a trusting relationship has been established.

Physical Examination

A complete physical examination, including weight, height, and vital signs, is usually performed during the initial prenatal visit. Oral health is particularly important during pregnancy and the physical examination should include assessment of the gingiva. The other aspect of the physical examination specific for pregnancy includes the abdominal examination with auscultation for fetal heart tones (if gestational age permits) and a pelvic examination with clinical pelvimetry. The techniques for the abdominal examination and clinical pelvimetry are presented in Appendix 21A and **Appendix 21B**.

Undressing and exposure of genitals may be a trigger for women who have a history of abuse or those experiencing ongoing abuse. Some women may decline to have a pelvic examination for personal or cultural reasons. Under no circumstances should an individual be coerced to undergo a pelvic examination. The midwife may explain the benefits of doing the examination and the information that can be obtained from the pelvic examination, but the woman is entitled to decline or to defer the examination to another time.

Laboratory Tests, Genetic Tests, and Carrier Screening

Laboratory tests recommended or offered to women at the first prenatal visit fall into one of the following four categories:

- Routine laboratory tests that detect maternal infection or immunity to infections known to have adverse perinatal effects, which are recommended for all pregnant women (**Table 21-7**).[5,43-45]
- Laboratory tests that detect disorders or infections in women who have an increased risk (**Table 21-8**).[5,43-45]
- Tests to screen or diagnose detect fetal aneuploidy are offered to all pregnant women via a shared decision-making process. These tests are reviewed in detail in the *Genetics* chapter. An algorithm for recommending these tests during prenatal care is presented in **Figure 21-1**.[46]
- Carrier screening of either biological parent to detect carrier status for inheritable autosomal disorders can be offered using an ethnic-specific, panethnic, or expanded carrier screening strategy.[47] Carrier screening can be performed for anyone at any age. The available carrier screening tests and controversy regarding ethnic-specific versus expanded carrier screening strategies are reviewed in detail in the *Genetics* chapter.

Table 21-7 Laboratory Tests Recommended for All Pregnant Women at Initial Prenatal Visit[a]

Laboratory Test[a]	Recommendation and Follow-Up
Blood Tests	
Blood type, Rhesus type, antibody screen	Rh (D)-negative women who do not have alloantibodies are offered prophylactic anti-D immune globulin at 28 weeks or to prevent alloimmunization. If Rh alloantibodies are present, the baseline titre will be used to monitor the condition.
Hemoglobin, hematocrit, MCV, platelets: usually ordered as a CBC	A complete blood count will detect anemia. The MCV will determine whether anemia is microcytic, normocytic, or macrocytic, with this information then being used to identify appropriate tests for further evaluation of anemia.
HBsAg	Recommended for all women at an initial examination and then again in the third trimester for women who were not already screened prenatally, those who engage in behaviors that put them at *high risk* for infection, and those with signs or symptoms of hepatitis at the time of admission to the hospital for delivery. Risk factors: Having had more than one sex partner in the previous six months Evaluation or treatment for an STI Recent or current injection-drug use An HBsAg-positive sex partner
HIV	Opt-out approach for all women Recommended for all women at an initial examination and then again in the third trimester for women at high risk for acquiring HIV
Rubella titer	Women who are not immune to rubella are counseled to avoid exposure to the disease during pregnancy and vaccinated postpartum.
Syphilis	Recommended for all women at an initial examination and then again in the third trimester for

(VDRL/RPR)	women with continued or new risk for STIs: Uninsured, living in poverty, sex worker, or use of illicit drugs Diagnosed with another STI Living in high-prevalence areas Not previously tested in pregnancy Had a positive test in the initial prenatal screen
Blood Tests	
Varicella immunity	Obtain laboratory test for women who do not have a history of infection or vaccination and women who are not immune to varicella are counseled to avoid exposure to the disease during pregnancy and vaccinated postpartum
Urine Tests	
Urinalysis	Urinalysis for protein is performed to establish a baseline for comparison if renal function needs to be assessed later in pregnancy
Urine culture	Untreated bacteriuria increases the risk a woman will develop pyelonephritis; treatment of bacteriuria is per standard guidelines
Endocervical Culture	
Chlamydia[b]	Chlamydia screening strategy may be universal or selective. If selective, the indications are: All women < 25 years Women > 25 years who have a risk for STIs: Women who have a new sex partner More than one sex partner A sex partner with concurrent partners A sex partner who has an STI Third trimester: Rescreen women < 25 years and women > 25 years who have risks for STIs If the culture is positive, a test of cure is recommended 3 weeks after treatment and rescreen 3 months after treatment
Gonorrhea	Gonorrhea screening strategy may be universal or selective. If selective, the indications are: All women < 25 years Women > 25 years who have a risk for STIs: Living in a high-morbidity area Previous or coexisting STI New or multiple sex partners Inconsistent condom use among persons not in mutually monogamous relationships Exchanging sex for money or drugs Third trimester: Rescreen all women who have continued high risks for STIs If the culture is positive, rescreen 3 months after treatment

Abbreviations: CBC, complete blood count; HBsAg, hepatitis B surface antigen; HIV, human immunodeficiency virus; MCV; mean corpuscular volume; RPR, rapid plasma reagin; STI, sexually transmitted infection; VDRL, Venereal Disease Research Laboratory.

[a] If these tests were performed preconceptionally, repetition is not necessary.

[b] Chlamydia can be detected via endocervical culture or via nucleic acid amplification (NAAT) of urine. Culture is preferred but not mandatory.

Based on American College of Obstetricians and Gynecologists, American Academy of Pediatrics. *Guidelines for Prenatal Care.* 8th ed. Washington, DC: American College of Obstetricians and Gynecologists; 2017[5]; U.S. Preventive Services Task Force. Published recommendations. Available at:

https://www.uspreventiveservicestaskforce.org/BrowseRec/Index/browse-recommendations. Accessed July 4, 2017[43]; Centers for Disease Control and Prevention. 2015 sexually transmitted diseases treatment guidelines. 2015. Available at: https://www.cdc.gov/std/tg2015/default.htm. Accessed July 4, 2017[44]; Centers for Disease Control and Prevention. TB guidelines testing and diagnosis. 2017. Available at: https://www.cdc.gov/tb/publications/guidelines/testing.htm. Accessed July 4, 2017.[45]

Table 21-8 Laboratory Tests Recommended for Selected Indications During Pregnancy

Condition (Test)	Recommendation and Follow-Up
Cervical cancer screening (Pap test)	Pregnancy does not change the recommended interval for cervical cancer screening
Type 2 diabetes (fasting blood glucose, HbA1c)	Risk factors for type 2 diabetes are BMI ≥ 25 kg/m^2 (23 kg/m^2 for Asian populations) and one of the following: Clinical condition associated with insulin resistance First-degree relative with diabetes Gestational diabetes mellitus in a previous pregnancy HbA1c ≥ 5.7% (39 mmol/mol) Impaired glucose tolerance or impaired fasting glucose on previous testing HDL cholesterol level < 35 mg/dL (0.90 mmol/L) and/or a triglyceride level > 250 mg/dL (2.82 mmol/L) High-risk race/ethnicity (e.g., African American, Latina, Native American, Asian American, Pacific Islander) History of cardiovascular disease Hypertension (≥140/90 mm Hg or on therapy for hypertension) Physical inactivity Polycystic ovary syndrome
Hepatitis C	Screen all pregnant women at increased risk Risk factors: Past or current injection-drug use Having received a blood transfusion before July 1992 Receipt of an unregulated tattoo Long-term dialysis Known exposure to hepatitis C
Thyroid function (TSH)	Women with signs or symptoms of thyroid disease and women using thyroid medications
Trichomoniasis (wet prep for microscopic visualization)	Screen women who are HIV-positive
Tuberculosis (tuberculin skin test)	Women at increased risk for latent tuberculosis infection: Those who were born or lived in countries with high prevalence rates Those who live in high-risk settings (e.g., homeless shelters, correctional facilities) Those with HIV Those in close contact with other persons known or suspected to have tuberculosis, and those who work in longterm care facilities, nursing homes, or correctional facilities

Abbreviations: BMI, body mass index; HbA1c, hemoglobin A1c or glycosylated hemoglobin; HDL, high-density

lipoprotein; HIV, human immunodeficiency virus; TSH, thyroid-stimulating hormone.

Based on American College of Obstetricians and Gynecologists, American Academy of Pediatrics. *Guidelines for Prenatal Care*. 8th ed. Washington, DC: American College of Obstetricians and Gynecologists; 2017[5]; U.S. Preventive Services Task Force. Published recommendations. Available at: https://www.uspreventiveservicestaskforce.org/BrowseRec/Index/browse-recommendations. Accessed July 4, 2017[43]; Centers for Disease Control and Prevention. 2015 sexually transmitted diseases treatment guidelines. 2015. Available at: https://www.cdc.gov/std/tg2015/default.htm. Accessed July 4, 2017[44]; Centers for Disease Control and Prevention. TB guidelines testing and diagnosis. 2017. Available at: https://www.cdc.gov/tb/publications/guidelines/testing.htm Accessed July 4, 2017.[45]

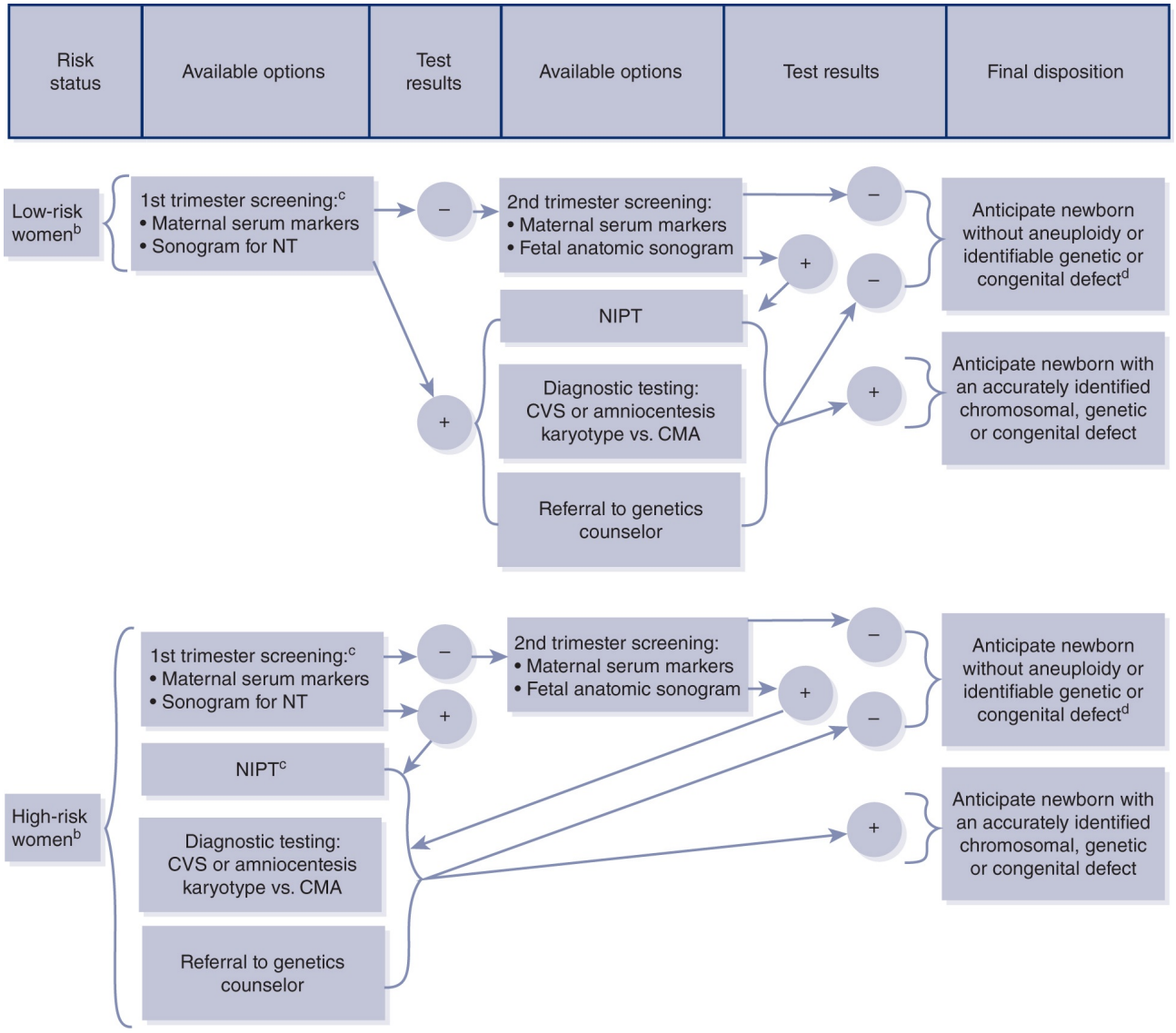


Figure 21-1 Algorithm for offering prenatal testing options.[a]

Abbreviations: CMA, chromosomal microarray analysis; CVS, chorionic villus sampling; NIPT, noninvasive prenatal testing; NT, nuchal translucency/thickness; ONTD, open neural tube defect.

[a] Based predominantly on Level A recommendations (good and consistent scientific evidence).

[b] In the absence of risk factors, women are considered “low risk.” Women are considered “high risk” when specific risk factors apply: advanced maternal age (age 35 years or older); high-risk ethnic/racial heritage (i.e., Ashkenazi Jewish, Mediterranean, African American); consanguinity (blood relationship of parents); family history (mother’s and father’s) of a known or suspected genetic condition; multiple affected family members with the same or related disorders; any major malformations (i.e., heart,

kidney, brain) or other birth defects occurring in the mother, father, grandparents, offspring, or close relatives (brothers/sisters); congenital blindness or deafness in family members; extremely tall or short stature of mother, father, or their relatives; developmental delays or mental retardation occurring in the mother, father, offspring, or close relatives; recurrent pregnancy losses (two or more) for the mother; environmental exposure to known or suspected teratogens; and infertility or premature ovarian failure.

[c] First-trimester screening for aneuploidy will not detect risk for ONTD. If a woman does not have second-trimester screening or amniocentesis, she should be informed that ONTD-specific screening is available (i.e., second-trimester ultrasound alone or in combination with maternal serum alpha-fetoprotein serum protein [MSAFP]).

[d] Negative screening tests are not a 100% guarantee that a fetus does not have aneuploidy or ONTD. Diagnostic testing has limitations—for example, it does not detect all possible genetic or congenital abnormalities.

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Routine Laboratory Tests

Normal values and reference ranges during pregnancy for common blood tests are listed in **Table 21-9**.[48]

Table 21-9 **Normal Laboratory Values in Pregnancy**

Test	Abbreviation	Nonpregnant Adult	First Trimester	Second Trimester	Third Trimester
Hematology					
Ferritin (ng/mL)[a]		10–150	6–130	2–230	0–116
Folate, serum (ng/mL)		5.4–18.0	2.6–15.0	0.8–24.0	1.4–20.7
Hemoglobin[a] (g/dL)	Hgb	10.4–15.2[b]	11.6–13.9	9.7–14.8	9.5–15
Hemoglobin A1c (%)	HbA1c	5.2	5.0	4.9	5.1
Hematocrit[a] (%)	Hct	32.0–45.0[b]	31–41	30–39	28–40
Iron, total iron-binding capacity[a] (mcg/dL)	TIBC	251–406	278–403	Not reported	359–609
Iron, serum[a] (mcg/dL)		41–141	72–143	44–178	30–193
Mean corpuscular hemoglobin (pg/cell)	MCH	23.2–33.3[b]	30–32	30–33	29–32
Mean corpuscular volume	MCV	73.4–98.3[b]	80–100	82–97	81–99
Platelets ($\times 10^9$/L)		172–453[b]	174–400	155–409	146–426
Red blood cell count	RBC	3.70–5.20[b]	3.42–4.55	2.81–4.49	2.71–4.43
Reticulocyte count (%)	Retic	0.5–1	1–2	Not reported	Not reported
White blood cell count ($\times 10^3$/mm^3)	WBC	3.9–12.5[b]	5.7–13.6	5.6–14.8	5.9–16.9
Neutrophils ($\times 10^3$/mm^3)		1.4–4.6	3.6–10.1	3.8–12.3	3.9–13.1
Lymphocytes ($\times 10^3$/mm^3)		0.7–4.6	1.1–3.6	0.9–3.9	1.0–3.6
Monocytes ($\times 10^3$/mm^3)		0.1–0.7	0.1–1.1	0.1–1.1	0.1–1.4
Transferrin (mg/dL)		200–400	254–344	220–441	288–530

Coagulation Studies					
International Normalized Ratio	INR	0.9–1.04	0.89–1.05	0.85–0.97	0.80–0.94
Fibrinogen (mg/dL)		233–496	244–510	291–538	373–619
Partial thromboplastin time, activated (sec)	aPTT	26.3–39.4	24.3–38.9	24.2–38.1	24.7–35.0
Prothrombin time (sec)	PT	12.7–15.4	9.7–13.5	9.5–13.4	9.6–12.9
Blood Chemistry					
Alanine transaminase (U/L)	ALT	7–41	3–30	2–33	2–25
Aspartate transaminase (U/L)	AST	12–38	3–23	3–33	4–32
Bilirubin, total (mg/dL)	T. Bili	0.3–1.3	0.1–0.4	0.1–0.8	0.1–1.1
Bilirubin, conjugated (mg/dL)		0.1–0.4	0–0.1	0–0.1	0–0.1
Creatinine (mg/dL)		0.5–0.9[c]	0.4–0.7	0.4–0.8	0.4–0.9
Lactate dehydrogenase (U/L)	LDH	115–221	78–433	80–447	82–524
Potassium (mEq/L)	K	3.5–5.0	3.6–5.0	3.3–5.0	3.3–5.1
Sodium (mEq/L)	Na	136–146	133–148	129–148	130–148
Blood urea nitrogen (mg/dL)	BUN	7–20	7–12	3–13	3–11
Uric acid (mg/dL)		2.5–5.6[c]	2.0–4.2	2.4–4.9	3.1–6.3
Endocrine/Metabolic					
Thyroid-stimulating hormone (μIU/mL)	TSH	0.34–4.25	0.60–3.40	0.37–3.60	0.38–4.04
Thyroxine, free (ng/dL)	fT_4	0.8–1.7	0.8–1.2	0.6–1.0	0.5–0.8
Vitamins/Minerals					
Vitamin B_{12} (pg/mL)		279–966	118–438	130–656	99–526
Vitamin D, 25-hydroxy (ng/mL)		14–80	18–27	10–22	10–18
Renal Function					
24-hour protein excretion (mg/24 hr)		< 150	19–141	47–186	46–185

[a] Values are based on populations with and without iron supplementation.
[b] Values for females 19–65 years old.
[c] Values for females only.

Based on Cunningham FG. Appendix B: laboratory values in normal pregnancy. In: Queenan JT, Hobbins JC, Spong CYT, eds. *Protocols for High-Risk Pregnancies: An Evidence-Based Approach.* 5th ed. Oxford, UK: Blackwell; 2010:587-595.[48]

Tests for Fetal Aneuploidy

Screening and diagnostic tests for fetal aneuploidy are offered to all pregnant women. Screening or diagnostic tests can be performed in the first or second trimester. First-trimester screening tests include a combination of ultrasound and blood tests that detect maternal biochemical markers for aneuploidy. Diagnostic procedures include chorionic villus sampling, which is performed in the first trimester; amniocentesis, which is performed in the second trimester; and maternal cell-free DNA, which can be performed at any time during pregnancy. Each test has different capabilities and limitations that are reviewed in the *Genetics* chapter. For example, cell-free DNA testing detects trisomies 21, 18, and 13, but does not detect other chromosomal abnormalities that are identified by the tests used to examine placental tissue or amniotic fluid obtained via chorionic villus sampling (CVS) or amniocentesis. Because CVS and amniocentesis have procedure-related risks, many women chose a sequential approach whereby they first obtain a screening test and then undergo diagnostic testing only if the screening test reveals a high risk.[43]

Carrier Screening

Carrier screening tests are reviewed in the *Genetics* chapter. In brief, all pregnant women are offered carrier testing for cystic fibrosis, hemoglobinopathies, and spinal muscular atrophy if these tests have not been performed previously.[47] The chosen strategy for selected carrier screening depends on factors including background population prevalence and the woman's race, ethnicity, and family history.[47]

First-Trimester Ultrasound

In the United States, one routine standard ultrasound in the first or early second trimester is recommended for all pregnant women.[49,50] In practice many women receive a first trimester limited ultrasound to confirm a viable pregnancy and establish gestational age and a standard ultrasound at 18–20 weeks for the purpose of evaluating fetal anatomy. Midwives may perform a first-trimester ultrasound if trained and certified or privileged to do so.[51] The standard ultrasound that occurs at 18 to 20 weeks is performed by a clinician who is trained and credentialed to conduct this examination. Prior to scheduling or performing an ultrasound, the midwife must review the indications, benefits, and risks with the woman.

Counseling and Anticipatory Guidance

Anticipatory guidance and instruction at the end of an initial prenatal visit can encompass many health education topics, but always includes the following components:

- Instruction about signs that may indicate a complication requiring medical evaluation (**Table 21-10**)
- Practices for health maintenance during pregnancy, including counseling about alcohol,

smoking, and substance abuse
- An orientation to the plan of care and timing of return visits
- Information about how to contact the midwife and the roles of all members of the healthcare team
- Expected costs to the patient of prenatal care and birth

Table 21-10	Signs of Pregnancy Complications
Contractions or uterine cramping or sudden, sharp, or continuing abdominal cramping or pain	
Decreased fetal movement	
Dysuria, hematuria, or inability to void	
Facial edema, edema of the hands, or unilateral lower-extremity edema	
Fever or chills	
Headache that is persistent or severe, and that is unrelieved by over-the-counter pain relievers	
Leaking of fluid or abnormal vaginal discharge	
Nausea and vomiting, unable to keep fluids down or to void	
Visual changes (e.g., blurring of vision, dizziness, spots before eyes)	
Vaginal bleeding	

All pregnant women should be screened for intimate-partner violence and mental health conditions during an early prenatal visit, but the timing for these screens should be individualized.

Diagnosis and Plan

The primary assessment at the end of the initial prenatal examination is a pregnancy diagnosis. Examples include “pregnancy of unknown location,” “viable intrauterine pregnancy,” or “threatened spontaneous abortion.” The assessment should include the estimated gestational age and dating criteria used.

Other assessments following the first prenatal visit include those related to the woman’s prepregnant health that may confer risk to the current pregnancy. The woman’s health record should also list any common discomforts of pregnancy for which a management plan was required. Finally, all tests ordered are recorded in the health record. Critical components of the assessments to be documented following the first prenatal visit are listed in **Table 21-11**.

Table 21-11	Critical Elements of Documentation Following the First Prenatal Visit
Obstetric Review of Systems	
First trimester:	
Vaginal bleeding	
Severe cramping or pelvic pain	
Excessive nausea and vomiting	
Headache	

Dysuria

Fever

Second/third trimesters:

The presence of expected fetal movement appropriate for gestational age

Signs and symptoms of preterm labor: uterine cramping or contractions, leaking of fluid, or vaginal bleeding

Maternal Vital Signs

Blood pressure should always be documented. If blood pressure is elevated, documentation will include the subjective symptoms of preeclampsia (headache, visual changes, and epigastric pain).

Other vital signs, including temperature, respiratory rate, and oxygen saturation, should be reported in the presence of maternal constitutional symptoms.

Fetal Well-Being

Detection of fetal heart tones: Documented at every prenatal visit after 12 weeks' gestation

Assessment of fetal growth relative to that expected for gestational age

Determination of fetal presentation > 36 weeks' gestation.

Management Plan

Diagnostic and therapeutic plans for each problem identified

Follow-up plan

Consultation (if indicated), including the name of the provider consulted, the information discussed with the consultant, and any recommendations provided

Interval Prenatal Visits

Before seeing a woman for an interval prenatal care visit, the midwife reviews the woman's record to determine the gestational age of the fetus; the woman's EDD; any significant problems that have been noted on a problem list; medications, treatments, and dietary interventions; results of laboratory tests or ultrasound assessments; and need for follow-up testing. The essential components of an interval prenatal visit are presented in **Table 21-12** and the skills for performing the physical examination are reviewed in Appendix 21A.

Table 21-12 **Essential Components of Routine Interval Prenatal Visits**

History
Obstetric review of systems:
Fetal movement: Appropriate for gestational age?
Vaginal bleeding or leaking of fluid
Uterine contractions (frequency, regularity, strength)
Interval medical history:
New bodily changes, symptoms, or concerns
Status of previous problems and response to treatment
Interval social history:
Social support or concerns
Safety at home
Preparation for labor and baby
Medications, including vitamins and OTC products
Physical Examination
Weight and interval weight gain
Blood pressure
Urine dipstick to test for proteinuria, specific gravity, or leucocytes if indicated
Obstetric abdominal examination:
Fundal height in centimeters
Fetal heart tones
Fetal presentation after 36 weeks' gestation
Third trimester: Assess for inverted nipples
Health Screening
Intimate-partner violence screening at least once every trimester
Depression screening
Alcohol and drug use
Laboratory Assessments
16–20 weeks' gestation: Second-trimester Down syndrome screening test (triple or quad screen) offered
24–28 weeks' gestation:
Gestational diabetes screen
Antibody screen if Rh negative and subsequent RhoGAM therapy for Rh-negative women
35–37 weeks' gestation:

Group B *Streptococcus* screening culture
Repeat STI screening (gonorrhea, chlamydia, syphilis, HIV, hepatitis B) if indicated
Ultrasound Assessments
18–20 weeks' gestation: Fetal anatomy survey
≥ 36 weeks' gestation: Confirm fetal presentation, if uncertain

Abbreviations: HIV, human immunodeficiency virus; OTC, over-the-counter; STI, sexually transmitted infection.

Schedule for Interval Prenatal Visits

Schedules for subsequent prenatal visits vary between practices, and based on the individual woman's needs or risks that develop during her pregnancy. Traditionally, interval prenatal visits have been scheduled monthly in the first and second trimesters, every 2 weeks between 28 to 36 weeks' gestation, and weekly thereafter.

Group Prenatal Care

In the last decade, alternatives to individual one-on-one prenatal visits have been developed, including group prenatal care. The best-studied group prenatal care model is CenteringPregnancy, which has been widely adopted. CenteringPregnancy differs from the traditional individual care model in that it combines the typical elements of a prenatal visit risk assessment with childbirth education and peer support.

CenteringPregnancy utilizes a standardized approach and group process that focuses on the creation of a self-empowering environment.[52] Groups of 8 to 12 women or couples at approximately the same gestational age, usually starting at 12 to 16 weeks' gestation, are formed. The group meets monthly for 4 months, and then biweekly, and once in the early postpartum period, for a total of ten 90-minute sessions. Additional individual examination room visits are scheduled as needed.

The effectiveness of group prenatal care has generally been investigated through observational, small studies, with varied results. Overall, group prenatal care—and in particular CenteringPregnancy—is associated with high rates of patient satisfaction.[52,53] Data on the effects of this model on perinatal outcomes are still emerging. Some studies have found lower rates of adverse outcomes such as preterm birth, low birth weight, and neonatal intensive care unit admissions, along with higher rates of breastfeeding initiation, later cervical dilation on admission, and improved pregnancy-weight management.[54-61] Nevertheless, these findings have not been replicated in all studies.[62-64] Notably, larger systematic reviews, which did not account for possible a priori risks within the population of women included in the studies, have not confirmed the links between group prenatal care and improved maternal or newborn health outcomes.[65-68]

Although more study is needed to determine the effectiveness of CenteringPregnancy in different populations, this prenatal care model is clearly as good as individual care. In some populations, it may even have significant advantages—especially in at-risk populations such as women who experience racial/ethnic disparities and adolescents.[54-60] For example, although

outcomes have not been published, the Akwesasne Mohawk Nation has integrated traditional Mohawk coming-of-age ceremonies and traditions into CenteringPregnancy prenatal care in the United States and Canada to reinforce social connections, customs, and values consistent with Mohawk traditions.

Interval History

The initial conversation and review of interval history is designed to address any questions, discomforts, or complications that the woman may have experienced since the last visit. In addition to the standard components of the interval prenatal examination, the physical examination may include a targeted examination to assess specific symptoms. For example, a speculum examination might be performed to assess a vaginal discharge or determine whether membranes have ruptured. A bimanual examination might be performed to evaluate cervical ripening or to perform membrane sweeping after 37 weeks' gestation.

Interval Physical Examination

The interval physical examination includes maternal weight, blood pressure, and abdominal examination, as detailed in Appendix 21A.

Screening, Laboratory Tests, and Adjunctive Studies

In many practices, a voided urine specimen is obtained at each prenatal visit to detect proteinuria, glucosuria, or signs of asymptomatic bacteriuria. However, the urine dipstick test —especially in the absence of symptoms or risk factors such as hypertension—is not a reliable means of detecting asymptomatic bacteriuria[69] or preeclampsia,[69,70] and is of questionable benefit in early detection of gestational diabetes. Most authors have advocated for discontinuation of such testing as a part of regular prenatal care.

Diabetes Screening

Gestational diabetes mellitus (GDM) is a condition of carbohydrate intolerance that develops during pregnancy. Traditionally, any woman who was found to have glucose intolerance during pregnancy was given the diagnosis of GDM. In recent years, however, the increasing rates of obesity in the general population have resulted in more women of childbearing age who have type 2 diabetes. Some women may be initially diagnosed with this condition during pregnancy. Because the prevalence of type 2 diabetes in pregnancy has increased, recommendations for screening and diagnosis have recently changed: Women who are diagnosed with diabetes in the first trimester are now considered to have type 2 diabetes, whereas women who are first diagnosed in the second or third trimester are classified as having GDM.[71]

Overall, approximately 6% to 9% of pregnant women have diabetes during pregnancy, with an estimated 90% of these women having GDM.[71] The prevalence of GDM is higher among Hispanic, African American, Native American, Asian, and Pacific Islander women.[72] GDM is subcategorized as either A1 GDM, in which alterations in diet result in euglycemia, or A2

GDM, in which medication is needed to maintain euglycemia.

Women who have GDM have an increased risk for developing preeclampsia, developing polyhydramnios, and having a cesarean section; their infants have an increased risk for macrosomia, shoulder dystocia, hypoglycemia, and hyperbilirubinemia. These risks increase in a dose–response manner as maternal blood glucose values increase. Importantly, GDM is a significant risk factor for developing type 2 diabetes later in life. Approximately 70% of women with GDM will develop type 2 diabetes within 22 to 28 years after the index pregnancy.[73] Among Latina women, approximately 60% will develop type 2 diabetes within 5 years of the index pregnancy.[74]

There are two time frames for screening for diabetes in pregnancy: early (first trimester or at entrance to care) and second trimester (24–28 weeks). Early screening is primarily done to identify women with undiagnosed pregestational diabetes or prediabetes.[75] Second-trimester screening is used to identify women with GDM.[75,76] **Figure 21-2** presents an algorithm for diabetes screening in pregnancy.[75,77]

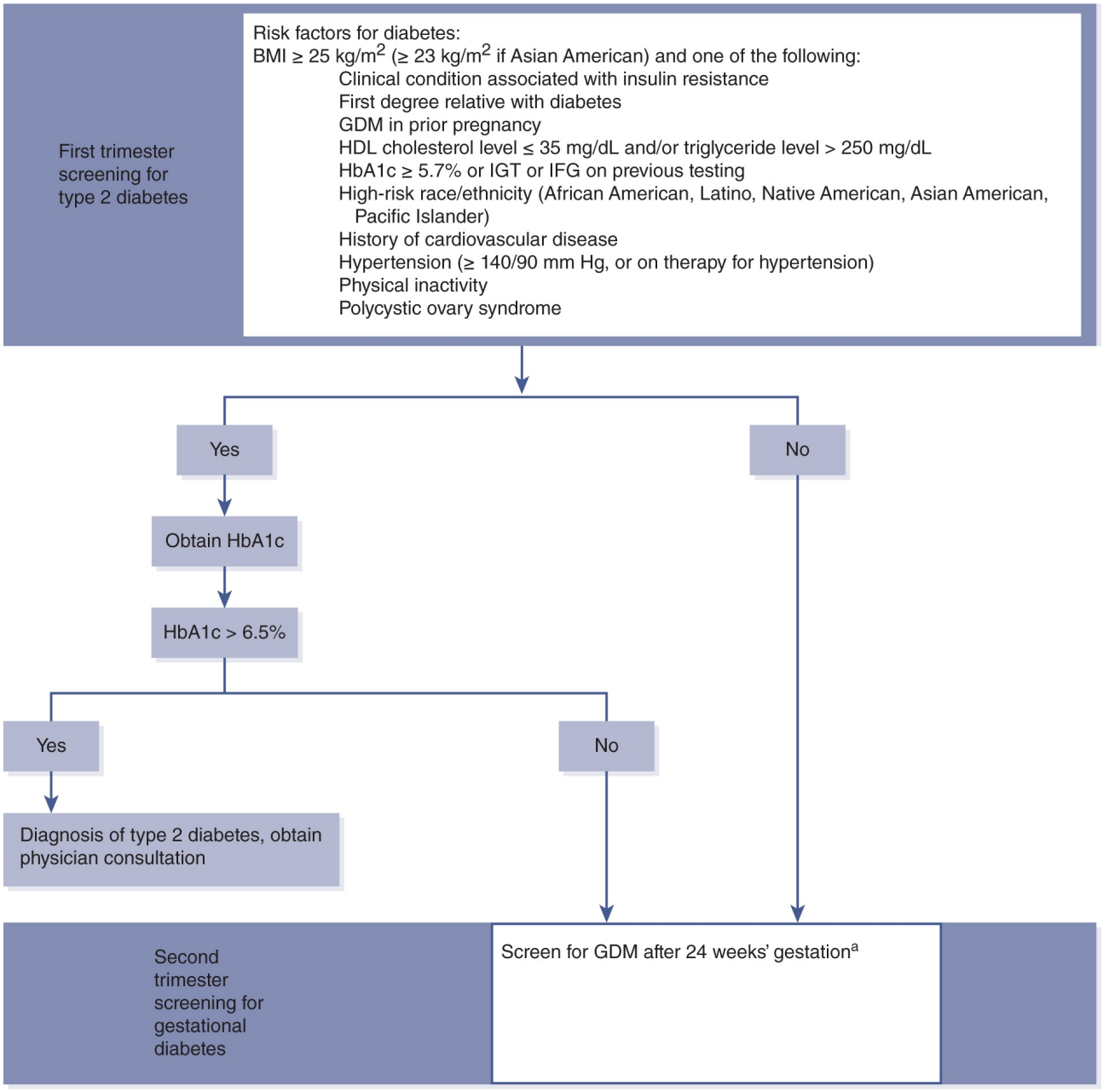


Figure 21-2 Screening and diagnosis of diabetes during pregnancy.

Abbreviations: BMI, body mass index; GDM, gestational diabetes mellitus; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; IFG, impaired fasting glucose; IGT, impaired glucose tolerance.

[a] Screening can be done via one of the following:

1. One-step 75-g glucose load with blood glucose obtained 2 hours later, or via 2-step process that includes an initial 50-g glucose load with blood glucose obtained 1 hour later.

2. A 3-hour glucose tolerance test is obtained if the blood glucose following the 50-g load is > 130 or 140 mg/dL.

Based on American Diabetes Association. 2. Classification and diagnosis of diabetes. *Diab Care*. 2016;39(suppl 1):S13-S22[75]; Committee on Practice Bulletins—Obstetrics. Practice Bulletin No. 137: gestational diabetes mellitus. *Obstet Gynecol*. 2013;122(2 pt 1):406-416.[77]

Early Screening for Undetected Type 2 Diabetes. Early screening for diabetes is recommended by the American Diabetes Association (ADA) and American College of Obstetricians and Gynecologists (ACOG) for women with risk factors for diabetes, as noted in **Figure 21-2**. The ADA recommends testing women with obesity who have two additional risk factors.[76] The ACOG criteria suggest early screening based on one risk factor.[77] The diagnosis of type 2 diabetes is based on the hemoglobin A1c (HbA1c) value obtained.

Second-Trimester Screening for Gestational Diabetes Mellitus. All professional associations recommend screening for GDM after 24 weeks' gestation, but their recommendations sometimes differ with regard to (1) who should be screened (risk based versus universal) and (2) which test to use. The first consideration is the indication—that is, screening on the basis of risk or universal screening for all pregnant women. The problem with risk factor screening is that 90% of the general population has one or more risk factors. Thus, the overwhelming majority of women would meet the criteria for early screening, which is why the U.S. Preventive Services Task Force (USPSTF) and ACOG recommend universal screening.[77],[78] Nonetheless, it is important to remember that women who do not have risk factors for GDM and who want to decline screening should be counseled that their choice is a safe one. Characteristics of women who are at low risk for GDM are listed in **Table 21-13**.

Table 21-13	**Women Who Are at Low Risk for Gestational Diabetes**
Younger than 25 years	
Not a member of one of the following racial or ethnic groups:	
African American	
South or East Asian	
Hispanic	
Native American	
Pacific Islands descent	
No known diabetes in a first-degree relative	
Body mass index of 25 kg/m^2 or less	
No history of glucose intolerance	
No history of adverse obstetric outcomes associated with gestational diabetes (e.g., stillbirth, macrosomia)	

Modified with permission from Moyer VA; U.S. Preventive Services Task Force. Screening for gestational diabetes mellitus: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2014;160:414.[78]

The best testing procedure for screening and diagnosis of GDM is also a continuing controversy. All strategies involve an oral glucose test. The strategy recommended by ACOG and the National Institutes of Health is a two-step process that initially involves a 50-gram, 1-hour glucose challenge. With this test, the woman ingests 50 grams of glucose and a blood glucose level is obtained 1 hour later.[77],[79] If this blood glucose level is elevated above 130 mg/dL or 140 mg/dL, a 3-hour glucose tolerance test (GTT) is performed, in which glucose levels are obtained before (fasting) and at 1, 2, and 3 hours after ingesting 100 grams of glucose. The results of the 3-hour glucose test determine the diagnosis.

The International Association of Diabetes and Pregnancy Study Groups (IADPSG) recommends a universal, single, one-step strategy using a 75-gram dose of glucose with a blood glucose level obtained 2 hours later. This testing strategy results in a significant increase in the number of women diagnosed with GDM.[80] As yet, there is no international consensus on which strategy to use. A concern with any of these approaches is the lack of evidence showing the benefit of the increased diagnoses.

Some women may prefer to eat a natural meal or food instead of ingesting the glucose drink. Two studies have evaluated glucose loading in the form of a prespecified number of jelly beans and found that this approach had similar sensitivity, specificity, and positive predictive value as that using the 50-gram glucose drink.[81,82] The primary reason that "confectionary screening" is not recommended is that jelly beans are metabolized into circulating glucose at differential rates, and it is unknown exactly how that glucose load affects blood glucose levels exactly 1 hour later.

Rh Alloimmunization Prevention

The treatment of Rh alloimmunization is a success story of the twentieth century. The Rhesus factor was first identified in the late 1930s, and Rh incompatibility was identified shortly thereafter as the etiology for large numbers of fetal deaths and stillbirths. In the late 1960s, an anti-D serum was approved.

Red blood cells have many different proteins on the cell membrane surface that are antigenic. Some of these proteins are controlled by a single gene locus. To date, more than 30 blood group system genes have been identified, along with their corresponding red cell surface proteins. They have been assigned a variety of names mostly based on when they were discovered and who discovered them. Because these blood groups are polymorphic, there are approximately 200 genetic variations. The polymorphic subtypes are designated with a second letter (e.g., RhD, RhC, or RhE). The A, B, O, and Rh red blood cell antigens are the most important clinically because they are highly immunogenic. An individual who is Rh (D) positive has the Rh (D) antigen on the membrane of his or her red blood cells. Conversely, a person who is Rh (D) negative does not have the Rh (D) antigen on his or her red blood cell membranes and, therefore, may create antibodies to Rh (D)-positive cells when exposed to Rh (D)-positive blood—a phenomenon called *alloimmunization.*

Fetal blood has several opportunities to mix with maternal blood during pregnancy and birth, although such mixing is not common during the prenatal period. During the third stage of labor, fetal blood commonly escapes into the maternal circulation as the placenta separates from the decidua. For a woman who is Rh (D) negative and pregnant with a Rh (D)-positive fetus, physiologic events that expose her circulation to fetal blood can cause her to develop immunoglobulin G (IgG) antibodies to the Rh (D) antigen. It is estimated that approximately 17% of women will become alloimmunized secondary to some fetomaternal blood transfer during birth.[83] The anti-Rh (D) IgG–type antibodies produced are small in size. During subsequent pregnancies, they can easily cross the placenta and attach themselves to any antigens found on Rh (D)-positive red blood cells of a fetus, causing hemolysis, erythroblastosis fetalis, and stillbirth.

The ABO blood groups can also be different between the mother and the fetus, although ABO antibodies are not likely to damage fetal red blood cells if the mother has a previous exposure to a different blood type and has created antibodies to the A, B, or AB blood groups. The A, B, and AB blood group proteins are not expressed on red blood cells as profusely as are Rh proteins, and are not well developed at birth compared to the Rh antigen. Antibodies to A, B, and AB are usually of the immunoglobulin M (IgM) class; they are larger in size compared to the anti-D IgG antibodies, so they are not able to cross the placenta. Furthermore, because many other cell types express the A, B, and AB proteins, antibodies to them are less likely to specifically attack red blood cells in enough numbers to cause harm.

Today Rh alloimmunization in pregnant women and hemolytic disease of the fetus or newborn are rarely seen, primarily because women who are Rh (D) negative are given anti-D immune globulin prophylaxis in the third trimester of pregnancy and after birth if the newborn is Rh (D) positive. Anti-D immune globulin (RhoGAM) is extracted from the plasma of individuals who have high titers of anti-D immune globulin antibodies. Since 1985, all plasma used for anti-D immune globulin production has been tested for viruses that can cause infection.[83] The anti-D immune globulin has a unique mechanism of action. A small amount of anti-D IgG attaches to any fetal cells that are present in maternal circulation and masks them, thereby preventing the maternal immune system from developing permanent antibodies. The amount of anti-D immune globulin is small enough that it does not induce a maternal antibody response and is removed from maternal circulation in approximately 12 weeks. Thus, every subsequent pregnancy becomes a “first pregnancy” in terms of Rh (D) antibody development.

Today, women are screened for Rh (D) status at the initial prenatal visit. If a woman is found to be RH negative, an antibody titer is usually ordered in the second trimester to ascertain whether she has become alloimmunized. If this titer is negative, she is given anti-D immune globulin at 28 weeks’ gestation.[83] Supportive data are limited, but some sources recommend a second prenatal dose of anti-D immune globulin if more than 12 weeks elapses between the second-trimester dose and the onset of labor (although this is not common practice).[83] The most likely time for alloimmunization to occur is following birth, however, and women who are Rh (D) negative should receive another dose of the agent within 72 hours after giving birth if the newborn is Rh positive. Anti-D immune globulin is also given after a first- or second-trimester pregnancy loss or termination to prevent alloimmunization.[83]

Special situations warranting additional administration of anti-D immune globulin and other antigens that can cause hemolytic disease in the fetus or newborn are discussed in the *Pregnancy-Related Conditions* chapter.

Group B Streptococcus Colonization

Group B *Streptococcus* is a gram-positive coccus that normally inhabits the gastrointestinal system and genital tract of approximately 15% to 40% of pregnant women. Colonization with GBS is generally asymptomatic and not pathogenic in women. In contrast, newborns colonized with GBS can develop severe sepsis. The newborn is most likely to become colonized at birth during passage through the vagina. Although rare in absolute numbers (0.25 case per 1000 live births), GBS is the leading cause of early-onset newborn sepsis in the United States.[84,85]

The Centers for Disease Control and Prevention (CDC) recommends universal screening for maternal carrier status performed between 35 and 37 weeks' gestation, followed by intrapartum prophylaxis for women who have a vaginal/rectal culture that is positive for GBS. Vaginal colonization can be intermittent or persistent, so women are screened during each pregnancy. A woman who was GBS positive in a prior pregnancy may have no vaginal colonization with this organism in a subsequent pregnancy. If GBS is identified in a urine culture at any time during a pregnancy, it is a sign of heavy colonization. Such women are offered intrapartum prophylaxis, without performing a confirming culture, at 35 to 37 weeks' gestation.[85]

The collection of a culture for GBS testing is a simple procedure, and a speculum is not needed. The woman can collect the specimen herself if desired. The vaginal introitus is swabbed, and then the swab is inserted into the anus.[85] Swabbing just within the external third of the vagina (not the cervix) and then the rectum is necessary to swab the areas most likely to have a large inoculum. Group B *Streptococcus* is not easy to culture, and cultures are more reliable if a large inoculum is obtained. If the GBS culture is positive, the midwife provides the woman with information about intravenous penicillin prophylaxis in active labor or after rupture of membranes. Birth centers and home birth midwives are equipped to administer intravenous GBS prophylaxis in those sites.

Second-Trimester Ultrasound Examination

A standard fetal anatomy survey, typically performed between 18 and 20 weeks' gestation, is recommended for all pregnant women. This survey is intended to identify fetal and placental structural abnormalities and confirm gestational age (if a first-trimester ultrasound was not performed). By 18 weeks' gestation, fetal organs are formed and large enough that ultrasound can detect anatomic abnormalities, particularly in the heart and brain. This ultrasound examination is generally performed prior to the gestational limit for termination of pregnancy, in the event that anomalies are detected. Abnormal ultrasound findings are reviewed in the *Pregnancy-Related Conditions* chapter.

Gestational Weight Gain and Nutrition

Individuals who have a healthy diet before becoming pregnant need only subtle changes to optimize nutrition in pregnancy. Energy requirements increase by approximately 200, 300, and 400 calories per day in the first, second, and third trimesters, respectively[86]—an increase that is equivalent to the addition of one healthy snack per day. In light of emerging epigenetic research findings that relate in utero nutrient exposure to health outcomes later in life, specific, accurate nutrition counseling during pregnancy is more important now than ever.[87]

Weight Gain

Gestational weight gain is just one aspect of a woman's nutritional status. Indeed, routine weight measurements during pregnancy are not the standard of care in all developed nations.[86] The United States is one of the few developed nations in which weight gain is routinely tracked over the course of pregnancy.

A relationship exists between both insufficient and excess gestational weight gain and adverse outcomes for both the woman and the fetus. Pregestational weight and subsequent gestational weight gain affect offspring weight gain and adiposity. In turn, birth adiposity affects adult weight and long-term health.[88] Inadequate weight gain is associated with low birth weight, small for gestational age infants, preterm birth, increased perinatal mortality, and failure to establish breastfeeding.[86,89] Conversely, infants of women who gain excessive weight are at increased risk of being large for gestational age, hypoglycemia, polycythemia, low Apgar scores, seizures, and birth trauma associated with difficult birth.[86] For women, excessive gestational weight gain is associated with cesarean section and postpartum weight retention. Long-term outcomes associated with excessive gestational weight gain may include metabolic syndrome, type 2 diabetes, and cardiovascular disorders.[86]

Guidelines for gestational weight gain in pregnancy have changed over the years. In 1990, the Institute of Medicine (IOM) issued pregnancy weight gain guidelines aimed at ensuring adequate weight gain to prevent low birth weight. At the time, the weight gain recommended by IOM was higher than the 12-pound weight gain typically recommended in clinical practice. Since then, obesity and excessive weight gain in pregnancy have become public health concerns in the United States. In 2009, IOM revisited its recommendations in light of the evidence accumulated since the early 1990s, with a particular focus on the role of retention of gestational weight gain in the development of long-term obesity in women, and issued revised gestational weight gain guidelines (**Table 21-14**).[88]

Table 21-14	Recommended Gestational Weight Gain for Women with a Singleton Pregnancy[a]	
Pregestational Weight Category: BMI (kg/m^2)	**Recommended Total Weight Gain: Pounds (kg)**	**Second- and Third-Trimester Rate of Weight Gain: Mean (Range)**
Underweight: < 18.5	28–40 (12.5–18)	1 (1–1.3) lb/wk 0.51(0.44–0.58) kg/wk

Normal weight: 18.5–24.9	25–35 (11.5–16)	1 (0.8–1) lb/wk 0.42(0.35–0.50) kg/wk
Overweight: 25.0–29.9	15–25 (7–11.5)	0.6(0.5–0.7) lb/wk 0.28(0.23–0.33) kg/wk
Obese: ≥ 30.0	11–20 (5–9)	0.5(0.4–0.6) lb/wk 0.22(0.17–0.22) kg/wk

Abbreviation: BMI, body mass index.

[a] Calculations assume a mean first-trimester weight gain of 2 kg (range: 1–3 kg) for women who are underweight, normal weight, and overweight. The assumed first-trimester weight gain for women who are obese is 1.5 kg (range: 0.5–2 kg).

Modified with permission from Rasmussen KM, Yaktine AL, eds. *Weight Gain During Pregnancy: Reexamining the Guidelines.* Washington, DC: National Academies Press; 2009. Available at: http://www.ncbi.nlm.nih.gov/books/NBK32815. Accessed October 9, 2017.[88]

The recommended gestational weight gain for women differs based on pregestational body mass index (BMI).[90] The IOM recommendations are for the general population in the United States; subpopulations—such as adolescents, women who smoke, and women with chronic illness—may require individualized weight gain targets.[91,92] The pattern of gestational weight gain is also different by race and ethnicity. For example, Hispanic, black, and Asian normal-weight women are more likely to experience inadequate gestational weight gain compared to white women.[86,93-95]

Persons are provided with information about their BMI category and target prenatal weight gain using the IOM recommendations at the first prenatal visit. Weight gain is then assessed and nutrition counseling reviewed at subsequent prenatal visits. Charting weight gain on a graph gives both the woman and the prenatal care providers a pictorial representation for easy visualization. Although a composite weight gain graph is often available in prenatal charts, weight gain graphs that show the mean and range specific for each BMI category are available.[90] (See the Resources section at the end of this chapter for weight chart sources.) Encouraging the pregnant woman to complete forms such as the "Starting the Conversation" or REAP questionnaires (reviewed in the *Nutrition* chapter), and assessing their results, can be a rapid method to gain valuable information about the woman's current dietary patterns.

Nutrition

The recommended Daily Reference Intakes for macronutrients and micronutrients for women during pregnancy are summarized in the *Nutrition* chapter. Diet can play a powerful role in maintaining a healthy pregnancy, and prenatal care is an ideal time to start the conversation about a healthy dietary pattern.[96] An increasing body of evidence supports the Barker hypothesis, which states that a woman's diet during pregnancy has effects on the fetus that are important postnatally and throughout her child's entire life, including genetic predispositions for some chronic diseases.[97]

Frequency of meals also plays a role during pregnancy. A substantial body of research supports the recommendation that pregnant women eat three meals a day and at least two snacks. Some pregnant women will want to observe their religion's periods of fasting, even

though Catholicism, Judaism, and Islam all customarily exempt a pregnant woman from any obligation to fast. As part of prenatal care, the midwife elicits this information and provides teaching as required. **Table 21-15** summarizes dietary recommendations during pregnancy.[96,98]

Table 21-15 Summary of Dietary Advice for All Pregnant Women

Supplements
Take folic acid in a multivitamin at least 1 month prior to conception and for the first 3 months after conception to prevent neural tube defects.
Diet
Gain the recommended weight for BMI by eating the following:
Three meals a day and two snacks to avoid prolonged periods of fasting
Five servings of fruits and vegetables, at minimum, per day
Whole-grain carbohydrates; limit food with high sugar content like desserts, juices, and sodas
Adequate amounts of protein, choosing more plant-based sources such as nuts and beans
At least two servings of best-choices fish weekly or one serving of good-choices fish:
Examples of best choices: catfish, clam, crab, flounder, pollock, salmon, sardines, shrimp, sole, trout, whitefish
Examples of good choices: bluefish, carp, Chilean sea bass, halibut, snapper, tuna (albacore, white tuna, canned and fresh or frozen, yellowfin)
Limit canned tuna to twice a week
Drink 8–10 glasses of water per day.
Limit caffeine to 200 mg/day (2 cups of coffee or 4 cups of black tea)
Avoid all alcoholic drinks
Limit sweetened drinks
Ensure adequate intake of these micronutrients:
Vitamin A as beta-carotene; limit food sources with preformed vitamin A such as liver (< 4 oz/week) or cod liver oil
Vitamin D from exposure to sunlight (amount will vary by geography, BMI, and skin pigmentation); if not feasible, consider supplementation with vitamin D_3 (1000–4000 IU depending on intake of vitamin D–fortified dairy products and vitamin D serum levels)
Folic acid through a multivitamin supplement
Women adhering to strict vegan diets: Take vitamin B_{12} supplements or consume vitamin B_{12}–fortified products
Iodine through diet (dairy and fish) or a multivitamin with iodide (preferably as potassium iodide)
Iron through diet, multivitamin, or additional low-dose supplement if anemic
Calcium through diet, with supplementation suggested for women at risk for preeclampsia
Choline-rich food sources such as meat, poultry, and eggs, since this nutrient is not typically included in prenatal vitamins
Foods to Avoid
Soft cheeses unless they clearly state they are made from pasteurized milk, including brie, feta, camembert, blue-veined cheeses, and Mexican-style cheeses such as queso blanco, queso fresco, and queso panela
Uncooked meats or refrigerated pâtés or meat spreads
Raw eggs, cookie dough
Raw fish
Mercury-concentrating fish: king mackerel, marlin, orange roughy, shark, swordfish, tilefish (Gulf of Mexico), tuna (bigeye)

Deli meat salads or smoked seafood
Raw sprouts
Raw unpasteurized fruit juice, raw milk, or unpasteurized dairy products
Refrigerated perishable foods that have not been consumed in 2–3 days
Food Handling
Wash cutting boards and equipment used to cut raw meat with hot soap and water
Wash and peel raw fruits and vegetables before eating
Wash hands after handling hot dogs, luncheon meats, or deli meats
Food Processing
Cook all deli meats, hot dogs, smoked meats, pâtés, and luncheon meats to steaming hot before eating
Cook meat and eggs to 71.1°C (160°F)
Cook seafood to 62.8°C (145°F)

Abbreviation: BMI, body mass index.

Based on U.S. Food and Drug Administration. Food safety for moms-to-be. Available at: https://www.fda.gov/food/foodborneillnesscontaminants/peopleatrisk/ucm081785.htm. Accessed July 4, 2017.[98]

Modified with permission from Barger MK. Maternal nutrition and perinatal outcomes. *J Midwifery Womens Health.* 2010;55(6):502-511.[96] © 2010, with permission from Wiley.

Vitamin and Mineral Supplementation

Prenatal vitamins are multivitamin supplements formulated to provide the vitamins and minerals needed most by pregnant women. Although prenatal vitamins are one of the hallmarks of prenatal care, the actual value of vitamin and micronutrient supplements for pregnant women is more nuanced and complex. First, a balanced diet generally provides all the nutrients needed for health during pregnancy. Unfortunately, a majority of the women in the United States do not consume a diet that provides all the vitamins and micronutrients needed during pregnancy. The specific vitamins and micronutrients that have documented adverse effects on fetal development if present in insufficient amounts in the diet are folic acid, iron, and iodine.[96]

Folic Acid. Supplemental folic acid is recommended for all pregnant women.[99] Supplemental folic acid started in the preconception period and taken through the first trimester lowers the risk of neural tube defects (NTDs) by approximately 30% to 70% (odds ratio [OR], 0.67; 95% confidence interval [CI], 0.58–0.77), with the amount of decrease depending on the study conducted and the amount of folic acid used by study participants.[100] The neural tube closes between 4 and 6 weeks after the LMP; consequently, women are advised to start supplements in the preconception period if possible, and at the first prenatal visit if not already taking a prenatal supplement. The recommended dose is 400 micrograms per day for women who do not have an a priori risk for neural tube defects. Women who had a previous pregnancy complicated by an NTD are advised to take 4 milligrams of folic acid per day starting 1 month prior to conception and through the first 4 months of pregnancy.

Iron. Iron is the single most difficult nutrient to obtain via the diet during pregnancy, and iron

intake needs are approximately doubled during pregnancy.[101-103] However, unlike folic acid supplementation, routine iron supplementation is controversial. The CDC recommends routine iron supplementation, but other organizations suggest that supplementation should be started only for women who do not have normal hemoglobin values.[101-103] Iron supplementation increases plasma hemoglobin levels but does not affect overall maternal and neonatal outcomes unless iron is used specifically to treat anemia during pregnancy. Moreover, iron supplements frequently cause gastrointestinal distress.

Two types of iron exist: heme iron, which is found in meat, and non-heme iron, which is found in plants and, to a smaller extent, dairy products. Iron is absorbed via the mucosal cells in the jejunum. Approximately 20% to 30% of heme iron is absorbed without being affected by other dietary factors. Non-heme iron is more difficult to absorb; only approximately 2% to 10% is absorbed, and an acidic environment in the gastrointestinal tract is necessary to reduce the ferric iron to ferrous iron for absorption. Tannins found in tea, caffeine, and whole grains can adversely affect non-heme iron absorption. Absorption increases substantially in pregnancy, when more iron is used and iron stores are low.

The absorption of iron in prenatal vitamins can depend on the amount of calcium in the vitamin. For example, more iron may be absorbed from a vitamin with a lower iron content without calcium than from a vitamin containing both calcium and iron. The absorption of iron in supplements depends on the amount of elemental iron in the formulation, whether it is a quick-release or extended-release formulation, and whether the iron is in the ferric, ferrous, or iron polymaltose complex form.[104] Doses and side effects of various iron supplements are detailed in the *Nutrition* chapter.

Because iron absorption increases in pregnancy, some authors have proposed that intermittent iron supplementation may be preferable to daily iron supplementation. Exposure of the intestinal mucosal cells to iron supplements on an intermittent basis may improve iron absorption because these cells might absorb iron better if not exposed continuously. Randomized controlled trials of intermittent iron supplementation rather than daily iron supplementation demonstrated equally positive pregnancy outcomes when iron supplements were taken one to three times per week rather than daily.[102]

Iodine. Iodine deficiency can cause significant intellectual impairment but is rare in the United States, given that table salt and many common foods are routinely fortified with iodine. Nevertheless, many multivitamins contain iodine. Iodine supplementation during pregnancy is recommended by the American Thyroid Association.[105]

Vitamin D. Data on the effects of vitamin D supplementation are conflicting. Some studies have shown that vitamin D supplementation reduces the incidence of preeclampsia and preterm birth, but these data are preliminary and more research is needed.[106]

Prenatal Multivitamins. Prenatal multivitamins generally contain sufficient amounts of the vitamins and minerals that may be needed by a pregnant woman and, therefore, are a convenient vehicle for delivering those micronutrients. Prenatal multivitamins are specifically

recommended for women who are in one of the following categories: nutritionally inadequate diet, multiple pregnancy, cigarette smoker, addictive drug or alcohol use, adolescent, vegetarian, history of bariatric surgery, eating disorder, or lactase deficiency.

The amounts of specific vitamins and minerals in commercially marketed prenatal vitamins vary greatly, and there is no known advantage for one combination over another. With the addition of extra calcium and the omega-3 long-chain polyunsaturated fats (docosahexaenoic acid [DHA] and eicosapentaenoic acid [EPA]), many prenatal vitamins become difficult to swallow. Some manufacturers provide chewable vitamins, whereas others recommend twice-daily dosing. If calcium, iron, and omega-3 supplementation are needed, midwives might consider separate tablets for each nutrient needed.

Prenatal multivitamins can cause increased gastrointestinal distress and constipation in some women. These symptoms presumably arise secondary to the iron content in the vitamin. For women with nausea and vomiting, discontinuing iron-containing pills does appear to improve nausea symptoms.[107] When a prenatal vitamin is discontinued, even if temporarily, it should be replaced with a folic acid supplement.

Vitamins are not always beneficial. For example, vitamin A in large doses can be a teratogen. Initially the threshold for adverse effects was thought to be more than 25,000 IU of vitamin A daily, but studies have since shown that cranial neural crest anomalies may occur with as little as 10,000 IU daily. Routine supplementation of vitamin A is unnecessary, and, if supplementation is used, it should not exceed 5000 IU daily. Typical prenatal multivitamins contain at least 800 IU of vitamin A. Women should be cautioned not to double or triple their intake of multivitamins in an attempt to get additional folic acid or calcium. Increasing intake of a multivitamin increases intake of all of that product's constituents, including vitamin A.

Special Nutritional Needs

Vegetarians

The term *vegetarian* can encompass a number of subpopulations: women whose diets include dairy (lacto-vegetarians); those who eat eggs (ovo-vegetarians); and those who eat both dairy and eggs (lacto-ovo vegetarians). Individuals who strictly eat only foods from plant sources and do not use animal products such as leather can be called *vegans*. Women who eat any of the vegetarian diets can meet all their nutritional needs in pregnancy and have healthy birth outcomes,[108] although some modifications may be needed. In particular, a vegetarian diet during pregnancy may not provide the recommended daily amounts of iron, vitamin D, vitamin E, and choline.[109] Vitamin B_{12} intake can be adequate among women who consume eggs, dairy products, and fortified foods; without a regular source of vitamin B_{12}, however, vegan diets tend to have low levels of this vitamin, and possibly low levels of calcium. Women consuming a vegan diet need to be certain that they are receiving vitamin B_{12} from either fortified soy and rice beverages, fortified foods, or nutritional yeast supplements. Fermented soy products are not a source of vitamin B_{12}.

Iron from plant sources is non-heme iron; thus, it is less easily absorbed and more foods can inhibit its absorption than is the case with heme iron. Nevertheless, iron can be obtained in a vegetarian diet. Nonanimal sources of zinc include nuts and beans. Similar to what happens

with iron, phytates can bind zinc, thereby preventing absorption of this micronutrient. Another source of zinc is fortified cereals.

If no dairy products are consumed, pregnant women may not have adequate vitamin D levels and, in the absence of sufficient sunlight exposure, may need vitamin D supplements. Iodine, which comes from fish or sea vegetables and dairy, will be low if these foods or iodized salt are not part of the diet.

Studies have shown that vegetarian diets, and especially vegan diets, are lower in the omega-3 long-chain polyunsaturated fats, DHA and EPA, compared to an omnivorous diet. These fatty acids are essential for development of the retina and central nervous system. It is generally assumed that they must be obtained from diet, although the exact amounts needed per day and the adverse effects of deficiencies have not been determined.[110,111] Women who eat eggs can increase their DHA by consuming DHA-fortified eggs from hens fed microalgae; otherwise, women can consume microalgae-derived DHA supplements to obtain sufficient amounts of these fatty acids.

Women with Medical Conditions

Medical conditions that adversely impact nutrition during pregnancy include eating disorders,[91] a history of bariatric surgery,[92] conditions associated with malabsorption, or a need for medications that affect folic acid metabolism. The most likely nutritional deficiencies in women with medical conditions that diminish their ability to absorb nutrients have been identified. Midwives caring for these women will want to consult with a nutritionist to individualize their care.

Midwives are increasingly providing care for women after bariatric surgery. Two types of bariatric surgery procedures are commonly performed: (1) restrictive (laparoscopic gastric banding or Lap-Band) and (2) restrictive/malabsorptive (Roux-en-Y gastric bypass [RYGB]). Lap-Band slippage and movement can occur during pregnancy and result in severe vomiting. Some advocate for deflating the band prior to pregnancy to allow for adequate nutrition, though there are no national guidelines. Current recommendations advise women to wait 12 to 18 months after bariatric surgery before starting a pregnancy, so that weight loss is stabilized.[112]

Knowing the type of bariatric procedure done allows the midwife to provide appropriate nutritional advice and risk screening. Gestational weight gain recommendations are based on pregravid BMI even after bariatric surgery. Any type of malabsorptive surgery will reduce food, micronutrient, and medication absorption. In turn, deficiencies in iron, vitamin B_{12}, folate, vitamin D, calcium, and protein can be common after such surgery.

Baseline vitamin and mineral status of the pregnant woman post bariatric surgery is evaluated at the first prenatal visit, with supplements recommended as needed. A complete blood count and measurement of vitamin B_{12}, iron, ferritin, calcium, and vitamin D levels every trimester should also be considered.[112] The midwife providing prenatal care for a woman following bariatric surgery must effectively coordinate care among a network of clinicians, including maternal–fetal medicine physicians, nutritionists, and ultrasonographers. Such team-based care optimizes a woman's chance for a safe and satisfying birth.

Women who take anticonvulsant medications (e.g., valproate [Depakote, Depakene],

carbamazepine [Tegretol]) should take 4 milligrams of folic acid per day starting 1 month prior to conception and continue that regimen throughout the pregnancy, as these medications have an antifolate effect.

Multifetal Gestation

There are no national guidelines for the increases in other nutritional requirements for women with multiple pregnancies, but extrapolations have been made from singleton to twin pregnancies. It is generally recommended that women with multiple fetuses take two prenatal vitamin supplements beginning in the second trimester to obtain the additional iron, folate, calcium, magnesium and zinc that is needed.[113] This recommendation is safe as long as the vitamin A in the supplements is not retinol; if it is retinol, then the dose needs to remain less than 10,000 IU.

Pica

Pica is the term applied to the unusual condition in which an individual purposefully ingests nonfood items. Pica is more common in pregnant women than in women who are not pregnant. The prevalence of pica varies by geographic region and culture, but has been noted to affect as many as 50% of women in some settings.[114] The most common substances eaten are earth, clay, or dirt; raw starches such as cornstarch; and ice and freezer frost. The etiology of pica is unknown, although several theories exist. There is a clear association between ice-ingestion pica and iron-deficiency anemia, but it is not clear whether pica causes anemia or whether anemia triggers pica. Pica can cause lead poisoning and may be associated with other micronutrient deficiencies. Management consists of diagnosis and treatment of nutritional deficiencies, assessment of hunger or eating disorders, and health education.

Women with Limited Resources

Women with low incomes are at increased risk for dietary deficiencies. The Special Supplemental Nutrition Program for Women, Infants, and Children (WIC)—an income-based, supplemental food program from the U.S. Department of Agriculture (USDA)—provides food resources and cultural-based nutrition education for pregnant women, breastfeeding women, infants, and children younger than the age of 5. WIC policy changes from 2009 aimed at consumption of healthier foods and access to farmers markets have improved access to fresh fruits and vegetables and improved dietary intakes among this program's participants. The program is also able to connect women with other important community resources to help improve their pregnancy outcomes. WIC is not an entitlement program, but rather a grant program that requires reallocation of funding annually. Therefore, changes may occur at any time based on federal and state funding.

Psychosocial Risk Screening and Counseling

Many nonbiologic factors affect mental and physical well-being during pregnancy. Screening for perinatal mental health disorders, substance abuse, and intimate-partner violence is sensitive and should be done in private. Screening should be performed early in pregnancy and repeated if there are indications the woman's situation has changed. Screening should be offered to all pregnant women regardless of race, ethnicity, or socioeconomic background.

Depression Screening

Depression is common among reproductive-age women. Untreated depression has been associated with adverse perinatal outcomes, impaired maternal–infant attachment, and disordered family relationships.[115] Screening for depression and anxiety is recommended for all pregnant women. Screening tools are reviewed in the *Mental Health Conditions* chapter.

Intimate-Partner Violence Screening

Screening for intimate-partner violence (IPV) deserves special note, because violence may begin during pregnancy, it may increase in severity, and women may need time to consider whether they want to reveal the violence during prenatal care.[116] Violence may take the form of threatened or actual physical, sexual, verbal, or psychologic abuse, and is more likely to escalate during pregnancy. Persons may be more vulnerable to the effects of violence while pregnant, due to their physical condition or their financial and emotional dependence. When a woman is exposed to violence, child abuse is also more likely to occur. Any individual can be the victim of violence, irrespective of her race, ethnicity, socioeconomic status, profession, sexual orientation, or gender identity.

It is recommended that women be assessed for the risk of intimate-partner violence at least once per trimester.[116] There are no reliable indicators of IPV, but women who are exposed to violence are more likely to have the following characteristics: late entry to prenatal care or missed appointments, nonmedical or excessive use of substances, sexually transmitted infections, poor weight gain and nutrition, multiple somatic complaints or hospital visits, and undesired pregnancy.

A variety of screening tools for IPV can be used, though all such screening should be conducted in private.[117] There is no single screening method or tool that has demonstrated consistently high sensitivity and specificity in screening for IPV, especially in outpatient settings with diverse populations. Midwives may choose to screen for IPV in a face-to-face interview, using a self-administered questionnaire, or using a computer-assisted self-interview.[118]

The midwife's primary role in working with a woman who is a victim of IPV is to assess her safety, to help her make a plan for safety if needed, and to accurately document her condition and refer her to appropriate resources. IPV is a very dangerous environment; when it is revealed, the midwife has an ethical responsibility to provide the woman with needed resources and to follow up as indicated. Midwives also need to be aware of legal reporting

requirements. In particular, child abuse is reportable in all jurisdictions in the United States.

Substance Use Screening

Screening for drug use, alcohol use, and smoking is often included while obtaining a general health history. Use of illicit drugs or illicit use of prescription drugs may cause fear of legal ramifications for the woman and her unborn child and delay presentation for care.

Smoking Cessation

Despite known adverse health effects associated with smoking, approximately 13.6% of women in the United States smoke.[119] Pregnancy is an ideal time to address smoking cessation. As many as 45% of women smokers do stop smoking during prenatal care, although the metabolism of nicotine during pregnancy is faster, which can make smoking cessation more difficult.

Smoking cessation interventions have been shown to lower the incidence of low-birth-weight infants and preterm birth.[119] Smoking cessation counseling via use of the 5 A's prenatal smoking cessation approach can be effective. A pilot study in West Virginia demonstrated a clear reduction in pregnant women's smoking after midwives were trained to use the 5 A's program.[120]

Behavioral interventions alone are not sufficient for the majority of women who try smoking cessation during pregnancy. Research on the effects of smoking cessation medications used during pregnancy is limited,[121] and the USPSTF has stated that the safety and efficacy of nicotine replacement products has not been sufficiently reviewed for use by pregnant women. Nonetheless, given the import of smoking cessation, the "window of opportunity" presented during prenatal care, and research evidence showing that nicotine replacement therapies can be effective during pregnancy, many providers offer nicotine replacement therapy. A summary of the therapies available and their pharmacologic effects can be found in recent reviews.[119,121,122]

At-Risk Alcohol Use

Fetal damage caused by maternal alcohol use ranges from mild to severe; thus, a general category known as fetal alcohol spectrum disorder has been described that encompasses the various forms of damage. Within this continuum, fetal alcohol syndrome is characterized by growth restriction, facial abnormalities, and central nervous system dysfunction. Fetal alcohol syndrome is one of the most common preventable causes of mental disability in the United States.

National guidelines in the United States and many other developed nations recommend that women do not consume alcohol during pregnancy. This recommendation reflects the fact that the dose–response relationship between alcohol intake and fetal effects is unknown. Alcohol freely crosses the placenta, and its elimination from both maternal and fetal systems is dependent upon maternal metabolism.

Surveys conducted by the CDC have found that 7.6% of pregnant women report using alcohol and 1.4% report binge drinking.[123] For women, at-risk drinking is defined as drinking more than three standard drinks on any day or exceeding seven standard drinks per week. A standard drink constitutes 5 ounces of wine (12% alcohol content), 12 ounces of beer (5% alcohol content), or 1.5 ounces of distilled liquor (40% alcohol content). No level of alcohol use is considered safe during pregnancy. All women should be screened for at-risk alcohol use; if a woman is identified to be at risk, the midwife should pursue her openness to intervention by using motivational interviewing techniques.[124]

Illicit Drugs

The complexity of individual motivations, the various physical reactions to drugs and their interactions, and the widespread tendency toward polypharmacy all contribute to the difficulty in assessing for substance abuse. Although many drugs can be used inappropriately, opioid use is a growing source of concern: The incidence of opioid dependency among pregnant women increased from 1.19 to 5.77 per 1000 hospital live births per year between 2000 and 2009 in the United States.[125]

Screening for substance use is recommended for all women early in pregnancy.[126] Several validated tools for substance use screening are available, as reviewed in the *Health Promotion Across the Lifespan* chapter. The care of a woman struggling with substance use requires collaboration with professionals in multidisciplinary programs who have skills in treating chemical abuse and addictions.

The factors that motivate recovery in a person who has been abusing drugs or alcohol are varied and unpredictable. Sometimes concern about a fetus during pregnancy may be the catalyst to begin the cycle of recovery. By providing ongoing care and support while recognizing the recovery/relapse pattern, the midwife can work to minimize maternal and fetal complications, encourage decreased substance use, and support the woman appropriately depending on where she is in the cycle of recovery.

Marijuana

Women’s reports of marijuana or cannabis product use during pregnancy may place the midwife in an awkward position. Marijuana is a legal medicinal and recreational product in a growing number of states, but remains illegal in others. Data indicating safety or harm to the fetus from maternal marijuana use during pregnancy are limited, which may lead women to believe marijuana is safe. Cannabinoids cross the placenta and have not been linked with fetal malformations, but a growing evidence base indicates that in utero exposure to marijuana may cause harm to the fetus.[127,128] ACOG advises cessation of cannabis use in pregnancy and lactation, even if prescribed for medicinal purposes.[129]

Health Education During Pregnancy

Health education about health promotion and health maintenance strategies is a core component of midwifery care for all women of all ages, as reviewed in the *Health Promotion Across the Lifespan* chapter. Nevertheless, many women do not access primary healthcare services until they are pregnant, and certain health promotion strategies are particularly important for fetal health. Therefore, prenatal care is considered one of the most opportune periods to address these health education topics. Health maintenance topics that are frequently of import to pregnant women are summarized in **Table 21-16**.[130-135]

Table 21-16 Health Maintenance During Pregnancy

Topic	Recommendations
Air travel	Air travel is not associated with known risks for healthy women. Flying increases the risk for venous thromboembolism; pregnant women are encouraged to wear support hose and/or move frequently if traveling a long distance.[130] Women with conditions that increase the risk for hypoxia, such as cardiac disease or fetal growth restriction, may need supplemental oxygen or should avoid flying.[130] It is safe to fly until 36 weeks' gestation, when the risk of spontaneous labor increases.[130]
Exercise and physical activity	Regular exercise is associated with cardiovascular fitness, prevention of low back pain, reduced symptoms of depression, and lower weight gain. Thirty minutes or more of moderate exercise per day is recommended for pregnant women. Avoid activities that increase the risk for falls or abdominal injury. Scuba diving and sky diving are not recommended.
Hair treatments	No data support a relationship between hair dye and teratogenic effects.
Hot tubs and saunas	Maternal fever or increased core temperature (> 102°F/38°C) is associated with an increased risk of neural tube defects (OR: 1.62; 95% CI: 1.10–2.17).[131] It takes approximately 10 minutes to raise the core temperature to > 40°C (104°F) in water of 40°C (104°F) and 15 minutes if the water temperature is 39°C (102°F).[132] Hot tubs should be avoided in the first trimester.
Medication use	Although only a few medications are known teratogens, the safety of most medications for use in pregnancy has not been proven. Women are counseled to check with a midwife or other healthcare provider before using medications or over-the-counter treatments.
Oral health	Pregnancy-associated changes increase a woman's risk of dental caries. Pregnancy-associated gingivitis increases the risk of developing periodontal disease. Women should be encouraged to see a dentist if it has been > 6 months since their last dental examination, or if they are experiencing any dental problems.[133] Dental X rays are safe if the abdomen and thyroid are shielded which is standard procedure. Treatment for most dental disorders can be performed during pregnancy.
Seat belts	Three-point seat belts are recommended. The lap belt is placed across the hips and below the uterus. The shoulder belt is placed between the breasts and lateral to the uterus.[134]
Sex during pregnancy	Sexual intercourse is not associated with adverse pregnancy outcomes. Sexual intercourse is contraindicated for women with a placenta previa, preterm labor, or ruptured membranes.[135]

Employment During Pregnancy and Maternity Leave

Working during pregnancy is generally quite safe. Information about the most common workplace hazards (e.g., anesthetic gases, formaldehyde, heat, night shifts or long work hours)

is available from the CDC.[136] Clinical guidelines for occupational lifting have been published and are shown in **Figure 21-3**.[136-138] Women who work in high-risk occupations and women who have medically complicated pregnancies can obtain work accommodations to assure safety.[139] The Pregnancy Discrimination Act of 1964 protects pregnant women from being fired secondary to being pregnant. It is also illegal to ban a woman from a particular job if she might become pregnant. Complaints about pregnancy discrimination are filed with the U.S. Equal Employment Opportunity Commission.

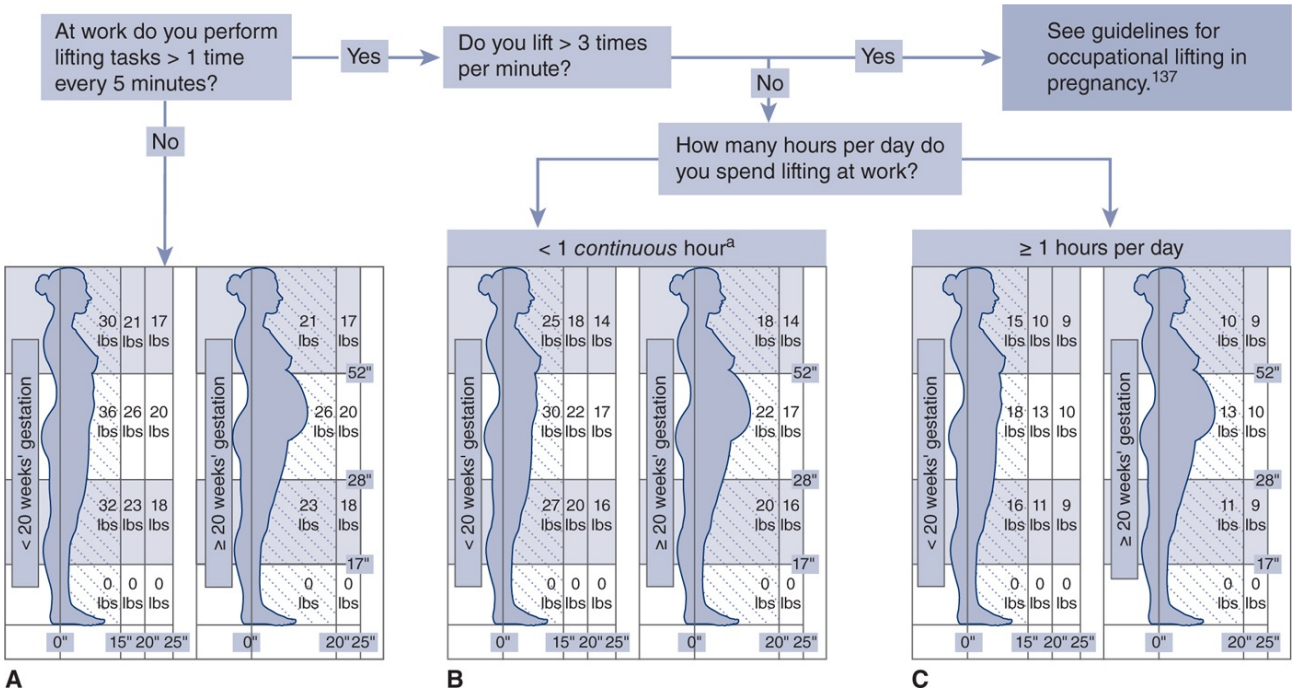


Figure 21-3 Provisional recommended weight limits for lifting at work during pregnancy. A. Infrequent lifting. B. Repetitive short duration lifting. C. Repetitive long duration lifting.

[a] Repetitive short duration lifting can encompass multiple hours of lifting per day; however, each continuous lifting period should be < 1 hour and followed by a minimum of 1 hour of nonlifting activity before the next continuous lifting period is initiated.

Reproduced with permission from MacDonald LA, Waters TR, Napolitano PG, et al. Clinical guidelines for occupational lifting in pregnancy: evidence summary and provision recommendations. *Am J Obstet Gynecol.* 2013;209(2):80-88.[137]

Laws and eligibility requirements governing pregnancy-related disability for childbirth vary by state. Maternity leave benefits are usually a combination of short-term disability, sick leave, vacation, and unpaid leave. Not all jobs offer maternity leave benefits. Although the federal Family Medical Leave Act (FMLA) mandates that individuals receive 12 weeks of unpaid leave for childrearing in a 12-month period, many women are not eligible for FMLA benefits. A current list of state laws and regulations regarding maternity leave benefits is included in the Resources section at the end of this chapter.

Environmental Exposures

The primary environmental exposures of concern in pregnancy are lead, mercury, ionizing and

non-ionizing radiation, and endocrine disruptors such as phthalates. A thorough review of environmental toxins and teratogens is presented in the *Preconception Care Visit* appendix in in the *Health Promotion Across the Lifespan* chapter. **Table 21-17** summarizes the environmental exposures for which there is the most evidence of adverse pregnancy effects.[140-146]

Table 21-17 Environmental Exposures with Adverse Pregnancy Effects

Exposure	Fetal Effects
Endocrine disrupters	Endocrine disrupters are chemicals that mimic or disrupt the effects of hormones such as estrogen, androgen, and thyroid in a way that results in adverse effects on development, reproduction, neurologic function, or immunity. The primary endocrine disrupters of concern for pregnant women are bisphenol A (BPA) and phthalates. Current evidence suggests that BPA and phthalates might potentially have adverse effects on a fetus, based on animal studies and effects on wildlife. To avoid these risks, women are counseled to use BPA-free baby bottles, avoid microwaving plastic containers, avoid use of plastic containers with the number 3 or 7 on the container, and limit intake of foods packaged in cans.[140] Phthalates are used to make soft plastics such as cling wrap, some baby toys, many cosmetics, and polyvinyl tubing. The most common phthalate, di(2-ethylhexyl) phthalate (DEHP), is currently voluntarily being removed by some manufacturers from baby toys.
Ionizing radiation (X ray)	X rays used in flat-plate imaging and computed tomography (CT) scans expose women to ionizing radiation that can damage DNA. High levels of exposure to ionizing radiation during organogenesis can cause congenital anomalies, and exposure to high doses up to 25 weeks' gestation can cause fetal growth restriction, mental retardation, and—at very high doses—death or stillbirth. Fetal effects are dose dependent, but are not noticeable when the exposure is < 0.05 Gy (5 rads).[141] Routine X rays expose the fetus to very low doses of ionizing radiation, with a chest X ray providing approximately 0.00007 rad.[142] Exposure in the first 2 weeks after conception is an "all or nothing" phenomenon: It either is fatal or has no adverse effects. Therefore, women can be assured that accidental exposure to one diagnostic X ray will not cause fetal harm.[143] If X-ray exposure has occurred, the woman can be referred to a radiologist who will calculate the exact dose and counsel her appropriately. Ultrasound and magnetic resonance imaging (MRI) are the imaging modalities of choice for imaging during pregnancy, but X rays and CT scans may be recommended if indicated and necessary for medical management.[143]
Lead exposure	Potential lead exposure from contaminated water supplies, outdated lead water pipes, and post-industrial soil contamination is widespread in the United States, and other countries.[144] Elevated lead levels are associated with miscarriage, gestational hypertension, low birth weight, and impaired neurodevelopment in offspring. Both chronic and acute lead poisoning can be dangerous during pregnancy, because lead is stored in bone and pregnancy is a time of increased bone turnover.[145] Although routine screening for lead exposure is not indicated, women who have risk factors for lead exposure are offered screening. Risk factors include drinking contaminated water; using water from outdated water pipes; dust exposure from home renovation or old, chipping paint; pica; gardening in soil contaminated by former industry; folk medications made outside of the United States; and use of unglazed pottery. Maternal venous lead levels of < 5 mcg/dL do not require any further follow-up. Women whose levels are between 10 mcg/dL and 45 mcg/dL should have the source of lead identified and be retested within a month, then within 1 month to 3 months, following the schedule recommended by the CDC.[145] Women whose lead level is > 45 mcg/dL are referred to a clinician who specializes in caring for individuals with lead toxicity.
Mercury exposure	Mercury exists naturally in the environment but is also a pollutant resulting from industrial processes. Excess mercury can impair neurologic development in a fetus. The primary source of mercury today is the methylmercury that accumulates in large fish. Pregnant

	women are counseled to avoid consumption of king mackerel, marlin, orange roughy, shark, swordfish, tilefish (Gulf of Mexico), and tuna (bigeye). In addition, women are advised to avoid fish caught by family and friends, unless local advisories have been reviewed. A former source of mercury was the thimerosal in vaccines. All vaccines offered to pregnant women and infants are now thimerosal free.
Non-ionizing radiation	Ultrasound, microwaves, electric blankets, computers, cell phones, and airport screening devices for metal all emit non-ionizing radiation. Exposure to non-ionizing radiation has not been shown to cause fetal harm.[146]

Infection and Disease Protection

Pregnant women, like all individuals, are exposed to infectious organisms. Some infections can harm a fetus or newborn, whereas others are more likely to cause serious or life-threatening illnesses in a woman during pregnancy if she becomes infected.

- Infections that can harm the fetus include listeriosis, parvovirus, rubella, syphilis, varicella, cytomegalovirus, and toxoplasmosis. Herpes and group B *Streptococcus* infection can be harmful to the newborn if infected at the time of vaginal birth. Zika virus, noted in late 2015 to be associated with congenital microcephaly, is an example of an emerging viral teratogen.[147]
- Influenza and varicella can become severe illnesses in pregnant women and are associated with an increased risk of maternal death.

Health education related to infection precautions is described in **Table 21-18**.[148-154] Additional information about management of exposure to these infections in nonimmune individuals is discussed in the *Medical Complications in Pregnancy* chapter.

Table 21-18 Prevention of Infection During Pregnancy

Disease-Causing Agent	Prevention Measures
Cytomegalovirus	Wash hands after changing a diaper or handling body fluids of an infant or toddler.
Herpes	Avoid sexual contact with persons who have active herpes lesions.
Listeriosis	Wash all raw vegetables before eating. Do not consume unpasteurized milk or soft cheeses made with unpasteurized milk. All meat, including precooked deli meats, should be properly heated before eating. Avoid smoked seafood and salads made in stores, such as ham and chicken salads, as *Listeria* can grow slowly at refrigerator temperatures.
Parvovirus B19 (fifth disease or slap-cheek disease)	Teachers, childcare workers, and others who work with children are at risk. Avoid contact with a child who has this illness.
Pertussis (whooping cough)	Teachers, childcare workers, and others who work with children are at risk. Avoid contact with a child who has this illness unless immunity is known. Vaccination during each pregnancy is recommended.
Rubella	Avoid contact with a child who has this illness unless immunity is known.
Toxoplasmosis	Avoid cleaning cat litter boxes. If this task cannot be avoided, wear gloves. Wash hands after

	cleaning and working in dirt or soil unless gloves are worn. Do not eat raw or uncooked meat.
Varicella (chickenpox)	Avoid contact with a child who has this illness unless immunity is known.
Zika virus	Avoid travel to areas where the *Aedes* mosquito is epidemic.

Based on Meaney-Delman D, Rasmussen SA, Staples JE, et al. Zika virus and pregnancy: what obstetric health care providers need to know. *Obstet Gynecol*. 2016;124(4):642-648[150]; Madjunkoy M, Chaudhry S, Ito S. Listeriosis during pregnancy. *Arch Gynecol Obstet.* 2017;296(2):143-152[151]; Sugishita Y, Akiba T, Sumitomo M, et al. Shedding of rubella virus among infants with congenital rubella syndrome born in Tokyo, Japan, 2013–2014. *Jpn J Infect Dis.* 2016;69(5):418-423[152]; Neu N, Duchon J, Zachariah P. TORCH infections. *Clin Perinatol.* 2015; 42(1):77-103[153]; Feldman DM, Keller R, Borgida AF. Toxoplasmosis, parvovirus and cytomegalovirus in pregnancy. *Clin Lab Med.* 2016;36(2):407-419.[154]

Immunizations

Maternal immune antibodies of the IgG class cross the placenta and generally confer passive immunity to the fetus. Vaccines that are composed of inactive antigen ingredients or toxoid are safe for use in pregnancy, whereas live-attenuated vaccines have a small risk of causing disease and are not recommended for use by pregnant women. Vaccines that are recommended and those that are contraindicated during pregnancy are listed in **Table 21-19**.[155]

Table 21-19 **Immunizations That Are Recommended or Contraindicated During Pregnancy**

Vaccine	Recommended in Pregnancy	Type of Vaccine
Hepatitis A	If at risk[a]	Inactivated
Hepatitis B	If at risk[b]	Inactivated
Human papillomavirus (HPV)	No[c]	Inactivated
Influenza, trivalent inactivated vaccine (TIV)	Recommended for all women during pregnancy	Inactivated
Influenza, live attenuated vaccine (LAIV)	Contraindicated	Live
Measles, mumps, rubella (MMR)	Contraindicated	Live
Meningococcal: polysaccharide or conjugate	If indicated	Inactivated
Pneumococcal polysaccharide	If indicated	Inactivated
Tetanus/diphtheria (Td)	Yes, but Tdap preferred if > 20 weeks' gestation	Toxoid
Tetanus/diphtheria/pertussis (Tdap), one dose only	Recommended for all women during each pregnancy, irrespective of having received the vaccine previously	Toxoid/inactivated
Varicella	Contraindicated	Live

[a] The hepatitis A vaccine is considered safe for pregnant women. It is recommended only if the woman has a high risk of exposure.

[b] Risk factors for hepatitis B include having more than one sex partner during the previous 6 months, having been

evaluated or treated for a sexually transmitted disease, recent or current injection drug use, or having had a hepatitis B surface antigen–positive sex partner.

[c] If a woman is in the middle of the HPV vaccine series and becomes pregnant, the rest of the series is delayed until after the pregnancy.

Based on Kim DK, Riley LE, Harriman KH, Hunter P, Bridges CB; ACIP Adult Immunization Work Group, Centers for Disease Control and Prevention. Advisory Committee on Immunization Practices recommended immunization schedule for adults aged 19 years and older—United States, 2017. *MMWR Surveill Summ.* 2017;66:5.[155]

All pregnant women and their immediate family members are offered vaccination for tetanus, diphtheria, and pertussis (Tdap) during pregnancy to protect the newborn from pertussis until infant vaccination is complete.[156] Annual influenza vaccines are offered during pregnancy in the fall before the spring influenza season. If women are in the middle of the hepatitis B series when they become pregnant, they are counseled to finish the series. In contrast, women who are in the middle of the human papillomavirus (HPV) vaccine series are counseled to wait until the pregnancy is over before finishing this series. Additional vaccines can be given during pregnancy if the woman is at increased risk for specific infections.

The topic of vaccine safety is important to the woman, her fetus, and her newborn. Controversies about purported associations between vaccines and methylmercury poisoning and between vaccines and autism have been reviewed extensively. Although thimerosal was not shown to be harmful in the amounts used in vaccines, it has been removed from all vaccines administered to pregnant women and children so vaccines no longer expose individuals to methylmercury.[157] The hypothesized relationship between vaccines and autism has been found to be spurious.[158]

Preparing for Birth

Health education topics specific to preparing for labor, birth, and parenting include discussions of where to give birth, what to expect during labor and birth, childbirth education classes, newborn feeding and newborn procedures such as circumcision, and preparing the home for the newborn (**Table 21-20**).

Table 21-20 Health Education Topics: Preparing for Birth

Topic	Recommendations
Birth place	Individual choice and prenatal risk screening can assist women in choosing a location for the birth: home, birth center, or hospital. Birth place arrangements are finalized in the third trimester.
Car seats	Car seats are required by state law in all states, for children up to specific age and weights. State laws vary. Infants should ride in a rear-facing seat until age 2 years. State laws for car seats can be found at http://www.iihs.org/iihs/topics/t/child-safety/topicoverview.
Circumcision	The American Academy of Pediatrics states that the health benefits of circumcision outweigh the risks of this procedure, but the benefits are not great enough to recommend routine circumcision of all male newborns. (See the *Neonatal Care* chapter.)
Newborn examinations and tests	Some examinations, tests, and immunizations such as eye prophylaxis, newborn hearing screening, and vitamin K administration are offered to newborns in the first few days after birth. A review of these tests and their purpose during prenatal visits can help women prepare and make informed choices about the tests.

Postpartum contraception	Some forms of contraception can be initiated in the immediate postpartum period. A discussion of options and timing for initiation is best started in the prenatal period.
Preparing for feeding the infant	Breastfeeding or chest-feeding is recommended unless medically contraindicated. Infants can thrive if not breastfed. It is important to respect personal choice and informed decision making.

Childbirth Preparation Classes

One could argue that childbirth education has existed forever in the form of information shared by women and midwives. Even today, pregnant women obtain the majority of the information they receive about birth from electronic media, books, or friends and family, rather than healthcare providers or childbirth educators.[159] Nevertheless, childbirth education classes, like prenatal vitamins, are generally recommended, albeit without strong evidence for their effectiveness.

In the United States, the first formal childbirth classes in the early 1900s focused on preparation for parenting, nutrition, and health care.[160] Formal childbirth preparation classes that addressed how to cope with labor first started in the United States in 1947, when the Maternity Center Association in New York sponsored Grantly Dick-Read's visit to the United States. Dick-Read's work served as the foundation for the different models of childbirth preparation taught today.[159] The focus of the childbirth preparation techniques was originally to teach women about the process of labor and to give them techniques for coping with labor pain, with the goal of having an unmedicated birth. Today, most childbirth classes also teach women about medical procedures that can occur during labor; the focus on having an unmedicated birth varies with the particular technique. **Table 21-21** briefly outlines the different types of childbirth education classes that are taught today.[160]

Table 21-21 Comparison of Childbirth Education Models and Organizations

Childbirth Education Model	Description
Lamaze Childbirth Education	Goal: increase confidence in giving birth by focusing on how to cope with labor pain Natural childbirth Breathing techniques used to enhance relaxation and decrease perception of pain Communication skills Evidence based Comfort measures Information about prenatal procedures Breastfeeding encouraged
Bradley Method (husband-coached childbirth)	Goal: facilitate natural birth via breathing techniques and partner support Natural childbirth Partner's active participation Avoidance of medications and medical procedures Nutrition and exercise

	Relaxation techniques, guided imagery, and coping mechanisms
	Immediate contact with newborn and immediate breastfeeding
	Six needs of laboring women: darkness and solitude; quiet; physical comfort during the first stage of labor; physical relaxation; controlled breathing; need to close eyes with the appearance of sleep
International Childbirth Education Association (ICEA)	Family-centered maternity care
	Freedom of choice
	Alternatives in childbirth
	Draws from other models
HypnoBirthing, Mongan Method	Calm, peaceful, natural childbirth
	Relaxation techniques and negate the fear–tension–pain cycle
	Education
	Evidence based
Mindfulness-Based Childbirth Preparation (MBCP)	Mindfulness techniques used to help individuals and their support persons cope with labor
	Meditation and yoga
	Stress reduction, awareness, and breathing
	Group dialogue
Birthing From Within	Natural childbirth goal
	Based on *primordial knowing* (knowledge of body) and *modern knowing* (knowledge of medical culture and how to give birth within this culture)
	Soulful and holistic approach
	Creative self-expression via artwork and journaling
	Instructor and couples co-create class together
	Coping with challenges
Hospital-based classes	Instructors can be certified in Lamaze, ICEA, or other childbirth education technique
	Philosophy of childbirth educators varies
	Increased focus on institutional policies and procedures with medical procedures information
	Breathing, relaxation, and massage technique

Modified with permission from Walker DS, Visger JM, Rossie D. Contemporary childbirth education models. *J Midwifery Womens Health.* 2009;54(6):469-471.[160] © 2009, with permission from Wiley.

The effectiveness of childbirth education has proved difficult to determine, as the goals of the programs are all somewhat different and there may exist differences between groups of women who seek classes and those who do not. Overall, formal childbirth education appears to improve knowledge, lower anxiety, and increase self-efficacy. The effect of childbirth education on perception of labor pain is modest at best.

Preparing for Breastfeeding

Breast milk is the optimal form of nutrition for newborns and infants. Prenatal breastfeeding education has multiple benefits that include higher rates of breastfeeding exclusivity and duration.[161] Although most women initiate breastfeeding in the immediate postpartum period, some individuals choose not to breastfeed. The discussion of infant feeding plans is a process of shared decision making.

Prenatal breastfeeding education has multiple components and is best done over several

visits. **Table 21-22** summarizes the topics included in the prenatal assessment. Through this assessment, the midwife can identify contraindications to breastfeeding, the woman's goals for infant feeding, and any previous experience with breastfeeding. Further discussion can then be tailored to the individual and the midwife can help the woman develop a plan for addressing problems that may be anticipated.

Table 21-22 Prenatal Assessment for Breastfeeding

Medical, Surgical, and Psychosocial History
Past medical history of PCOS, diabetes, or condition that requires medications which might affect breastfeeding.
Contraindications to breastfeeding (maternal infectious disease such as HIV or untreated TB, or required medications that are contraindicated during breastfeeding such as statins)
History of breast surgery including augmentation or reduction
Breast cancer: mastectomy or lumpectomy
History of physical, sexual, or psychological abuse
Breastfeeding History
Plan regarding breastfeeding
Prior unsuccessful breastfeeding
Family or support persons attitudes toward breastfeeding
Physical Examination
Breast size, symmetry, and shape; any scars or abnormalities
Nipple type, texture, and extensibility

Abbreviations: HIV, human immunodeficiency virus; PCOS, polycystic ovary syndrome; TB, tuberculosis.

Any history of sexual trauma or abuse should be discussed with regard to the plan for breastfeeding once a therapeutic relationship has been established because it may trigger underlying stressors that could affect the breastfeeding relationship.

Colostrum discharge may be noted after about 16 weeks' gestation. Colostrum may be clear, yellowish, or milky, and signals normal changes consistent with preparation for lactation. However, inability to express colostrum prenatally should not be interpreted as a sign of pending lactation failure.

Significant breast asymmetry or a wide space between breasts may be a marker for marginal or inadequate functional tissue. Wide variations exist in nipple configuration, size and shape. Preparation of the nipple tissue is unnecessary and has not been proven to be beneficial. Likewise, the prenatal use of breast shields for women who have inverted nipples does not improve breastfeeding outcomes and are not recommended. A postpartum consultation with a lactation consultant may be of benefit for women with unusual breast configurations or inverted nipples.

Postpartum Contraception

The initial conversation about postpartum contraception begins prenatally. Women often think about their family size during the third trimester. Intentions about future pregnancies can be

explored, and the recommendation reviewed that pregnancies be spaced by at least 18 months, or 2 years in the case of cesarean section.

Contraceptive choices may also depend on infant feeding choice. Some women assume that pregnancy cannot occur while they are breastfeeding and not menstruating. Women should be informed that they may have a return to fertility prior to the return of menses. Also, health education should note that breastfeeding does not reliably prevent pregnancy, even if it prevents return of regular menses, unless the requirements of the lactational amenorrheic method are met as outlined in the *Nonhormonal Contraception* chapter.

While postpartum contraception is traditionally initiated at the 6-week postpartum visit, progesterone-only methods can be initiated in the immediate postpartum period, including Depo-Provera and long-acting reversible contraception (LARC). Women unlikely to attend a postpartum visit can be offered immediate postpartum initiation to help them prevent an unintended pregnancy. A delay in the initiation of estrogen-containing contraceptives is recommended until 8 weeks postpartum to decrease the risk of thromboembolism. More information on contraceptive choices is included in the *Family Planning* chapter.

Preparation of the Home

Women often have questions about items needed for child care. Current American Academy of Pediatrics recommendations state that the infant should sleep on his or her back in a separate crib or bassinet with a firm sleep surface, preferably in the same room with the parent for the first year of life.[162] Many women, while aware of the recommendations for safe sleep, choose to co-sleep with their infant, which provides an opportunity for a discussion of safe sleep practices.

An infant car seat is required by law for all infants placed in cars, and checking for one is often a part of the discharge process after a hospital birth. Car seats and bassinets are sometimes available through health insurance companies or local programs for women with limited resources.

Conclusion

Although protocols and guidelines abound, prenatal care is more than a simple series of visits with scripted interventions. In reality, the complex nature of a pregnant person's experience requires flexible and knowledgeable midwifery care and a multifaceted approach. For the midwife, prenatal care requires an extensive knowledge of pregnancy, a broad understanding of common primary care topics, and recognition of the continuum of mental health states.

The midwife caring for a pregnant woman continually multitasks, holding several different perspectives at the same time, simultaneously offering support and health education, conducting ongoing risk assessment, and monitoring normal fetal development and pregnancy changes. Integrating these perspectives and helping women empower themselves, all in a short series of visits, is a unique and rewarding undertaking.

Resources

Organization	Description	Webpage
Dietary Advice and Weight Gain During Pregnancy		
Government of Canada	Prenatal Nutrition Guidelines for Health Professionals: Gestational Weight Gain. Based on the U.S. Institute of Medicine 2009 guidelines, this document has weight gain charts specific for each BMI category.	https://www.canada.ca/en/health-canada/services/food-nutrition/healthy-eating/p 2010.html
U.S. Department of Agriculture, (USDA)	ChooseMyPlate.gov provides information and educational materials on MyPlate diets, including vegetarian versions.	https://www.choosemyplate.gov
U.S. Food and Drug Administration (FDA)	Food safety during pregnancy, including current recommendations for fish consumption.	https://www.foodsafety.gov/risk/pregnant/
Education Materials		
American College of Nurse-Midwives (ACNM)	*Share with Women* handouts, featuring many prenatal care topics.	http://www.midwife.org/Share-With-Women
Centers for Disease Control and Prevention (CDC)	Information and educational materials for women and clinicians, including safety of vaccines in pregnancy.	https://www.cdc.gov/pregnancy/index.html
International Childbirth Education Association (ICEA)	Source of childbirth and parenting preparation information and materials.	https://icea.org
Lamaze International	Source of childbirth and parenting preparation information and	http://www.lamaze.org

	materials.	
March of Dimes	Source of information related to preterm birth and congenital illness prevention.	https://www.marchofdimes.org/giving/support-give.aspx?utm_campaign=semevergreen2017&gclid=Cj0KCQjwwLHLBRDEARIsAN1A1Q
Employment Protection		
A Better Balance	Organization that works to improve laws and policies regarding employment and caregiving for family. This site has multiple resources that summarize state laws regarding paid family leave, pregnancy, and discrimination in the workplace.	http://www.abetterbalance.org/know-your-rights
Centers for Disease Control and Prevention (CDC)/National Institute for Occupational Safety and Health (NIOSH)	Provisional recommended weight limits for manual lifting during pregnancy.	http://journals.sagepub.com/doi/10.1177/0018720813502223
U.S. Department of Labor	Employment protections for workers who are pregnant or nursing.	https://www.dol.gov/wb/maps/
Environmental and Toxic Exposure		
Centers for Disease Control and Prevention (CDC)	Information about environmental and workplace exposures that can have adverse effects on pregnancy.	https://www.cdc.gov/niosh/topics/repro/specificexposures.html

References

1. U.S. Public Health Service Expert Panel on the Content of Prenatal Care. *Caring for Our Future: The Content of Prenatal Care*. Washington, DC: U.S. Public Health Service, Department of Health and Human Services; 1989.
2. Alexander GR, Kotelchuck M. Assessing the role and effectiveness of prenatal care: history, challenges and directions for future research. *Public Health Rep*. 2001;116:306-310.
3. Gregory KD, Johnson CT, Johnson TRB, Entman SS. The content of prenatal care: update 2005. *Women's Health Issues*. 2006;16(4):198-215.
4. Dowswell T, Carroli G, Duley L, et al. Alternative versus standard packages of antenatal care for low-risk pregnancy. *Cochrane Database Syst Rev*. 2015;10:CD000934.pub3. doi: 10.1002/14651858.CD000934.pub3.
5. American College of Obstetricians and Gynecologists, American Academy of Pediatrics. *Guidelines for Prenatal Care*. 8th ed. Washington, DC: American College of Obstetricians and Gynecologists.
6. Krans EE, Davis MM. Preventing low birthweight: 25 years, prenatal risk, and the failure to reinvent prenatal care. *Am J Obstet Gynecol*. 2012;206(5):398-403.
7. Gadson A, Akpovi E, Mehta PK. Exploring social determinants of racial/ethnic disparities in prenatal care utilization and maternal outcome. *Semin Perinatol*. 2017;41(5):308-317.
8. U.S. Department of Health and Human Services, Health Resources and Services Administration. Prenatal–first trimester care access. Available at: https://www.hrsa.gov/quality/toolbox/508pdfs/prenatalmoduleaccess.pdf. Accessed March 13, 2017.
9. Hanson L, VandeVusse L, Roberts J, Forristal A. A critical appraisal of guidelines for antenatal care: components of care and priorities in prenatal education. *J Midwifery Womens Health*. 2009;54:458-468.
10. Lorch SA, Enlow E. The role of social determinants in explaining racial/ethnic disparities in perinatal outcomes. *Pediatr Res*. 2016;79(1-2):141-147.
11. Latendresse G. The interaction between chronic stress and pregnancy: preterm birth from a biobehavioral perspective. *J Midwifery Womens Health*. 2009;54(1):8-17.
12. Muglia LJ, Katz M. The enigma of spontaneous preterm birth. *N Engl J Med*. 2010;362:529-535.
13. Lefmann T, Combs-Orme T, Ore JG. Examining the inter-correlated effects of low income, life stress, and race on birth outcomes: a representative state study. *Int J Dev Neurosci*. 2017;60:1-7.
14. Palma-Gudiel H, Córdova-Palomera A, Eixarch E, Deuschle M, Fañanás L. Maternal psychosocial stress during pregnancy alters the epigenetics signature of the glucocorticoid receptor gene promoter in their offspring: a meta-analysis. *Soc Work Health Care*. 2017;56(6):450-469.
15. Stewart DE, Vigod S. Postpartum depression. *New Engl J Med*. 2016;375(22):2177-2186.
16. Ruedinger E, Cox JE. Adolescent childbearing: consequences and interventions. *Curr Opin Pediatr*. 2012;24(4):446-452.
17. LoGiudice J, Beck C. The lived experience of childbearing from survivors of sexual abuse: "It was the best of times, it was the worst of times." *J Midwifery Womens Health*. 2016;61:474-481.
18. Obedin-Maliver J, Makadon H. Transgender men and pregnancy. *Obstet Med*. 2016;9(1):4-8.
19. Light A, Obeind-Maliver J, Sevelius J, Kerns J. Transgender men who experience pregnancy after female–male gender transitioning. *Obstet Gynecol*. 2014;124:1120-1127.
20. Levy BS, Wilkinson FS, Marine WM. Reducing neonatal mortality rate with nurse-midwives. *Am J Obstet Gynecol*. 1971;109(1):50-58.
21. Hatem M, Sandall J, Devane D, Soltani H, Gates S. Midwife-led versus other models of care for childbearing women. *Cochrane Database Syst Rev*. 2008;4:CD004667. doi: 10.1002/14651858.CD004667.pub2.
22. Opara EL, Zaidi J. The interpretation and clinical application of the word "parity": a survey. *BJOG*. 2007;114:1295-1297.
23. Beebe KR. The perplexing parity puzzle. *AWHONN Lifelines*. 2005;9:394-399.
24. Spong CY. Defining "term" pregnancy. *JAMA*. 2013;309(23):2445-2446.
25. Butler SA, Khanlian SA, Cole LA. Detection of early pregnancy forms of human chorionic gonadotropin by home pregnancy test devices. *Clin Chem*. 2001;47(12):2131-2136.
26. Cole LA, Khanlian SA, Sutton JM, Davies S, Rayburn WF. Accuracy of home pregnancy tests at the time of missed menses. *Am J Obstet Gynecol*. 2004;190(1):100-105.
27. Benagiano G. The fate of fertilized human oocytes. *Reprod BioMed Online*. 2010;21:731.
28. Connolly AM, Ryan DH, Stuebe AM, Wolfe HM. Reevaluation of discriminatory and threshold levels for serum-hCG in early pregnancy. *Obstet Gynecol*. 2013;121(1):65-70.
29. Hunter L. Issues in pregnancy dating: revisiting the evidence. *J Midwifery Womens Health*. 2009;54:184-190.
30. Snell BJ. Assessment and management of bleeding in the first trimester of pregnancy. *J Midwifery Womens Health*. 2009;54:483-491.
31. Seeber B. What serial hCG can tell you, and cannot tell you, about an early pregnancy. *Fertil Steril*. 2012;98:1074-1077.
32. Cole LA, Ladner DG, Byrn FW. The normal variabilities of the menstrual cycle. *Fertil Steril*. 2009;91:522-527.

33. Sapra KJ, Buck Louis GM, Sundaram R, et al. Signs and symptoms associated with early pregnancy loss: findings from a population-based preconception cohort. *Human Reprod*. 2016;31(4):887-896.
34. Harville EW, Wilcox AJ, Baird DD, Weinberg CR. Vaginal bleeding in very early pregnancy. *Hum Reprod*. 2003;18(9):1944-1947.
35. Hasan R, Baird DD, Herring AHH, Olshan AF, Jonsson Funk ML, Hartmann KE. Association between first-trimester vaginal bleeding and miscarriage. *Obstet Gynecol*. 2009;114(4):860-867.
36. Naegele FS. *Erfahrung and Abhandlungen des Weiblichen Geschlechtes*. Mannheim, Germany: Ben Lobias Loeffler; 1812.
37. American College of Obstetricians and Gynecologists. Committee Opinion No. 700: methods for estimating the due date. *Obstet Gynecol*. 2017;129:e150-e154.
38. Chambliss LR, Clark SL. Paper gestational age wheels are generally inaccurate. *Am J Obstet Gynecol*. 2014;210(2):145.e1-145.e4.
39. Doubilet PM. Should a first trimester dating scan be routine for all pregnancies? *Semin Perinatol*. 2013;37(5):307-309.
40. Whitworth M, Bricker L, Mullan C. Ultrasound for fetal assessment in early pregnancy. *Cochrane Database Syst Rev*. 2015;7:CD007058. doi: 10.1002/14651858.CD007058.pub3.
41. Neilson JP. Symphysis-fundal height measurement in pregnancy. *Cochrane Database Syst Rev*. 1998;1:CD000944. doi: 10.1002/14651858.CD000944.
42. Hertz RH, Sokol RJ, Knoke JD, Rosen MG, Chik L, Hirsch VJ. Clinical estimation of gestational age: rules for avoiding preterm delivery. *Am J Obstet Gynecol*. 1978;131:395-400.
43. U.S. Preventive Services Task Force. Published recommendations. Available at: https://www.uspreventiveservicestaskforce.org/BrowseRec/Index/browse-recommendations. Accessed July 4, 2017.
44. Centers for Disease Control and Prevention. 2015 sexually transmitted diseases treatment guidelines. 2015. Available at: https://www.cdc.gov/std/tg2015/default.htm. Accessed July 4, 2017.
45. Centers for Disease Control and Prevention. TB guidelines testing and diagnosis. 2017. Available at: https://www.cdc.gov/tb/publications/guidelines/testing.htm. Accessed July 4, 2017.
46. Latendresse G, Deneris A. An update on current prenatal testing options: first trimester and noninvasive prenatal testing. *J Midwifery Womens Health*. 2015;60(1):24-36.
47. American College of Obstetricians and Gynecologists. Committee Opinion No. 690: carrier screening in the age of genomic medicine. *Obstet Gynecol*. 2017;129:e35-e40.
48. Cunningham FG. Appendix B: laboratory values in normal pregnancy. In: Queenan JT, Hobbins JC, Spong CYT, eds. *Protocols for High-Risk Pregnancies: An Evidence-Based Approach*. 5th ed. Oxford, UK: Blackwell; 2010:587-595.
49. American College of Obstetricians and Gynecologists. Practice Bulletin No. 175: ultrasound in pregnancy. *Obstet Gynecol*. 2016;128(6):e241.
50. Reddy UM, Filly RA, Copel JA; Pregnancy and Perinatology Branch, Eunice Kennedy Shriver National Institute of Child Health and Human Development, Department of Health and Human Services, National Institutes of Health. Prenatal imaging: ultrasonography and magnetic resonance imaging. *Obstet Gynecol*. 2008;112:145.
51. Shaw–Battista J, Young–Lin N, Bearman S, Dau K, Vargas J. Interprofessional obstetric ultrasound education: successful development of online learning modules; case–based seminars; and skills labs for registered and advanced practice nurses, midwives, physicians, and trainees. *J Midwifery Womens Health*. 2015;60(6):727-734.
52. Rising SS, Kennedy HP, Klima CS. Redesigning prenatal care through CenteringPregnancy. *J Midwifery Womens Health*. 2004;49(5):398-404.
53. Novick G, Sadler LS, Kennedy HP, Cohen SS, Groce NE, Knafl KA. Women's experiences of group prenatal care. *Qual Health Res*. 2011;21(1):97-116.
54. Grady MA, Bloom KC. Pregnancy outcomes of adolescents enrolled in a Centering Pregnancy program. *J Midwifery Womens Health*. 2004;49:412-420.
55. Ickovics JR, Earnshaw V, Lewis JB, et al. Cluster randomized controlled trial of group prenatal care: perinatal outcomes among adolescents in New York City health centers. *Am J Public Health*. 2016;106:359-365.
56. Picklesimer A, Billings D, Covington-Kolb S, Hale N, Blackhurst D. The impact of Centering Pregnancy group prenatal care on preterm birth in a low-income population. *Am J Obstet Gynecol*. 2012;206:S55-S56.
57. Tilden EL, Emeis CL, Caughey AB, Weinstein SR, Futernick SB, Lee CS. The influence of group versus individual prenatal care on phase of labor at hospital admission. *J Midwifery Womens Health*. 2016;61(4):427-434.
58. Kennedy HP, Farrell T, Paden R, et al. A randomized clinical trial of group prenatal care in two military settings. *Mil Med*. 2011;176:1169-1177.
59. Mazzoni SE, Hill P, Webster K, Heinrichs GA, Hoffman MC. Group prenatal care for women with gestational diabetes. *J Matern Fetal Neonatal Med*. 2016;29:2852-2856.
60. Trotman G, Chhatre G, Darolia R, Tefera E, Damle L, Gomez-Lobo V. The effect of Centering Pregnancy versus traditional prenatal care models on improved adolescent health behaviors in the perinatal period. *L Pediatr Adolesc Gynecol*. 2015;28:395-401.

61. Klima C, Norr K, Vonderheid S, Handler A. Introduction of CenteringPregnancy in a public health clinic. *J Midwifery Womens Health*. 2009;54(1):27-34.
62. Schellinger MM, Abernathy MP, Amerman B, et al. Improved outcomes for Hispanic women with gestational diabetes using the Centering Pregnancy group prenatal care model. *Matern Child Health J*. 2017;21:297-305.
63. Trudnak TE, Arboleda E, Kirby RS, Perrin K. Outcomes of Latina women in CenteringPregnancy group prenatal care compared with individual prenatal care. *J Midwifery Womens Health*. 2013;58(4):396-403.
64. Parikh LI, Jelin AC, Iqbal SN, et al. Glycemic control, compliance, and satisfaction for diabetic gravidas in centering group care. *J Matern Neonatal Med*. 2017;30(10):1221-1226.
65. Manant A, Dodgson JE. CenteringPregnancy: an integrative literature review. *J Midwifery Womens Health*. 2011;56(2):94-102.
66. Catling CJ, Medley N, Foureur M, et al. Group versus conventional antenatal care for women. *Cochrane Database Syst Rev*. 2015;2:CD007622.
67. Carter EB, Temming LA, Akin J, et al. Group prenatal care compared with traditional prenatal care: a systemic review and meta-analysis. *Obstet Gynecol*. 2016;3:551-561.
68. Mazzoni SE, Carter EB. Group prenatal care. *Am J Obstet Gynecol*. 2017;216:552-556.
69. Lumbiganon P, Laopaiboon M, Thinkhamrop J. Screening and treating asymptomatic bacteriuria in pregnancy. *Curr Opin Obstet Gynecol*. 2010;22(2):95-99.
70. Gribble RK, Fee SC, Berg RL. The value of routine urine dipstick screening for protein at each prenatal visit. *Am J Obstet Gynecol*. 1995;173:214-217.
71. DeSisto CL, Kim SY, Sharma AJ. Prevalence estimates of gestational diabetes mellitus in the United States, Pregnancy Risk Assessment Monitoring System (PRAMS), 2007–2010. *Prev Chronic Dis*. 2014;11:E104.
72. Schwartz N, Nachum Z, Green MS. The prevalence of gestational diabetes mellitus recurrence: effect of ethnicity and parity: a metaanalysis. *Am J Obstet Gynecol*. 2015;213(3):310-317.
73. England LJ, Dietz PM, Njoroge T, et al. Preventing type 2 diabetes: public health implications for women with a history of gestational diabetes mellitus. *Am J Obstet Gynecol*. 2009;200:365.e1-365.e8.
74. Kjos SL, Peters RK, Xiang A, Henry OA, Montoro M, Buchanan TA. Predicting future diabetes in Latino women with gestational diabetes. Utility of early post-partum glucose tolerance testing. *Diabetes*. 1995;44:586-591.
75. American Diabetes Association. 2. Classification and diagnosis of diabetes. *Diabetes Care*. 2016;39(suppl 1):S13-S22.
76. Salmeen K. Gestational diabetes testing: making sense of the controversy. *J Midwifery Womens Health*. 2016;61(2):203-209.
77. Committee on Practice Bulletins—Obstetrics. Practice Bulletin No. 137: gestational diabetes mellitus. *Obstet Gynecol*. 2013;122(2 pt 1):406-416.
78. Moyer VA; U.S. Preventive Services Task Force. Screening for gestational diabetes mellitus: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2014;160:414.
79. Vandorsten JP, Dodson WC, Espeland MA, et al. NIH consensus development conference: diagnosing gestational diabetes mellitus. *NIH Consensus State Sci Statements*. 2013;29(1):1-31.
80. International Association of Diabetes and Pregnancy Study Groups Consensus Panel. International Association of Diabetes and pregnancy study groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. *Diab Care*. 2010;33(3):676-682.
81. Boyd KL, Ross EK, Sherman SJ. Jelly beans as an alternative to a cola beverage containing fifty grams of glucose. *Am J Obstet Gynecol*. 1995;173(6):1889-1892.
82. Lamar ME, Kuehl ATC, Gayle J, Holleman S, Allen SR. Jelly beans as an alternative to a fifty-gram glucose beverage for gestational diabetes screening. *Am J Obstet Gynecol*. 1999;181(5):1154-1157.
83. American College of Obstetricians and Gynecologists. Practice Bulletin No. 181: prevention of Rh D alloimmunization. *Obstet Gynecol*. 2017;130:e57-e70.
84. Centers for Disease Control and Prevention. Prevention of perinatal group B streptococcal disease. Revised guidelines, 2010. Available at: https://www.cdc.gov/mmwr/pdf/rr/rr5910.pdf. Accessed July 15, 2017.
85. Scharg SJ, Verani RJ. Intrapartum antibiotic prophylaxis for the prevention of perinatal group B streptococcal disease: experience in the United States and implications for a potential group B streptococcal vaccine. *Vaccine*. 2013;31(suppl 4):D20-D26.
86. Kominiarek MA, Peasman AM. Gestational weight gain. *Am J Obstet Gynecol*. 2017. [Epub ahead of print]. doi: 10.1016/j.ajog.2017.05.040.
87. Lowensohn R, Stadler D, Naze C. Current concepts in maternal nutrition. *Obstet Gynecol Survey*. 2016;71(7):413-426.
88. Rasmussen KM, Yaktine AL, eds. *Weight Gain During Pregnancy: Reexamining the Guidelines*. Washington, DC: National Academies Press; 2009. Available at: http://www.ncbi.nlm.nih.gov/books/NBK32815. Accessed October 9, 2017.
89. Goldstein RF, Abell SK, Ranasinha S, et al. Association of gestational weight gain with maternal and infant outcomes: a systematic review and meta-analysis. *JAMA*. 2017;317:2207.

90. Siega-Riz AM, Deierlein A, Stuebe A. Implementation of the new Institute of Medicine gestational weight guidelines. *J Midwifery Womens Health*. 2010;55:515-519.
91. Harris AA. Practical advice for caring for women with eating disorders during the perinatal period. *J Midwifery Womens Health*. 2010;55(6):579-586.
92. Harris AA, Barger MK. Specialized care for women pregnant after bariatric surgery. *J Midwifery Womens Health*. 2010;55(6):529-530.
93. Liu J, Gallagher AE, Carta CM, Torres ME, Moran R, Wilcox S. Racial differences in gestational weight gain and pregnancy related hypertension. *Ann Epidemiol*. 2014;24:441-447.
94. Fontaine PL, Hellerstedt WL, Dayman CE, Wall MM, Sherwood NE. Evaluating body mass index–specific trimester weight gain recommendations: differences between black and white women. *J Midwifery Womens Health*. 2012;57:327-335.
95. Pritkin R. Nutritional support in obstetrics and gynecology. *Obstet Gynecol*. 1976;19(3):489-513.
96. Barger MK. Maternal nutrition and perinatal outcomes. *J Midwifery Womens Health*. 2010;55(6):502-511.
97. Langley-Evans SC. Nutrition in early life and the programming of adult disease: a review. *J Human Nutr Diet*. 2015;28(suppl 1):1-14.
98. U.S. Food and Drug Administration. Food safety for moms-to-be. Available at: https://www.fda.gov/food/foodborneillnesscontaminants/peopleatrisk/ucm081785.htm. Accessed July 4, 2017.
99. Wolff T, Witkop CT, Miller T, Syed SB. *Folic Acid Supplementation for the Prevention of Neural Tube Defects: An Update of the Evidence for the U.S. Preventive Services Task Force. Evidence Synthesis No. 70*. AHRQ Publication No. 09-051132-EF-1. Rockville, MD: Agency for Healthcare Research and Quality; May 2009.
100. Goh YI, Bollano E, Einarson TR, Koren G. Prenatal multivitamin supplementation and rates of congenital anomalies: a meta-analysis. *J Obstet Gynaecol Can*. 2006;28:680-688.
101. Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. *MMWR*. 1998;47:1-29.
102. Pena-Rosas JP, Viteri FE. Effects of routine oral iron supplementation with or without folic acid for women during pregnancy. *Cochrane Database Syst Rev*. 2006;3:CD004736. doi: 10.1002/14651858.CD004736.pub2.
103. Graves BW, Barger MK. A “conservative” approach to iron supplementation during pregnancy. *J Midwifery Womens Health*. 2001;46:159-166.
104. Santiago P. Ferrous versus ferric oral iron formulations for the treatment of iron deficiency: a clinical review. *Sci World J*. 2012;846824.
105. Leung AM, Pearce EN, Braverman LE. Iodine nutrition in pregnancy and lactation. *Endocrin Metabol Clin*. 2011;40(4):765-777.
106. De-Regil LM, Palacios C, Lombardo LK, Peña-Rosas JP. Vitamin D supplementation for women during pregnancy. *Cochrane Database Syst Rev*. 2016;1:CD008873. doi: 10.1002/14651858.CD008873.pub3.
107. Gill SK, Maltepe C, Koren G. The effectiveness of discontinuing iron-containing prenatal multivitamins on reducing the severity of nausea and vomiting of pregnancy. *J Obstet Gynaecol*. 2009;29(1):13-26.
108. Penny DS, Miller KG. Nutritional counseling for vegetarians during pregnancy and lactation. *J Midwifery Womens Health*. 2008;53(1):37-44.
109. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Updated US Department of Agriculture food patterns meet goals of the 2010 dietary guidelines. *J Acad Nutr Diet*. 2012;112(10):1648-1655.
110. Jordon RG. Prenatal omega-3 fatty acids: review and recommendations. *J Midwifery Womens Health*. 2010;55:520-528.
111. Gunaratne AW, Makrides M, Collins CT. Maternal prenatal and/or postnatal n-3 long chain polyunsaturated fatty acids (LCPUFA) supplementation for preventing allergies in early childhood. *Cochrane Database Syst Rev*. 2015;7:CD010085. doi: 10.1002/14651858.CD010085.pub2.
112. American College of Obstetricians and Gynecologists. Practice Bulletin No. 105: bariatric surgery and pregnancy. *Obstet Gynecol*. 2009;113(6):1405-1413.
113. Goodnight W, Newman R. Optimal nutrition for improved twin pregnancy outcome. *Obstet Gynecol*. 2009;114(5):1121-1134.
114. Fawcett EJ, Fawcett JM, Mazmanian D. A meta-analysis of the worldwide prevalence of pica during pregnancy and the postpartum period. *Int J Gynaecol Obstet*. 2016;133(3):277-283.
115. Breedlove G, Frzyelka D. Depression screening during pregnancy. *J Midwifery Womens Health*. 2011;56(1):18-25.
116. American College of Obstetricians and Gynecologists. Committee Opinion No. 518: intimate partner violence. *Obstet Gynecol*. 2012;119:412-417.
117. Chisholm CA, Bullock L, Ferguson JE. Intimate partner violence and pregnancy: screening and intervention. *Am J Obstet Gynecol*. 2017;217(2):145-149.
118. Renker PR. Breaking the barriers: the promise of computer-assisted screening for intimate partner violence. *J Midwifery Womens Health*. 2008;53:496-503.
119. Barona LK, Lovelace D, Daniels JL, McDaniel L. Tobacco harms: nicotine pharmacology and pharmacologic smoking

cessation interventions for women. *J Midwifery Womens Health*. 2017;62:253-269.
120. Azulay Chertok IR, Archer SH. Evaluation of a midwife- and nurse-delivered 5 A's prenatal smoking cessation program. *J Midwifery Womens Health*. 2015;60:175-181.
121. Coleman T, Chamberlain C, Davey MA, Cooper SE, Leonardi-Bee J. Pharmacological interventions for promoting smoking cessation during pregnancy. *Cochrane Database Syst Rev.* 2015;12:CD010078. doi: 10.1002/14651858.CD010078.pub2.
122. DeLong NE, Barra NG, Hardy DB, Holloway AC. Is it safe to use smoking cessation therapeutics in pregnancy? *Expert Opin Drug Saf*. 2014;13(12):1721-1731.
123. Tan C, Denny C, Cheal N, Sniezek J, Kanny D. Alcohol use and binge drinking among women of childbearing age—United States, 2011–2013. *MMRW.* 2015;64(37);1042-1046.
124. Shogren M. Substance abuse screening and brief interventions: an introduction to screening, brief intervention, and referral to treatment in women's health. *J Midwifery Womens Health*. 2017;62(6):746-754.
125. Reddy UM, Davis JM, Ren Z, Greene MF; Opioid Use in Pregnancy, Neonatal Abstinence Syndrome, and Childhood Outcomes Workshop Invited Speakers. Opioid use in pregnancy, neonatal abstinence Syndrome, and childhood outcomes: executive summary of a joint workshop by the Eunice Kennedy Shriver National Institute of Child Health and Human Development, American College of Obstetricians and Gynecologists, American Academy of Pediatrics, Society for Maternal-Fetal Medicine, Centers for Disease Control and Prevention, and the March of Dimes Foundation. *Obstet Gynecol*. 2017;130(1):10-28.
126. Goodman DJ, Wilff KB. Screening for substance abuse in women's health: a public health imperative. *J Midwifery Womens Health*. 2013;58(3):278-287.
127. Volkow ND, Compton WM, Wargo EM. The risks of marijuana use during pregnancy. *JAMA*. 317(2):129-130.
128. Foeller ME, Lyell DJ. Marijuana use in pregnancy: concerns in an evolving era. *J Midwifery Womens Health*. 2017;62(2):363-367.
129. American College of Obstetricians and Gynecologists. Committee Opinion No. 722: marijuana use during pregnancy and lactation. *Obstet Gynecol.* 2017;130(4):e205-e209.
130. Freeman M, Ghidini A, Spong CY, Tchabo N, Bannon PZ, Pezzullo JC. Does air travel affect pregnancy outcome? *Arch Gynecol Obstet.* 2004;269:274.
131. Duong HT, Shahrukh Hashmi S, Ramadhani T, Canfield MA, Scheuerle A, Kim Waller D; National Birth Defects Prevention Study. Maternal use of hot tub and major structural birth defects. *Birth Defects Res.* 2011;91(pt 1):836-841.
132. Harvey MA, McRorie MM, Smith DW. Suggested limits to the use of the hot tub and sauna by pregnant women. *Can Med Assoc J*. 1981;125:50-53.
133. Oral Health Care During Pregnancy Expert Workgroup 2012. Oral health care during pregnancy: A national consensus statement−summary of an expert workgroup meeting. Washington, DC: National Maternal Child Oral Health Center.
134. Motozawa Y, Hitosugi M, Abe T, Tokudome S. Effects of seat belts worn by pregnant drivers during low-impact collisions. *Am J Obstet Gynecol.* 2010;203:62.e1.
135. Sayle AE, Savitz DA, Thorp JM Jr, Hertz-Picciotto I, Wilcox AJ. Sexual activity during late pregnancy and risk of preterm delivery. *Obstet Gynecol.* 2001;97(2):283-289.
136. Centers for Disease Control and Prevention. Reproductive health and the workplace. Available at: https://www.cdc.gov/niosh/topics/repro/specificexposures.html. Accessed September 22, 2017.
137. MacDonald LA, Waters TR, Napolitano PG, et al. Clinical guidelines for occupational lifting in pregnancy: evidence summary and provision recommendations. *Am J Obstet Gynecol*. 2013;209(2):80-88.
138. Waters TR, MacDonald LA, Hudock SD, Goddard DE. Provisional recommended weight limits for manual lifting during pregnancy. *Hum Factors*. 2014;56(1):203-214.
139. Jackson RA, Gardner S, Torres LN, Huchko MJ, Zlatnik MG, Williams JC. My obstetrician got me fired: how work notes can harm pregnant patients and what to do about it. *Obstet Gynecol.* 2015;126(2):250-254.
140. Peretz J, Vrooman L, Ricke WA, et al. Bisphenol A and reproductive health: update of experimental and human evidence, 2007–2013. *Environ Health Perspect.* 2014;122(8):775-786.
141. International Commission on Radiological Protection, eds. *Annals of the ICRP, Publication 84: Pregnancy and Medical Radiation*, 30(1). Tarrytown, NY: Pergamon/Elsevier Science; 2000.
142. Williams PM, Fletcher S. Health effects of prenatal radiation exposure. *Am Fam Physician*. 2010;82(5):488-493.
143. American College of Obstetricians and Gynecologists. Committee Opinion No. 656: guidelines for diagnostic imaging in pregnancy. *Obstet Gynecol.* 2016;127:e75-e80.
144. Alba A, Carlton L, Dinkel L, Ruppe R. Increased lead levels in pregnancy among immigrant women. *J Midwifery Womens Health*. 2012;57:509-514.
145. Ettinger AS, Wengrovitz AG, eds. *Guidelines for the Identification and Management of Lead Exposure in Pregnant and Lactating Women*. Atlanta, GA: Centers for Disease Control and Prevention; 2010. Available at: http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf. Accessed July 4, 2017.
146. Shaw GM. Adverse human reproductive outcomes and electromagnetic fields: a brief summary of the epidemiologic

literature. *Bioelectromagnetics*. 2001;5(suppl):S5.
147. Oduyebo T, Polen KD, Walke HT, et al. Update: interim guidance for health care providers caring for pregnant women with possible Zika virus exposure—United States (including U.S. territories), July 2017. *MMWR*. July 24, 2017. doi: http://dx.doi.org/10.15585/mmwr.mm6629e1.
148. Delgado AR. Listeriosis in pregnancy. *J Midwifery Womens Health*. 2008;53(3):255-259.
149. Villazanakretzer DL, Napolitano PG, Cummings KF, Magann EF. Fish parasites: a growing concern during pregnancy. *Obstet Gynecol Surv*. 2016;71(4):253-259.
150. Meaney-Delman D, Rasmussen SA, Staples JE, et al. Zika virus and pregnancy: what obstetric health care providers need to know. *Obstet Gynecol*. 2016;124(4):642-648.
151. Madjunkoy M, Chaudhry S, Ito S. Listeriosis during pregnancy. *Arch Gynecol Obstet*. 2017; 296(2):143-152.
152. Sugishita Y, Akiba T, Sumitomo M, et al. Shedding of rubella virus among infants with congenital rubella syndrome born in Tokyo, Japan, 2013–2014. *Jpn J Infect Dis*. 2016;69(5):418-423.
153. Neu N, Duchon J, Zachariah P. TORCH infections. *Clin Perinatol*. 2015;42(1):77-103.
154. Feldman DM, Keller R, Borgida AF. Toxoplasmosis, parvovirus and cytomegalovirus in pregnancy. *Clin Lab Med*. 2016;36(2):407-419.
155. Kim DK, Riley LE, Harriman KH, Hunter P, Bridges CB; ACIP Adult Immunization Work Group, Centers for Disease Control and Prevention. Advisory Committee on Immunization Practices recommended immunization schedule for adults aged 19 years and older—United States, 2017. *MMWR Surveill Summ*. 2017;66:5.
156. Lloyd K. Protecting pregnant women, newborns, and families against pertussis. *J Midwifery Womens Health*. 2013;58(3):288-296.
157. Thompson WW, Price C, Goodson B, et al.; Vaccine Safety Datalink Team. Early thimerosal exposure and neuropsychological outcomes at 7 to 10 years. *N Engl J Med*. 2007;357(13):1281-1292.
158. Price CS, Thompson WW, Goodson B, et al. Prenatal and infant exposure to thimerosal from vaccines and immunoglobulins and risk of autism. *Pediatrics*. 2010;126:656-664.
159. Zwelling E. Childbirth education in the 1990s and beyond. *JOGNN*. 1996;25(5):425-432.
160. Walker DS, Visger JM, Rossie D. Contemporary childbirth education models. *J Midwifery Womens Health*. 2009;54(6):469-471.
161. Dyson L, McCormick F, Renfrew M. Interventions for promoting the initiation of breastfeeding. *Cochrane Database Syst Rev* 2005;(2):CD001688.
162. Task Force on Sudden Infant Death Syndrome. SIDS and other sleep-related infant deaths: updated 2016 recommendations for a safe infant sleeping. *Pediatrics*. 2016;138(5). pii: e20162940.

APPENDIX

21A

Abdominal Examination During Pregnancy

TEKOA L. KING and CECILIA M. JEVITT

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The components of the abdominal examination in pregnancy include (1) abdominal inspection and palpation; (2) measurement of fundal height; (3) Leopold's maneuvers for determining fetal lie, presentation, position, variety, and engagement; (4) estimation of fetal weight; and (5) detection of fetal heart tones.

While ultrasound can effectively be used to assess fetal growth and position—and should be used when discrepancies arise or assessment is challenging—being able to employ one's hands to gain information about the progress of a pregnancy is an invaluable skill. The standard descriptions in this appendix assume a woman of normal habitus and a singleton pregnancy. Maternal height and weight, uterine abnormalities, fetal growth pattern, and multiple pregnancies are all factors that can challenge interpretation of clinical findings and their effects are discussed.

Equipment

- Measuring tape with centimeter increments
- Pinard, Allen, DeLee, or Doppler fetoscope

Abdominal Inspection

The following aspects of the abdomen are noted during inspection:

1. Surgical scars. The scar of a previous cesarean section is of particular importance; it may be either vertical or horizontal. It is useful to know if the woman has undergone an appendectomy so that appendicitis can be ruled out in the event of right-sided abdominal pain during the pregnancy.
2. Bruises or abrasions. Document a description and location of any bruises and discuss their origin with the woman, paying particular attention to the possibility of intimate-partner violence and abuse.
3. Linea nigra. This hyperpigmented vertical line on the lower abdomen may be visible by approximately 20 weeks' gestation, or it may be faintly present from a previous pregnancy and begin to darken in the first trimester. The linea nigra will fade during the first 6 months postpartum.
4. Striae gravidarum. Many women develop these pink or red streaks during pregnancy as the dermis is stretched. Silvery white streaks may be present from previous weight changes or previous pregnancy.
4. Abdominal contours give some initial indication of the fetal lie, presentation, position, and attitude, although none of the following observations is diagnostic:
    a. Uterine shape is a longitudinal ovoid: Fundal height in the expected range suggests a longitudinal lie.
    b. Uterine shape is a transverse ovoid: Fundal height lower than expected for gestational age may indicate a transverse lie, fetal growth restriction, or oligohydramnios.
    c. A long smooth curve prominent on one side of the abdomen suggests that the back of the fetus is on that side of the abdomen.
    d. A saucer-like depression appearing just below the umbilicus and a bulge like a full bladder appearing above the symphysis pubis suggests a possible occiput posterior position or maternal full bladder.
    e. Movement of fetal small parts seen all over the abdomen suggests an occiput posterior position.

Abdominal Palpation

The midwife may palpate these structures during physical examination:

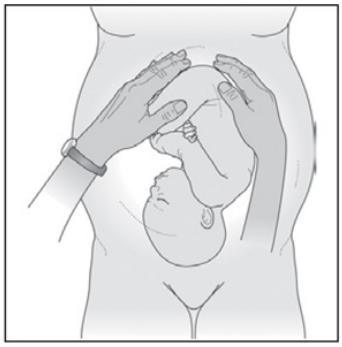
1. Diastasis recti (midline separation of the rectus abdominus muscle) presence and width are palpated by placing one or two fingers parallel to the abdominal midline, just below the epigastric region, and feeling the separation of the rectus abdominis muscle after asking the woman to lift her head while lying supine. This examination is routinely performed at the postpartum visit but may also be useful at the first prenatal visit, especially for a multiparous woman, as she may have a persistent diastasis recti.
2. Umbilical hernia (intestinal protrusion through a separation in the abdominal muscles behind the umbilicus) can be detected on palpation at any time during pregnancy.
3. Uterine muscle tone (hard versus soft), contractions (palpable or not palpable), and

tenderness are evaluated to assess for signs of preterm labor in the second and third trimesters, for signs of labor after 37 weeks' gestation, and for signs of infection.

4. Amniotic fluid volume. When performing an abdominal examination, the midwife can estimate the quantity of amniotic fluid by placing one hand on each side of the uterus and gently pushing first on one side of the uterus, then the other. A fluid wave or thrill might be palpated depending on gestational age, size and position of the fetus, and amount of fluid present. If a woman has polyhydramnios, it will be difficult to palpate small parts of the fetus and may be difficult to determine fetal position. The opposite will be true if a woman has oligohydramnios.

Leopold's Maneuvers

The process of evaluating fetal lie, position, and presentation with Leopold's maneuvers also offers the opportunity to assess the abdomen for muscle tone, uterine tone and contractility, and palpable fetal movement, and to estimate fetal weight. The four Leopold's maneuvers and the combined Pawlik's grip are shown in **Figure 21A-1**.





First maneuver. Identify the part of the fetus that resides in the fundus. Curve fingers of both hands around the top of the fundus.



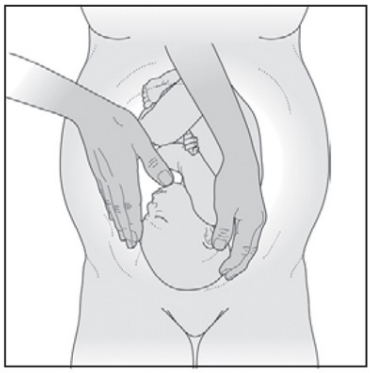


Second maneuver. Identify the location of the small parts and fetal back. Place both hands on the side of the uterus.





Third maneuver. Identify the presenting part. With the thumb and middle finger of one hand, press gently but deeply into the woman's abdomen immediately above the symphysis pubis and grasp the presenting part.





Fourth maneuver. Check whether the presenting part is engaged and the location of the cephalic prominence. Place both hands on the sides of the lower uterus, press deeply, and move the fingertips toward the pelvic inlet.





Combined Pawlik's grip. Combine Leopold's third maneuver with one hand and palpation of the fundus using the other hand. Compare the two poles for final determination of lie and presentation.

Figure 21A-1 The four Leopold's maneuvers and the combined Pawlik's grip.

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Key Points

1. The woman's bladder should be empty.
2. The woman's abdomen is typically exposed from just below the breasts to the symphysis pubis. Provide privacy and consider cultural variations in comfort with exposure of skin. Ask the woman to move her clothing instead of doing it for her. This allows her to control the extent and amount of skin exposure.
3. Relaxation of the abdominal muscles can be facilitated by:
   a. Placing a pillow under the woman's head and upper shoulders
   b. Asking her to place her arms by her sides or across her chest

c. Having her bend her knees slightly

4. Before palpating, the midwife lightly rests her hand on the woman's abdomen (**Figure 21A-2**). This action gives the woman an opportunity to adjust to the sensation of the midwife's touch and allows any initial muscle-tightening reaction to dissipate.



Figure 21A-2 Midwife preparing to do an abdominal palpation.

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Procedure for Leopold's Maneuvers

First Maneuver: Determine Fetal Lie and Presentation

Facing the woman's head, palpate the area of the uterine fundus, noting the shape of the fetal part in the fundus. A fetal part that feels round and hard, and that can be balloted between the midwife's fingers or hands, suggests a fetal head. The mobility arises from the head being able to move independently of the trunk. A fetal part that feels irregular, larger or bulkier, and less firm than a head, and that cannot be well delineated or readily moved or balloted, suggests the fetal breech. The breech cannot move independently of the trunk. In these cases the lie is longitudinal. If neither of the preceding conditions is felt in the fundus, a transverse lie may be present.

Second Maneuver: Determine Location of Fetal Back

1. Continue to face the woman's head. Place the hands on both sides of the uterus about midway between the symphysis pubis and the fundus.
2. Apply slight pressure with one stabilizing hand against the side of the uterus, thereby

pushing the fetus to the other side of the abdomen against the examining hand and stabilizing it there. Maintain this pressure while the examining hand palpates the side of the uterus to identify fetal parts.

3. Palpate the entire area with the examining hand, from the abdominal midline to the lateral side, and from the fundus to the symphysis. Use firm, smooth pressure and rotary movement.
4. Reverse the procedure to examine the other side of the uterus.
5. The flat palmar surfaces of the fingers are used for palpating—*not* the fingertips. Smooth, deep pressure is used—as firm as is necessary to obtain accurate findings without causing pain or discomfort.

A firm, convex, continuously smooth, and resistant mass extending from the breech to the neck indicates the fetal back. The location of the back in the anterior, lateral, or posterior portion of the abdomen helps to determine the variety (position). Small, knobby, irregular masses that might move when pressed on suggest the fetal small parts—hands, feet, knees, elbows. When palpating the uterus, the fetal small parts should be opposite the fetal back. If the back is difficult to feel and seems to be just out of reach in the posterior portion of the abdomen, and small parts are palpable all over the abdomen, a posterior position may be present.

Third Maneuver: Determine the Fetal Presentation and Engagement

The third maneuver is called *Pawlik's grip*. Clasp the lower abdomen immediately above the symphysis pubis between the thumb and middle finger of one hand with the thumb and middle finger spread so they start the procedure as far laterally into the inguinal area as possible. It will be necessary to press gently but firmly into the abdomen to feel the presenting part between the thumb and finger. Pawlik's grip can be uncomfortable and may be omitted as the fetal presentation can be determined with the fourth maneuver. A kicking motion palpated in the fundus during Pawlik's grip increases confidence that the presentation is cephalic.

If the fetal head is above the pelvic brim, it is readily movable and ballottable, as described for the first maneuver. A procedure that is sometimes added to this maneuver is called the combined Pawlik's grip, in which Pawlik's grip is utilized with one hand and the fundus is grasped in the same way with the other hand at the same time (**Figure 21A-3**). This combination enables the simultaneous comparison of what is in the two poles for final determination of the fetal lie and presentation.



Figure 21A-3 Combined Pawlik's grip.

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Fourth Maneuver: Cephalic Prominence and Fetal Attitude

1. Turn and face the woman's feet.
2. Place both hands on the sides of the uterus with the palms just above the symphysis and iliac crest with the fingers directed toward the symphysis pubis.
3. Press with the fingertips into the lower abdomen slowly but firmly, and move them toward the pelvic inlet to determine the *cephalic prominence*; note if it is on the side of the fetal back or fetal front. If the cephalic prominence is on the same side as the fetal small parts, the fetal *attitude* is flexed and the fetal head is tucked toward the chin. If the presentation is vertex and the cephalic prominence is on the same side as the fetal back, the fetus is in a face or brow presentation.

At the conclusion of the four maneuvers, share the findings with the woman, and offer to help her feel and identify various parts of her fetus if she would like.

Estimation of Fetal Weight

Estimated fetal weight (EFW) is used during the prenatal period, particularly after 30 weeks' gestation, as one clinical measurement in the overall evaluation of gestational age and progressive fetal growth. During Leopold's maneuvers, the midwife can compare the palpated fetal size to known volumes such as a liter bag of IV fluid, a 5-pound bag of flour, or a gallon jug of milk to estimate size. Neither experienced hands nor an ultrasound is necessarily more

accurate than the other method when assessing fetal weight in a full-term fetus. The EFW is important in the intrapartum period, when this estimation is compared with the clinical evaluation of the pelvis to ascertain the adequacy of a woman's pelvis. Estimating fetal weights and comparing those estimations to ultrasound estimations or birth weights for feedback sharpens a midwife's skill in estimating fetal weight.

Auscultation of Fetal Heart

The sound of the fetal heart is transmitted best through the convex portion of the fetus closest to the anterior uterine wall, which is usually the fetal back. Thus, if the position of the fetus is known, fetal heart tones can be readily located, allowing for some variation depending on how far the fetus has descended into the pelvis (**Figure 21A-4**). Location of the fetal heart is an additional piece of data that either confirms or calls into question the diagnosis of fetal presentation and position.

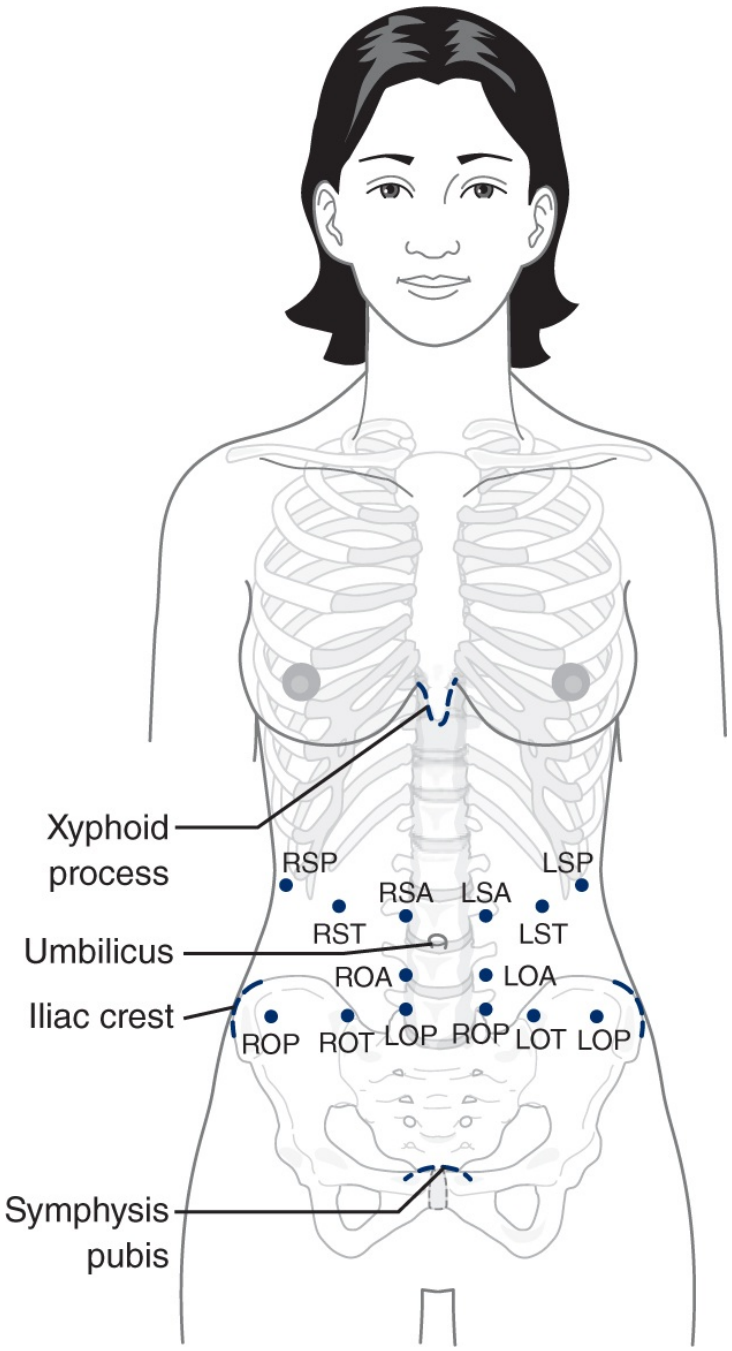


Figure 21A-4 Location of the point of maximum intensity of auscultation of the fetal heart tones for specific fetal presentations and positions.

Abbreviations: LOA, left occiput anterior; LOP, left occiput posterior; LOT, left occiput transverse; LSA, left sacrum anterior; LSP, left sacrum posterior; LST, left sacrum transverse; ROA, right occiput anterior; ROP, right occiput posterior, ROT, right occiput transverse; RSA, right sacrum anterior; RSP, right sacrum posterior; RST, right sacrum transverse.

Measuring Fundal Height

Fundal heights are measured at each prenatal visit. Serial fundal height measurements provide information about the enlargement of the uterus and, therefore, about the growth of the fetus.

The height of the fundus, measured in centimeters from the top of the symphysis pubis to the top of the fundus, is used to monitor fetal growth and can be a screening tool for detection of multifetal gestation, fetal growth restriction, polyhydramnios, oligohydramnios, and other complications of fetal growth. Fundal height is of greatest value when it is measured the same way by the same midwife at successive prenatal visits.

The fundal height (measured in centimeters) and gestational age (expressed in weeks from the last menstrual period [LMP]) is closely correlated between 20 and 32 weeks' gestation, when the number of weeks' gestation is close to the number of centimeters measured between the symphysis and the fundus.[1-6] However, the correlation between weeks' gestation and fundal heights depends on several factors, including maternal habitus and fetal lie. For example, a petite woman who is only 5 feet (1.5 meters) tall may have different fundal measurements than a taller woman of 6 feet (1.8 meters) in height at the same point in pregnancy. Similarly, women with large deposition of abdominal adipose tissue may have a fundal height measurement that is larger than expected. **Table 21A-1** lists the approximate expected location of the fundal height at different gestational ages for a woman with a normal body mass index (BMI) and singleton fetus in a longitudinal lie.

Table 21A-1 Approximate Expected Fundal Height at Specific Weeks' Gestation

Weeks' Gestation	Approximate Expected Fundal Height
12	Level of the symphysis pubis
16	Halfway between symphysis pubis and umbilicus
20	Within 1 fingerbreadth of umbilicus
24	2–4 fingerbreadths above umbilicus
28–30	One-third of the way between umbilicus and xiphoid process (3 fingerbreadths above umbilicus)
32	Two-thirds of the way between umbilicus and xiphoid process (3–4 fingerbreadths below xiphoid process)
36–38	1 fingerbreadth below xiphoid process
40	2–3 fingerbreadths below xiphoid process if lightening has occurred

In clinical practice, it is common to use a rule of gestational age plus or minus 2 centimeters to diagnose adequate fetal growth. That is, during 20–32 weeks' gestation, a woman should have a fundal measurement in centimeters that is within 2 cm of the number of weeks' gestation. No strong evidence supports this approach, most likely because of the maternal and fetal variations noted earlier. Despite a lack of evidence, deviation from the 2-cm rule of thumb can be used as an alert to carefully assess for size–dates discrepancy. Assessment of size–dates discrepancies first includes remeasurement when the woman has an empty bladder, as a full bladder can elevate the uterus. Ultrasound is used to diagnose the etiology of a size–dates discrepancy.

Key Points

1. Early in pregnancy, the uterus is contained within the pelvic girdle. The uterine fundus can be palpated abdominally by 10 to 12 weeks' gestation just at or above the symphysis pubis. Prior to 18 to 20 weeks' gestation, the uterine size is best described in relation to the level of the uterine fundus between the symphysis pubis and the umbilicus, using the midwife's fingerbreadth (fb) as the measuring tool (e.g., 1 fb above symphysis, 4 fb below umbilicus). (For more information, see the *Anatomy and Physiology of Pregnancy* chapter.)
2. Between 20 and 32 weeks' gestation, the measurement should be within 2 cm of the number of weeks' gestation.
3. In the last few weeks of pregnancy, as the fetal presenting part moves into the pelvis, more variation is seen between measurements, and the absolute fundal height may remain stable or decrease slightly.
4. Used alone, the fundal height measurement may be specific, but lacks sensitivity. When a measurement is not within the expected value for the woman's gestational age:
   a. First assess for confounding factors such as a full bladder or excess abdominal adipose tissue.
   b. Refer the woman for ultrasound evaluation if confounding factors do not sufficiently account for the size–dates discrepancy.

Procedure for Measuring Fundal Height

Fundal height can be measured in a number of different ways. For an extensive review of fundal height measurement, the interested reader is referred to the classic series of articles written by Engstrom et al.[1-7]

1. Assessment of the fundal height should take place with the woman in a supine or semi-recumbent position.
2. Some midwives advocate turning the tape measure to hide the centimeter markings, thereby minimizing inadvertent bias by the midwife. Alterations in maternal position, a full urinary bladder, multiple examiners, and provider awareness of the weeks' gestation all create bias that can lead to inaccurate measurements.
3. Before measuring, the symphysis pubis and uterine fundus are identified (**Figure 21A-5**).
   a. The woman may need to partially remove her clothing to allow access to the symphysis and fundus. The abdomen is exposed from the xiphoid process to the top of the pelvis.
   b. Assessment is done with a gentle, firm touch using the palmar surface of the fingers.
   c. Facing the woman's head as she lies in a semi-reclining position, the midwife's hands are placed on each lateral side of the uterus approximately midway between the symphysis and the fundus, with the fingers pointed vertically toward the woman's head.
   d. The uterine fundus is identified by walking one's hands up the sides of the uterus until they meet at the top. Smooth motions avoid the sensation of kneading or poking and will decrease uterine contractility during the examination.

e. Measuring “over the top” of the fundus is a common error that can be avoided by paying attention to the curve of the uterus, the location of the fetal part highest in the uterus, and any laxity of the maternal abdomen.

4. Two techniques are commonly employed:
   a. Place the zero line of the tape measure on the superior border of the symphysis and measure *upward* in a straight line, following the abdominal midline to the crest of the uterine fundus, and taking care not to cross over the top and begin to measure the posterior aspect of the uterus.
   b. After identifying the fundus, place the tape at that point and measure *downward* to the superior border of the symphysis.

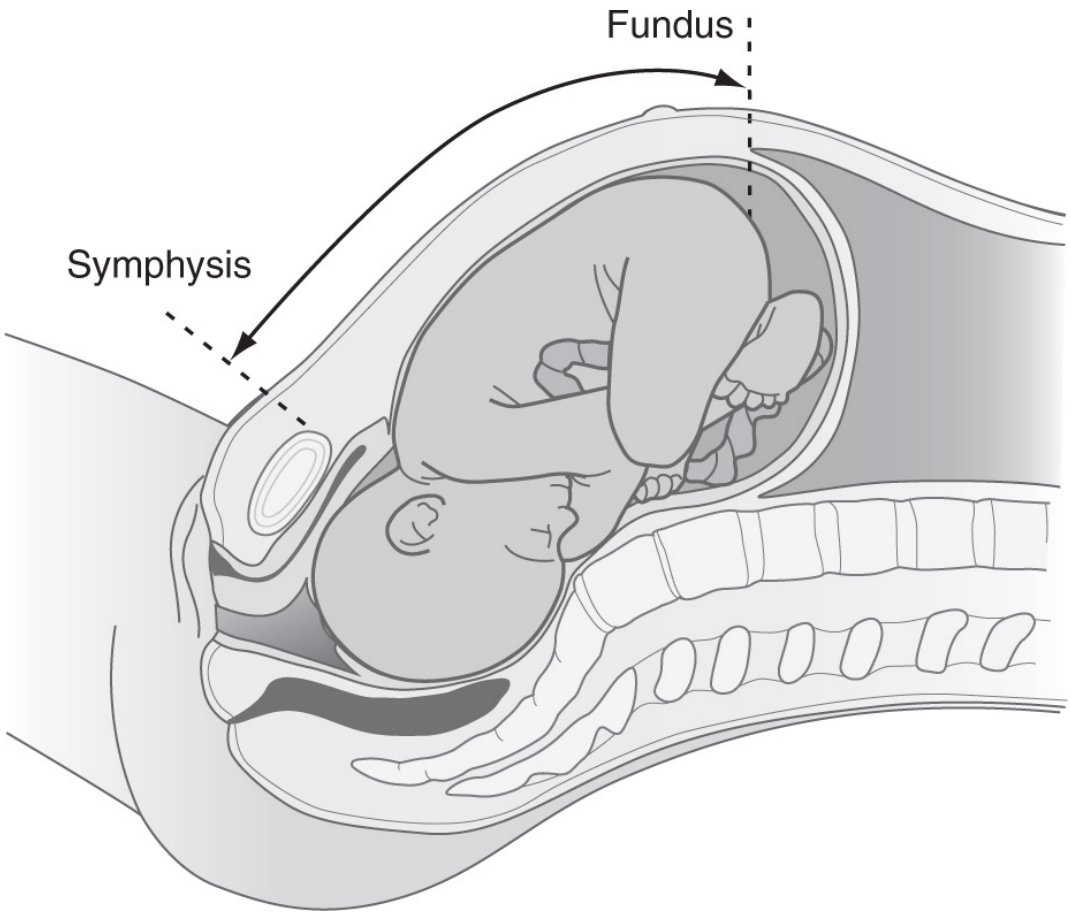


Figure 21A-5 Measurement of fundal height.

References

1. Engstrom JL. Measurement of fundal height. *J Obstet Gynecol Neonatal Nurs.* 1988;17(3):172-178.
2. Engstrom JL, Ostrenga KG, Plass RV, Work BA. The effect of maternal bladder volume on fundal height measurements. *Br J Obstet Gynaecol.* 1989;96(8):987-991.
3. Engstrom JL, Sittler CP. Fundal height measurement. Part 1: techniques for measuring fundal height. *J Nurse-Midwifery.* 1993;38(1):5-16.
4. Engstrom JL, McFarlin BL, Sittler CP. Fundal height measurement. Part 2: intra- and interexaminer reliability of three measurement techniques. *J Nurse-Midwifery.* 1993;38(1):17-22.
5. Engstrom JL, Piscioneri LA, Low LK, McShane H, McFarlin B. Fundal height measurement. Part 3: the effect of

maternal position on fundal height measurements. *J Nurse-Midwifery.* 1993;38(1):23-27.
6. Engstrom JL, McFarlin BL, Sampson MB. Fundal height measurement. Part 4: accuracy of clinicians' identification of the uterine fundus during pregnancy. *J Nurse-Midwifery.* 1993;38(6):318-323.
7. Engstrom JL, Sittler CP, Swift KE. Fundal height measurement. Part 5: the effect of clinician bias on fundal height measurements. *J Nurse-Midwifery.* 1994; 39(3):130-141.

APPENDIX

21B

Clinical Pelvimetry

JENNIFER M. DEMMA, KAREN TRISTER GRACE, AND TEKOA L. KING

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Pelvimetry comes from the Latin word *pelvis* (meaning "basin or bowl") and the Greek word *metron* (meaning "measure").[1] Thus, pelvimetry refers to measurement of diameters of the pelvis. It is possible to evaluate the general structure (size and shape) of the pelvis and measure some of the critical diameters of the pelvis during a vaginal examination. This assessment, which is referred to as "clinical pelvimetry," is classically done when an initial vaginal examination is performed during pregnancy and/or during the course of labor.

Pelvic diameters are most accurately measured by X ray or computerized tomographic (CT) X ray. However, X-ray pelvimetry is no longer used because of the potential hazards of exposing the fetus to radiation and lack of research supporting this method's accuracy. CT scans are reserved for prenatal assessment of the pelvis in women who plan a vaginal breech birth.

Historically, clinical pelvimetry was an essential piece of information that was used to guide clinical management. A woman whose pelvis was contracted in one or more dimensions was likely to have cephalopelvic disproportion (CPD) in labor and could be assisted with a cesarean section or use of high or mid-forceps.[2] The shape and size of the pelvis partly determined which method of birth would be safest. Because diseases such as rickets, scurvy, and polio were more common in the past, CPD was a more frequent occurrence and clinical dilemma.[3]

Today the diagnosis of CPD is not made until there is a failure of progress or descent in labor; thus, the predictive value of clinical pelvimetry is controversial.[4,5] Nevertheless, it is important that we do not "throw the baby out with the bath water": The results of clinical pelvimetry can help confirm cephalopelvic disproportion and, therefore, inform and help guide intrapartum management. In addition, clinical pelvimetry continues to have an important role in low-resource nations, wherein a contracted pelvis is more common.[3]

Key Points

1. The midwife should have an understanding of the four classic pelvic shapes and dimensions of the pelvic planes, as described in the *Anatomy and Physiology of the Female Reproductive System* chapter.
2. Before doing clinical pelvimetry, it is important that the midwife measure the lengths of the fingers as well as the width of the fist, as these lengths will used to determine specific measurements.
   a. The length of the reach of the examining fingers is measured from the tip of the longest finger to the juncture of the first finger (palm) and thumb.
   b. The fist is measured from the lateral (ulnar) aspect to the medial (radial) aspect of the tops of the knuckles of the fingers where they attach to the hand. If this does not measure at least 8 cm, then position the thumb in the way that it will be positioned each time and add the joint or knuckle of the thumb into the measurement.
3. Clinical pelvimetry is best performed with the thumb of the examining hand tucked into the palm. If the thumb is extended, it will come in contact with the clitoris or another part of the external vulva, which disallows use of the full length of the examining fingers in reaching for the sacrum or diagonal conjugate.
4. The procedure for performing clinical pelvimetry can be practiced using a bony pelvis model but must be performed consistently and methodically at the completion of a vaginal examination to truly understand the effects of soft tissue and movements that cause discomfort.
5. Some midwives believe that it is necessary to palpate and evaluate completely both sides of the bony pelvis. Others believe that it is sufficient to palpate only one side of the bony pelvis and assume both sides are equilateral, unless an obvious pelvic deformity is observed or the woman has a history of pelvic trauma. In such cases, both sides of the bony pelvis should be evaluated.
6. Clinical pelvimetry may be performed in several different ways. Some texts advocate assessing the diagonal conjugate first and moving from the back of the pelvis forward; others advocate the reverse order. Some texts do not include assessment of the retropubic arch or sidewalls. The order of maneuvers recommended in this text is designed to cause a woman the least discomfort.
7. Clinical pelvimetry uses measures of the length and shape of pelvic structures to indirectly assess the size of the three pelvic planes as well as the back and forepelvis. The pelvic planes cannot be measured directly. Thus, clinical pelvimetry is used to categorize the pelvis as adequate, borderline, or contracted for the birth of an average-size fetus by determining the shape and size of the pelvic inlet, midplane, and outlet.

Procedure for Performing Clinical Pelvimetry

The procedure for performing clinical pelvimetry is presented in **Table 21B-1**. The pelvis is palpated from the forepelvis posteriorly toward the sacrum, and the shape and length of the following structures are noted: symphysis pubis, ischial rami, sidewalls, ischial spines, interspinous diameter, sacrospinous ligament, coccyx, sacrum, and diagonal conjugate. The

pubic arch is measured as the midwife's hand is removed from the vagina, and the intertuberous diameter is measured externally at the end of the examination. **Figure 21B-1** and **Figure 21B-2** show the pelvic diameters and shape of the sacrum as described in **Table 21B-1**.

Table 21B-1 Procedure for Performing Clinical Pelvimetry

Step	Obstetric Plane	Area Measured	Technique	Description of Findings	Clinical Implications During Labor
1	Forepelvis and AP diameter of the pelvic inlet	Retropubic arch	Insert two fingers just inside the vagina; rotate so the palmar surface is up. Separate fingers gently to avoid trauma to the urethra, and gently palpate the posterior aspect of the symphysis pubis to determine the inclination.	*Parallel inclination*: The longitudinal axis of the symphysis pubis is parallel to the sacrum. *Anterior inclination*: The superior margin of the symphysis pubis is tilted toward the sacral promontory, and the inferior margin is tilted away from the sacrum. *Posterior inclination:* The inferior margin of the symphysis pubis is tilted toward the sacrum, and the superior margin is tilted away from the sacral promontory.	When the symphysis pubis is not at least approximately parallel to the sacrum, the anteroposterior diameters of the inlet and the outlet can be changed significantly (Figure 21B-1). However, a contracted inlet is uncommon in developed nations today, so this step may be omitted by some practitioners.
2	Transverse diameters of midplane and outlet	Sidewalls (interior surface of the ischium)	Sweep the examining fingers down the pubic rami to the inferior wall of the ischium, and move them back along the ischium toward the ischial spine.	*Convergent*: Sidewalls converge toward the outline. *Divergent*: Sidewalls converge toward the ischial spines and flare toward the outline. *Parallel*: Most common finding in gynecoid pelvis.	Convergent sidewalls are associated with deep transverse arrest.
3	Transverse diameter of midplane	Ischial spines	Proceed along the sidewalls downward and backward to locate the ischial spine. Gentle lateral pressure may be required to locate the spine. If unable to locate the spine in this manner, move the fingers medially to the sacrum and locate the sacrospinous ligament. Follow it laterally to the ischial spine.	*Blunt*: Not easily palpated, more flattened against the pelvic wall; the most common finding in gynecoid pelvis. *Prominent*: Easily palpable. *Encroaching*: Easily palpable and long, almost sharp, encroaching toward each other.	The transverse diameter of the midplane is the narrowest diameter that the fetus must traverse. A narrowed midplane can result in deep transverse arrest or failure to descend in the second stage of labor. This dystocia is associated with marked molding and caput.
4	Transverse of midplane	Interspinous diameter	Sweep the finger across the pelvic cavity to the ischial spine on the opposite side of the pelvis to estimate the transverse diameter of the midplane.	Estimate diameter in centimeters (normal is 10 cm or greater).	
5	Transverse diameter of midplane and posterior aspect of pelvis	Sacrosciatic ligament	Move the examining finger back to the first ischial spine palpated and follow the sacrosciatic ligament to the sacrum to measure the width of the sacrosciatic notch.	$>$ 2 fb (3 fb = average). $<$ 2 fb suggests forward inclination of sacrum.	

6	AP diameter of outlet	Coccyx shape and mobility	Press firmly on the coccyx to determine its shape and mobility.	Mobility: Describe as a *mobile* or *fixed*. Shape: *J-shaped* or *flat*.	A coccyx that is J-shaped or immobile may break during childbirth. Labor may or may not be impeded, but significant pain in the area of the coccyx can occur postpartum when the coccyx does break.
7	AP diameter of midpelvis	Sacrum shape	Palpate the sacrum from sacrococcygeal junction upward; "walk up the sacrum," estimating the degree of curvature and inclination.	Curve is described as *straight* (flat), *angular* (J-shaped), or *hollow*. Inclination is described as *backward, average,* or *forward*.	A hollow sacrum is indicative of an anthropoid pelvis and persistent occiput posterior position.
8	AP diameter of inlet	DC	Drop both wrists and elbow toward the floor, and extend the fingers posteriorly and cephalad with the inferior margin of the symphysis in contact with the dorsal aspect of the examining hand. Avoid undue pressure on the urethra or clitoris. Most often the sacral promontory cannot be reached.	A diameter of 11.5 cm or greater implies an adequate DC. The obstetric conjugate is approximately 1.5 cm less than the measured DC (Figure 21B-1). Recorded as (1) greater than or equal to the measured distance of the midwife's reach or (2) equal to the measured distance of the midwife's reach.	A narrow diagonal conjugate can result in failure of engagement.
9	Transverse diameter of outlet and forepelvis	Subpubic arch	The midwife's hand is rotated to the side so the fingertips face the side of the pelvis as the hand is removed from the vagina. Just prior to removing the hand, rotate the examining fingers so the palmar surface is up. Evaluate how easily two examining fingers are accommodated under the arch. If two fingers are easily accommodated, the angle is approximately 90°. If the fingers must overlap to fit into the pubic arch, the arch is $< 90°$.	Degree of angle relative to 90°. Record as (1) $> 90°$(gynecoid) or (2) $\leq 90°$ (android).	The fetal head must descend lower to traverse a narrow pubic arch, which increases the chance of perineal lacerations.
10	Transverse diameter of outlet	Intertuberous diameter	Measure the transverse diameter of the outlet (intertuberous diameter; biischial diameter) by placing a premeasured fist between the tuberosities.	Record measurement of the intertuberous diameter as greater than, equal to, or less than the premeasured width of the fist.	

Abbreviations: AP, anterior–posterior; DC, diagonal conjugate; fb, fingerbreadth.

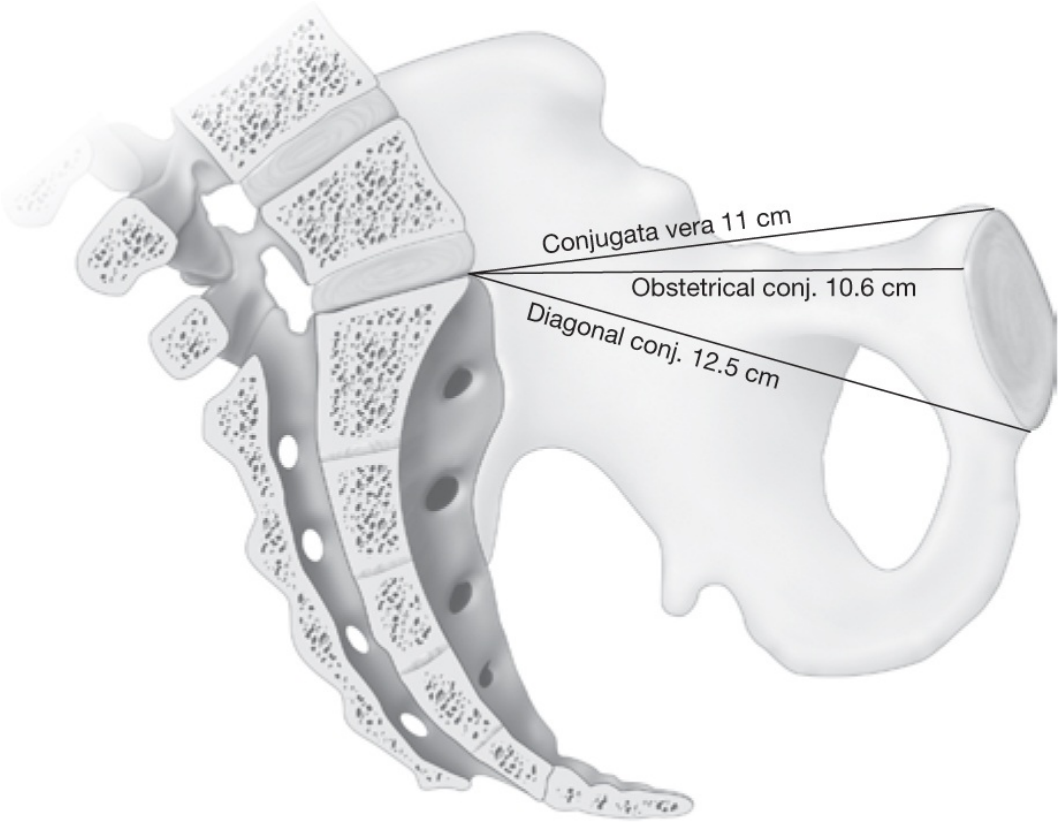


Figure 21B-1 Diameters and planes of the pelvis.

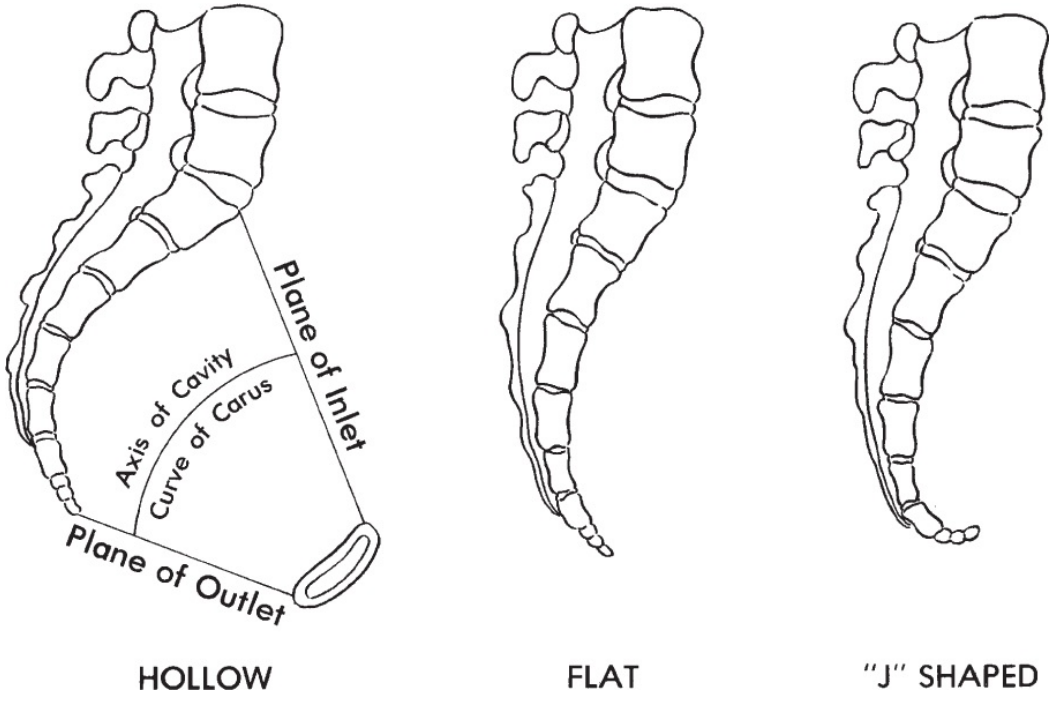


Figure 21B-2 Sacral shapes.

References

1. Yeomans ER. Clinical pelvimetry. *Clin Obstet Gynecol.* 2006;49(1):140-146.

2. Caldwell WE, Moloy HC. Anatomical variations in the female pelvis: their classification and obstetrical significance. *Proc R Soc Med*. 1938;32(1):1-30.
3. Maharaj D. Assessing cephalopelvic disproportion: back to the basics. *Obstet Gynecol Surv*. 2010;65(6): 387-395.
4. Blackadar CS, Viera AJ. A retrospective review of performance and utility of routine clinical pelvimetry. *Fam Med*. 2004;36(7):505-507.
5. Pattison RC, Cuthbert A, Vannevel V. Pelvimetry for fetal cephalic presentations at or near term for deciding on mode of delivery. *Cochrane Database Syst Rev.* 2017;3:CD000161. doi:10.1002/14651858.CD000161.pub2.

CHAPTER

22

Pregnancy-Related Conditions

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Introduction

Every woman deserves the personalized care and skill of a midwife during pregnancy and birth. The majority of women experience healthy pregnancies, and midwifery care provides the appropriate support and interventions needed. Women with pregnancy-related conditions and complications also benefit from midwifery care, although they may need physician care as well. Medical complications existing prior to pregnancy or aggravated by pregnancy are presented in the *Medical Complications in Pregnancy* chapter. This chapter first addresses management of common discomforts and first-trimester loss (e.g., miscarriage, ectopic pregnancy, and tubal pregnancy). The chapter then reviews fetal, placental, and maternal complications that are related to the condition of being pregnant.

Overview of Midwifery Management of Women with Pregnancy-Related Conditions

Assessment of women for conditions related to pregnancy begins with the first prenatal visit and continues throughout the gestation. At each visit, risks can be identified and care instituted to minimize adverse effects. **Table 22-1** lists examples of conditions that may arise during prenatal care and that indicate a need for consultation or referral to a physician. The care that a midwife provides to women who have pregnancy-related conditions or complications during their pregnancy depends on several factors, including the practice setting and institutional guidelines, the midwife's experience and education, the availability of and collaborative relationship with physician consultants, and legal or regulatory statutes.[1]

Table 22-1	Selected Indications for Medical Consultation or Referral During Pregnancy[a]
Gynecologic or Reproductive History	
Large uterine fibroids	Prior unexplained third-trimester fetal loss
History of cervical insufficiency	Uterine anomaly
Previous uterine surgery, including prior cesarean section	
Medical Complications Prior to Pregnancy or That Worsen During Pregnancy	
Anemia not responding to oral iron supplementation	Infectious disease such as hepatitis, HIV, gonorrhea, syphilis, pneumonia, or tuberculosis
Asthma requiring medication	Mental disorder that requires medication
Autoimmune disorders	Morbid obesity
Breast mass	Mitral valve prolapse without previous cardiology evaluation
Cholestasis	Renal calculi
Diabetes	Persistent proteinuria
Hypertension	Seizure disorder
Deep vein thrombosis or pulmonary embolus: history or current	Skin rashes or lesions without diagnosis
Gastrointestinal disease such as Crohn's disease, previous bariatric procedure	Suspected pneumonia
Headaches: severe, recurrent, or associated with hypertension	Syphilis
Hemoglobinopathy	Thyroid disorder
Obstetric Complications That Develop During Pregnancy	
Abnormal screening results for chromosomal conditions such as aneuploidy or genetic disorders	Post-dates pregnancy (> 41 0/7 weeks)
Short cervix via transvaginal ultrasound or clinical examination	Preterm labor
First-trimester bleeding with suspected pregnancy of unknown origin or ectopic	Pyelonephritis
Gestational diabetes	Placenta previa with significant bleeding or persisting after 20 weeks
Isoimmunization	Pregnancy-induced hypertension
	Vaginal bleeding suspected not to be bloody show or related to cervicitis

Hypertension	Varicose veins with pain, swelling, or stasis
Malpresentation of the fetus after 36 weeks	Ultrasound with abnormal findings such as fetal growth restriction, polyhydramnios, fetal anomalies, or unresolved size–dates discrepancy
Multifetal gestation	
Nausea and vomiting that requires hospitalization	

[a] This list is offered for consideration and is not absolute or all inclusive. The list is intended as an adjunct to clinical management based on the individual woman, the midwife, the consulting physician, and institutional guidelines.

In addition, the appropriate level of care for prenatal care and site of birth must be determined. Recently, levels of maternal care have been established for facilities that can help guide decisions about transfer to other facilities (**Table 22-2**).[2] Midwives provide care to women in each of these settings.

Table 22-2 **Levels of Maternal Care Considerations in Pregnancy with Obstetric Complications**

Level of Care	Capabilities	Services	Providers
Birth center	Term, singleton, vertex	Low risk, uncomplicated	Midwives
Level I	Low-risk pregnancy Term gestation Ability to manage unanticipated complications until transfer to a higher level of care	Ability to provide emergency cesarean birth on a 24/7 basis	Midwives and physicians Obstetric provider credentialed for cesarean birth
Level II	Pregnancy with complications that do not require subspecialty care	Medical and surgical consultants available to stabilize women prior to transfer	Midwives Anesthesia available on a 24/7 basis Obstetrician gynecologist available on a 24/7 basis MFM available for consultation on site or by telemedicine 24/7
Level III	Care of more complex maternal medical, obstetric, and fetal conditions	Medical and surgical ICU with ability to collaborate with MFM Subspecialists available for inpatient consultation	Midwives Anesthesia available on a 24/7 basis Obstetrician gynecologist on site on a 24/7 basis MFM available on site or by tele-medicine with inpatient privileges
Level IV (regional perinatal health center)	Care of most complex medical conditions and critically ill pregnant women	On-site obstetric ICU Medical and surgical ICU Medical and surgical subspecialists available on site on a 24/7 basis, including transplant specialists, cardiac surgery, and advanced neurosurgery	Midwives Obstetrician gynecologist on site on a 24/7 basis MFM available on site on a 24/7 basis MFM with expertise in critical care obstetrics Anesthesia with special training in obstetrics

Abbreviations: ICU, intensive care unit; MFM, maternal–fetal medicine specialist.

Based on American College of Obstetricians and Gynecologists. Obstetric Care Consensus No. 2: levels of maternal care. *Obstet Gynecol*. 2015:125:502-515.[2]

Common Discomforts of Pregnancy

The physiologic changes of pregnancy have wide-ranging effects on all body systems. These changes result in many symptoms, which, although not pathologic in nature, can distress or disrupt a woman's life to varying degrees throughout her pregnancy (**Table 22-3**). Common discomforts of pregnancy are presented in this chapter because some—such as dyspnea, nausea and vomiting, or urinary frequency—may be an indication of a disorder that requires treatment. Differentiating normal versus abnormal symptoms is the first task when these symptoms are reviewed during a prenatal care visit. A methodical approach to considering the differential diagnosis is recommended (every time for every woman), despite the tendency to assume the discomfort is a normal physiologic change of pregnancy.

Table 22-3 Common Discomforts of Pregnancy[a]

Description	Differential Diagnosis	Strategies to Decrease Discomfort
Back Pain		
Upper backache develops during the first trimester from increased size of breasts. Low back pain typically occurs in the last half of pregnancy, secondary to the increasing weight of the uterus and relaxation of the sacroiliac ligaments. The exaggerated lordosis of pregnancy strains the back muscles and causes pain.	Sciatica Early labor, pyelonephritis, kidney stone	For upper back pain: A well-fitting and supportive bra. For lower back pain: Proper body mechanics for lifting, pelvic rock/pelvic tilt exercises. External abdominal support (e.g., a maternity girdle or supportive elastic "belly band," or sacroiliac support belt). Heat, ice packs, or massage. A supportive mattress or positioning with pillows to straighten the back and alleviate pulling and strain. Evaluation and treatment by a chiropractor experienced in working with pregnant women may be beneficial.
Breast Tenderness		
Tenderness, tingling, increase in size. Appears in first trimester and discomfort resolves in second trimester but enlarged size remains.	Fibrocystic breast changes, benign breast mass, breast cancer, mastitis	Wear a correctly fitted and supportive bra. Avoiding caffeine or other methylxanthines may help prevent additional breast tenderness, although effectiveness of this practice has not been proven.
Constipation		
Effect of progesterone on gastrointestinal tract, pressure from the enlarging uterus while it is a pelvic organ. May be exacerbated by iron supplements. Occurs in first trimester and resolves by second trimester.	GI disorder	Increase fluids and change in diet. Warm liquids to stimulate peristalsis. Foods that contain roughage, bulk, and natural fiber. Mild bulk-forming laxatives, stool softeners, and glycerin suppositories are safe for use in pregnancy. Stimulant laxatives such as senna are possibly safe but are not recommended for use during pregnancy. These agents directly increase peristalsis and may cause dehydration. Castor oil should be avoided, as it can cause uterine contractions.

Dyspareunia		
Physiologic changes that cause pelvic/vaginal congestion. Most likely to appear in the second half of pregnancy.	Vaginitis, vulvovaginal hemorrhoids, preexisting dyspareunia Screen for abuse or history of abuse if indicated.	Positional changes to accommodate the larger uterus or pain from deep penetration may be all that is needed. Use of water-based vaginal lubricants may be helpful. Treat vaginitis as indicated. Referral to counseling is appropriate for women who have a history of abuse. If current abuse is suspected, offer support and referral, and assess safety.
Dyspnea		
Sensation as needing to take an “extra” breath, not associated with tachypnea. Etiology is not clearly known but may be secondary to altered respiratory center sensitivity (progesterone) and diaphragm displacement. May appear at any time in pregnancy.	Dyspnea with tachycardia: may suggest anxiety or panic attack Respiratory infection Cardiac disorder	Explain the physiologic basis for shortness of breath to relieve anxiety. Deliberately regulate the speed and depth of respirations at normal rates when aware of hyperventilating. Use diaphragmatic or intercostal breathing as opposed to abdominal breathing.
Edema		
Lower-extremity edema (dependent edema) is secondary to impaired venous circulation and increased venous pressure in the lower extremities from the enlarged uterus. Appears in the third trimester.	DVT, phlebitis (would be unilateral) Preeclampsia: rule out non-dependent edema (hands or face) and excessive edema/sudden weight gain	Regular exercise and avoiding prolonged sitting or standing may help. Elevate legs periodically and position on the side when lying down. Keep legs uncrossed when sitting. Graduated compression or support hose can reduce venous pooling in the lower extremities.
Fatigue		
Increased energy requirements, weight gain, and disrupted sleep may be present. Common during the first and third trimesters.	Anemia, depression, cardiac conditions, sleep apnea, thyroid dysfunction, viral or other systemic illnesses	Regular exercise may help improve sleep patterns if fatigue is related to disrupted sleep. Avoid caffeine at night.
Flatulence and Gas Pain		
Decreased GI motility and uterine displacement of intestines. First develops in first trimester and may occur at any time in pregnancy.	Dietary sensitivities, IBS, celiac disease Appendicitis, IBS, or GI disorder	Exercise can aid in improving GI motility. Use of caffeine can stimulate GI motility. Note that caffeine may increase the risk of heartburn, and accentuate insomnia. Caffeine has a prolonged period of metabolism during pregnancy and a standard dose will be present in higher plasma concentrations compared to that dose consumed by a non-pregnant person. The knee–chest position may help with discomfort from unexpelled gas.
Gingivitis		
Pregnancy changes in oral mucosa	Epulis (pregnancy	Increasing the frequency of oral hygiene usually

increase the likelihood of gingivitis. Appears in the second trimester.	tumor or benign overgrowth of gingiva), periodontal disease	resolves hyperemia and bleeding. Refer for dental care if gingivitis persists after increasing oral hygiene.
Heartburn (Pyrosis)		
Regurgitation, or reflux, of acidic gastric contents into the lower esophagus. Progesterone-induced relaxation of LES, slower emptying of gastric contents, smaller-capacity stomach. Appears in the third trimester.	GERD, hiatal hernia, peptic ulcer disease, cholecystitis, pancreatitis If unremitting, rule out preeclampsia	Take small, frequent meals. Avoid foods that make heartburn worse. Elevate the head of the bed, and avoid lying down after eating. A stepwise approach recommended starting with antacids, then moving as necessary to H_2 receptor antagonists and lastly to proton pump inhibitors.
Heart Palpitations or Short Period of Sinus Tachycardia		
A skipped or extra beat, or sinus tachycardia. The etiology may be a pregnancy-induced increase in blood volume and pulse; enlarged heart is more arrhythmogenic. Occurs at any time in pregnancy.	Anxiety Cardiac disorder, arrhythmia Thyroid disorder	Can be aggravated by dehydration, stress, strenuous physical activity, caffeine, tobacco, and alcohol use. No specific relief measures. Pregnancy changes in the cardiac and hematologic systems increase the incidence of short benign arrythmias. Reassurance and education that this is a normal symptom of pregnancy are usually sufficient.
Hemorrhoids		
Progesterone-induced relaxation of the vein walls in the rectum, along with the enlarged uterus, causes pelvic venous congestion. Appears at any time but is most likely in the third trimester.	Thrombosed hemorrhoid If rectal bleeding persists and hemorrhoid is not visible, consider referral for cancer evaluation	Avoid constipation and straining during defecation. Use witch hazel compresses or Epsom salt compresses. Topical anesthetics and topical cortisone creams or suppositories are safe for use in pregnancy.
Insomnia		
Difficulty sleeping may be related to frequent waking from back pain, heartburn, or nocturia.	Sleep disorder, sleep apnea, anxiety, RLS	If no signs of pathology: Take warm baths, reduce stimulation prior to bedtime, avoid caffeine. Regular exercise, and regulation of the temperature in the room may improve sleep. Antihistamines such as diphenhydramine 50–100 mg (Benadryl) or doxylamine succinate 25 mg (Unisom) orally 30 minutes before bedtime may help some women but no pharmaceutical sleep aid should be used regularly.
Leg Cramps		
Sharp sudden cramp in the calf or thigh, which often occurs at night. May be linked to changes in calcium, magnesium, and phosphorus levels or the ability of calcium to enter the muscles, but causes have not been proven. Occurs in the second and third trimesters.	DVT or phlebitis Electrolyte imbalances, muscle or neurovascular disorders Peripheral neuropathy RLS	Straighten the affected leg and dorsiflex the ankle. Magnesium supplementation of 350 mg at bedtime has been shown to be helpful to some women in studies but not all participants in these studies.

Leukorrhea		
Profuse, thin or thick vaginal secretion that typically appears in the second trimester.	STI, vaginitis, ruptured membranes, preterm labor	Avoid douching or using feminine hygiene sprays; clean the perineal and vaginal areas only with water. Reassurance and education that this is a normal symptom of pregnancy are usually sufficient.
Nasal Congestion		
Hyperemia and increased blood flow in nasal passages (estrogen effect). Occurs at any time during pregnancy.	URI, sinusitis, clotting disorder, hypertension May be an indication of drug use	Use a humidifier to generate cool mist at night. Avoid blowing the nose hard. Nasal sprays with epinephrine may provide temporary relief but can be habit forming and result in rebound congestion (especially the long-acting formulations). For epistaxis, elevate the head and compress the nose against the midline septum continuously for 5–10 minutes.
Nausea and Vomiting		
Nausea and/or vomiting and retching may be mild to severe. The exact etiology is unknown. Presents and peaks in the first trimester.	Hyperemesis with dehydration or electrolyte changes GERD or other GI disorder Viral GI infection	Consume small, frequent meals. Avoid fried fatty foods and foods with strong odors. Temporarily discontinue prenatal vitamins that contain iron, but continue folic acid supplementation. Ginger, acupressure bands, and vitamin B_6 may help women with mild to moderate symptoms. Antiemetic medications are recommended in a stepwise fashion depending on the severity of symptoms.
Ptyalism		
Excessive salivation often associated with nausea and vomiting. Etiology unknown.	Hyperemesis	Ptyalism usually resolves spontaneously, although the condition may not disappear until after the pregnancy is over. Some women obtain temporary relief from gum chewing or sucking on hard candies.
Round Ligament Pain		
Round ligaments increase in length as the uterus rises in the abdomen. The pain probably stems from ligament stretching. Often worsens or is elicited with exercise or turning the torso. Appears early in the second trimester and is usually unilateral.	Preterm labor, appendicitis, constipation, gas pain, inguinal hernia, and muscle strain or sprain	Positions that place less tension on the round ligament and wearing a maternity abdominal support or girdle may provide relief for some women.
Sciatica		
Pain in the pelvis and buttock, which may radiate down the back of the lower extremity. Can be sudden and severe. Pressure on the sciatic nerve from joint laxity, often elicited by twisting, lifting, or moving the leg. Typically appears in the third trimester and is usually unilateral.	Cauda equina syndrome: loss of bowel or bladder function Herniated disc: loss of reflex or strength	Rest in a side-lying position on the contralateral side from the affected leg. Heat packs, ice, or an abdominal support girdle may offer some relief. Rest and avoid twisting the torso. Use careful body mechanics when lifting. Acetaminophen: Maximum daily dosage is 4000 mg/day. Referral to a chiropractor or physical therapy may be indicated.

Syncope (Supine Hypotensive Syndrome)		
Dizziness or syncope when suddenly standing or lying on back. The enlarged uterus impairs venous return and causes hypotension. Typically appears in the third trimester.	Hypoglycemia Seizure disorder	If transient and no loss of consciousness, sit down with the head lowered or lie on the side.
Tingling and Numbness of Fingers		
Kyphosis places pressure or traction on nerves in the arm, which can cause tingling and numbness of the fingers. Often occurs at night.	Carpal tunnel syndrome	Move arms at the level of the shoulder to decrease pressure on the nerves when tingling starts. Wrist splints that keep the wrist in a neutral position may be worn while sleeping or during work or other activities.
Urinary Frequency and Nocturia		
Mechanical pressure from the enlarging uterus in the first trimester results in decreased bladder capacity. Frequently occurs in the first and third trimesters.	Urinary tract infection, pylonephritis	Decreasing fluid intake at night may offer some relief but women should not restrict fluids in general.
Varicosities		
Occurs secondary to venous distension from increased venous pressure and vasodilation. Familial tendency may increase risk. Most common in the legs and/or vulva Appears in the second or third trimester.	DVT, peripheral arterial or venous disease	Rest and use of compression stockings.

Abbreviations: DVT, deep vein thrombosis; GERD, gastroesophageal reflux disease; GI, gastrointestinal; IBS, irritable bowel syndrome; LES, lower esophageal sphincter; RLS, restless leg syndrome; STI, sexually transmitted infection; URI, upper respiratory infection.

[a] Educational counseling is primarily based on expert opinion and common practice. Little strong evidence exists in this area.

Most of the common discomforts of pregnancy are transient and resolve spontaneously. Many nonpharmacologic treatments are recommended but very few have been systematically studied to determine evidence for their use. The plethora of suggested remedies that may or may not be helpful for an individual woman is illustrated well in the care of women with nausea and vomiting.

Nausea and Vomiting

Nausea occurs in as many as 85% of pregnant women.[3] The peak prevalence is at 11 weeks' gestation, with the average time of onset between 5 and 6 weeks. For most women, nausea and vomiting in pregnancy (NVP) will resolve by 14 to 16 weeks' gestation, although a small percentage of women will have NVP that persists beyond 20 weeks' gestation.[4] Hyperemesis —a severe form of NVP—occurs in approximately 3% of women during pregnancy.[3] Women with a multifetal gestation and those with a hydatidiform mole may have more severe NVP. Nausea, with or without vomiting, is erroneously termed "morning sickness" because it is not

limited to the morning hours, but rather can occur at any time of day. Nausea is more severe when the stomach is empty, which may explain why some women notice it more in the morning. Women with severe NVP or hyperemesis often require hospitalization to break the cycle of vomiting and establish adequate rehydration.

The cause of NVP is not fully known. Likely causes include the interplay between hormonal changes of pregnancy (e.g., human chorionic gonadotropin, estrogen, progesterone, placental prostaglandin E_2), slowed peristalsis, and genetic factors. Other factors such as preexisting gastroesophageal reflux disease (GERD) or *Helicobacter pylori* infection can contribute to the occurrence of NVP in many women. There is little support for an older theory that NVP reflects the transformation of psychological distress into physical symptoms. Nevertheless, a woman's experience of NVP does have biological, psychological, and sociocultural components. Severe NVP can be associated with severe psychological distress, as one would expect. Thus, depression can be the result of NVP but is not the cause of the disorder.[5,6] NVP can be mild or it may be severe enough to require hospitalization. Additional differential diagnoses include thyroid dysfunction, gastrointestinal disorders such as GERD, peptic ulcer disease, cholecystitis, and eating disorders such as bulimia.

Hyperemesis Gravidarum

There is no standard definition for hyperemesis gravidarum, the most severe form of NVP. However, most diagnostic criteria include:

1. persistent vomiting before 9 weeks' gestation
2. dehydration and/or ketonuria
3. weight loss greater than 5% of initial body weight
4. electrolyte imbalance.[7]

Risk factors for hyperemesis include previous pregnancy complicated by hyperemesis, molar pregnancy, multifetal gestation, prepregnancy history of gastrointestinal disorders, and hyperthyroid disorders. Interestingly, women older than 30 years and women who smoke have a lower risk of hyperemesis, and women who have hyperemesis are more likely to carry a female fetus.[6] In the United States, hyperemesis is the second most common reason for hospitalization during pregnancy (following preterm labor).

Women with hyperemesis may have a coexisting, transient hyperthyroid state. This occurs because human chorionic gonadotropin (hCG) is structurally similar to thyroid-stimulating hormone and is able to increase production of free thyroxine (T_4). This hyperthyroid state does not need to be treated and resolves spontaneously by 18 to 20 weeks' gestation.

Assessment and Management of Nausea and Vomiting

Midwives and physicians frequently collaborate in the care of women with NVP, with midwives initiating outpatient management or conducting initial assessment and treatment following hospital admission. Physician consultation or referral is recommended for women with severe symptoms.

The initial goals in evaluating a woman with NVP (**Table 22-4**) are first to rule out differential diagnoses and then to establish the severity of the disorder, which is frequently categorized as mild, moderate, or severe. The differentiation between severe NVP and hyperemesis is a definitional subtlety that is not clinically important. In practice, severe NVP and hyperemesis are likely the same and both are treated with the same interventions.

Table 22-4 Evaluation of Nausea and Vomiting in Pregnancy

History
Abdominal pain (GI disorder)
Blood in vomitus (peptic ulcer or esophagitis from repeated vomiting)
Frequency of nausea, vomiting, and retching episodes (PUQE score to determine severity)
Dietary history (determine severity of NVP)
Elimination (frequency, amount, constipation, diarrhea)
Exposures to viral infection or contaminate food
History of eating disorders
History of chronic disease associated with nausea/vomiting
Medications (e.g., medications that cause nausea such as iron supplements)
Physical Examination
Weight (compare to previous weights to determine severity of NVP)
Vital signs with blood pressure (signs of infection)
Skin turgor, moistness of mucous membranes (signs of dehydration to determine severity of NVP)
Abdominal palpation for organomegaly, tenderness, distension (GI disorders)
Bowel sounds (GI disorders)
Assessment of uterine size (rule out multiple pregnancy or hydatidiform mole)
Laboratory Tests
Complete blood count (signs of dehydration or infection)
Urinalysis and urine dipstick for specific gravity and ketones (dehydration indicates increased severity of NVP)
BUN and electrolytes (electrolyte imbalance indicates increased severity of NVP)
Liver function tests (rule out hepatitis, pancreatitis, and cholestasis)
TSH and T_4 (rule out thyroid disease)
Ultrasound
Confirm pregnancy and rule out multiple pregnancy and/or hydatidiform mole

Abbreviations: BUN, blood urea nitrogen; GI, gastrointestinal; NVP, nausea and vomiting of pregnancy; PUQE, Pregnancy-Unique Quantification of Emesis/Nausea; T4, thyroxine; TSH, thyroid-stimulating hormone.

Several tools to measure the severity of NVP have been validated for use in clinical practice.[3,4,8] A critical component of this evaluation is assessment of how the symptoms interfere with the woman's ability to maintain and engage in daily activities. The PUQE index is based on only three questions, is highly correlated with scores on the more detailed Rhodes index, and is easy to use in practice (**Table 22-5**).[8]

Table 22-5 Modified PUQE Index

1. On an average day, for how long do you feel nauseated or sick to your stomach?				
> 6 hours (5 points)	4–6 hours (4 points)	2–3 hours (3 points)	≤ 1 hour (2 points)	Not at all (1 point)
2. On an average day, how many times do you vomit or throw up?				
7 or more (5 points)	5–6 (4 points)	3–4 (3 points)	1–2 (2 points)	None (1 point)
3. On an average day, how many times do you have retching or dry heaves without bringing anything up?				
7 or more (5 points)	5–6 (4 points)	3–4 (3 points)	1–2 (2 points)	None (1 point)

Abbreviations: NVP, nausea and vomiting of pregnancy; PUQE, Pregnancy-Unique Quantification of Emesis/Nausea.

Total score is the sum of the points for each of the three questions.

Nausea score: mild NVP = ≤ 6; moderate NVP = 7–12, severe NVP = ≥ 13.

Reproduced with permission from Lacasse A, Rey E, Ferreira AE, Morin C, Berard A. Validity of a modified Pregnancy-Unique Quantification of Emesis and Nausea (PUQE) scoring index to assess severity of nausea and vomiting of pregnancy. *Am J Obstet Gynecol.* 2008;198(1):71.31-71.37.[8]

Treatment of Nausea and Vomiting

Treatments for mild, moderate, and severe NVP are presented in **Table 22-6**. First-line treatments for mild NVP for which there is some evidence of effectiveness include ginger products, vitamin B_6, acupressure bands, and dietary changes. Extensive research has established the safe use in pregnancy of Pyridoxine (vitamin B_6) and the antihistamine doxylamine. A delayed-release version of a drug that combines doxylamine and pyridoxine (Diclegis) has been tested extensively and found to be safe and effective; it is now available in the United States for women who are not able to get effective relief from nonpharmacologic therapies.[9]

Table 22-6 Selected Treatments for Nausea and Vomiting in Pregnancy

Treatment	Drug Dosage	Clinical Considerations
Mild NVP		
Temporarily discontinue prenatal vitamins with iron but continue folic acid supplementation		Iron can increase gastrointestinal distress
Acupressure bands		
Ginger	250 mg every 6 hours	May cause acid reflux; several preparations available
Pyridoxine (vitamin B_6)	10–25 mg every 8 hours	
Moderate NVP Without Dehydration		

Doxylamine/pyridoxine (Diclegis)	Combination slow-release product containing 10 mg of pyridoxine hydrochloride and 10 mg doxylamine succinate 2 tablets at night Maximum dose: 4 tablets/day	May cause drowsiness; anticholinergic
Moderate NVP Not Resolved with Doxylamine/Pyridoxine **May add one of the following:**		
Diphenhydramine (Benadryl)	50–100 mg every 4–6 hours PO or PR if vomiting frequently	May cause drowsiness; can offset the anxiety caused by metoclopramide
Metoclopramide (Reglan)	5–10 mg every 6–8 hours PO	FDA black box warning about agitation, anxiety, and acute dystonic reactions
Ondansetron (Zofran)	4–8 mg every 6–8 hours	Headache; concern for potential cardiac arrhythmia—FDA black box warning about QT prolongation for IV single doses greater than16 mg
Promethazine (Phenergan)	12.5–25 mg every 4–6 hours PO/IM/IV/PR	Sedation, anticholinergic effects, dry mouth, dystonic reaction; hypotension if given IV too quickly
Prochlorperazine (Compazine)	5–10 mg every 6–8 hours PO/IM/IV/PR	Sedation, anticholinergic effects, dry mouth, dystonic reaction; hypotension if given IV too quickly.
Moderate NVP with Dehydration		
Non-dextrose IV fluid replacement with multivitamin supplement Physician consultation May use one of the above listed medications once the woman is hydrated and stable		Multivitamin supplement is needed with IV fluids to prevent Wernicke's encephalopathy which can develop secondary to thiamine deficiency

Abbreviations: FDA, U.S. Food and Drug Administration; IM, intramuscular; IV, intravenous; NVP, nausea and vomiting of pregnancy; PO, by mouth; PR, by rectum.

Women with moderate NVP may need medication. If they are dehydrated, intravenous dextrose-containing fluids should be avoided for initial rehydration due to the slight risk of precipitating Wernicke's encephalopathy in women with persistent vomiting. Wernicke's encephalopathy is a neuropsychiatric syndrome resulting from thiamine deficiency; it presents with a classic triad of ocular abnormalities, ataxia, and confusion. Persistent vomiting prevents adequate absorption of thiamine, and administration of carbohydrate-containing IV fluids causes rapid depletion of the limited thiamine left in the body.[10] Without thiamine, metabolic activity in the brain is reduced or switches to lactic acid pathways, resulting in neuronal injury and metabolic acidosis. The standard intravenous solution used for fluid replacement in women with NVP is normal saline, which helps prevent hyponatremia. Potassium chloride can be added as needed, and thiamine (vitamin B_1) or a multivitamin solution that includes thiamine should be added at least once a day to prevent Wernicke's encephalopathy.

For women with severe symptoms, physician consultation and hospitalization are

recommended. Intravenous or intramuscular administration of antiemetics such as metoclopramide (Reglan) or ondansetron (Zofran) is most commonly used to break the initial cycle of vomiting. Of these medications, ondansetron is particularly effective at controlling vomiting. In some studies, this agent has been associated with a slightly increased risk of fetal cardiac septum defects. For this reason, and because treatment of hyperemesis is an off-label use for ondansetron, many providers prescribe ondansetron only after other medications have been tried.[11]

As marijuana gains legal status in many states, an increasing number of women are using marijuana to manage NVP. While women's reports of improved symptoms must be acknowledged, the safety of marijuana exposure for the fetus has not been determined. While it is difficult to isolate the health impacts of marijuana due to confounding factors such as polysubstance abuse, psychiatric comorbidity, and socioeconomic status, research has associated marijuana use with risks of preterm birth, low birth weight, exaggerated startle response, high-pitched cry, and sleep cycle changes in the newborn.[12]

Midwives should also be aware of a counterintuitive response to marijuana that can be mistaken for hyperemesis gravidarum, called cannabinoid hyperemesis. Cannabinoid hyperemesis occurs following heavy marijuana use and is a response to saturated cannabinoid type 1 receptors in the gastrointestinal system.[13] With this condition, women present with symptoms of nausea and vomiting unresponsive to antiemetic treatment. Notably, many women with cannabinoid hyperemesis report that these symptoms can be relieved by hot baths or showers. If cannabinoid hyperemesis is suspected, treatment requires rehydration therapy during 24 to 48 hours of abstinence from marijuana. Symptoms will return if heavy marijuana use resumes.

Most of the common discomforts of pregnancy are not associated with adverse outcomes. However, some can be initial symptoms of a complication that requires evaluation and treatment. The rest of this chapter reviews pregnancy-related complications—those complications that are the result of the condition of pregnancy itself.

Early Pregnancy Loss

Vaginal bleeding occurs in 20% to 40% of pregnant women during the first trimester of pregnancy.[14] Vaginal bleeding may be fresh (bright red) or old (dark brown), be light, and either occur infrequently or persist for several days. The pattern of bleeding and amount of bleeding do not predict miscarriage. Most women who have an episode of minor vaginal bleeding and documented fetal cardiac motion on ultrasound will remain pregnant. The list of differential diagnoses is long, however, some of those diagnoses are associated with significant morbidity, and it can take several days or a few weeks to determine a conclusive diagnosis. Thus, all women who report any vaginal bleeding during pregnancy must be evaluated promptly and may require physician consultation or referral early in the evaluation process.

Differential Diagnoses of First-Trimester Bleeding

Vaginal bleeding in pregnancy can originate in the uterus, cervix, or vagina. Some causes of vaginal bleeding, such as miscarriage, are related to pregnancy; others, such as cervicitis, are not related to pregnancy directly. The most common etiologies of bleeding in the first trimester are miscarriage, subchorionic hemorrhage, anembryonic pregnancy, and vaginitis or lesions of the cervix. The terms *miscarriage*, *spontaneous abortion*, and *early pregnancy loss* are often used interchangeably as there is no consensus on terminology at this time. The terms miscarriage and pregnancy loss are used in this text as these terms more clearly differentiate spontaneous abortion from induced abortion and may be more acceptable to women. **Table 22-7** describes conditions related to pregnancy and early pregnancy loss that cause vaginal bleeding in the first trimester.

Table 22-7 Definitions of Conditions Associated with Early Pregnancy Loss

Term	Definition	Clinical Presentation
Ectopic pregnancy	Pregnancy that is implanted outside of the uterus Most common is tubal pregnancy, in which the fertilized egg becomes implanted in the fallopian tube Ovarian and abdominal pregnancy rare	May be asymptomatic initially and associated with irregular vaginal bleeding or no bleeding. Pregnancy test (urine or serum beta-hCG) may or may not be positive. Adnexal mass may be visible on TVUS; the mass may or may not be palpable on physical examination. The uterus may be slightly enlarged due to hormonal stimulation of the endometrium. If an ectopic pregnancy ruptures, the woman may experience hypotension, signs of shock, sharp severe pain that may be unilateral, CMT, bulging posterior vaginal fornix, and referred pain to the neck/shoulder if there is bleeding into the peritoneum.
Hydatidiform mole	Complete: Haploid sperm fertilizes an ovum that has lost DNA; the sperm DNA replicates and the resulting hydatidiform	The uterus may be larger than expected. Significant NVP and uterine bleeding are present. FHR is not detected.

	mole has a genotype of 46XX or 46 XY Partial: Two haploid sperm fertilize a normal ovum; the resulting pregnancy is triploid (69XXY) or greater	May be associated with hypertension and early preeclampsia Ultrasound has diagnostic features.
Pregnancy of uncertain viability	Ultrasound shows a gestational sac but no embryonic heartbeat	This diagnosis is based on findings on TVUS that do not definitively detect a viable fetus or a nonviable pregnancy. Examples: A gestational sac that is visible on TVUS days before a yolk sac or embryo appears Embryo with a crown rump length greater than 7 mm but no FHR
PUL	Positive pregnancy test but pregnancy cannot be detected on TVUS	Neither ectopic pregnancy nor IUP is confirmed. Women with PUL are monitored carefully until an IUP is visualized, miscarriage occurs, or ectopic pregnancy is diagnosed
Subchorionic hemorrhage	Bleeding between the chorion and the myometrium, or between the chorion and the placenta	Irregular, slight vaginal bleeding occurs. Subchorionic hemorrhage may result in miscarriage if large enough to impede placental growth. Most often the hemorrhages are small and resolve spontaneously as the placenta grows. Visible on ultrasound.
Miscarriage		
Biochemical pregnancy or anembryonic gestation (old term: blighted ovum)	Development of a gestational sac without development of an embryo	Gestational sac > 18 mm without an embryo. Declining beta-hCG levels after beta-hCG level < 1000 mIU/mL is noted with a gestational sac visible on TVUS.
Missed miscarriage[a] (also known as missed abortion)	Nonviable products of conception are retained with or without vaginal bleeding	Irregularly shaped/collapsing gestational sac; no embryo, mean sac diameter of ≥ 25 mm. Crown-rump length ≥ 7 mm and lack of cardiac motion. Absence of embryo with heart beat ≥ 2 weeks after an ultrasound showed a gestational sac without yolk sac or ≥ 11 days after ultrasound showed a gestational sac with a yolk sac.
Recurrent miscarriage	Spontaneous abortion that has terminated the course of ≥ 3 consecutive pregnancies	No diagnostic ultrasound findings.
Threatened miscarriage	Painless vaginal bleeding at < 20 weeks' gestation without cervical dilation or effacement	May or may not show sonographic signs of abnormal sac or embryo. May show subchorionic bleeding.
Inevitable miscarriage	< 20 weeks' gestation with cervical dilation and/or rupture of the membranes in addition to vaginal bleeding and lower abdominal or back pain but no passage of tissue	Embryo > 5 mm in size, without cardiac activity. Embryonic bradycardia after 8 weeks' gestation. Serum progesterone < 5 ng/mL.
Incomplete miscarriage	Passage of some fetal or placental tissue through the cervix at < 20 weeks' gestation	Tissue visible in the uterus without evidence of viable gestation.
Complete	Spontaneous expulsion of fetal and	Uterus empty on sonogram.

miscarriage	placental tissue from the uterine cavity at < 20 weeks' gestation	
Septic miscarriage	Serious maternal infection that occurs after any abortion	May or may not show retained products of conception on sonogram.

Abbreviations: Beta-hCG, beta-human chorionic gonadotropin; CMT, cervical motion tenderness; FHR, fetal heart rate; IUP, intrauterine pregnancy; NVP, nausea and vomiting of pregnancy; PUL, pregnancy of unknown location; TVUS, transvaginal ultrasound.

[a] Findings that are suggestive but not diagnostic for early pregnancy loss include crown-rump length < 7 mm without a heart beat; Mean sac diameter of 16–24 mm without embryo; Absence of embryo with heart beat 7–13 days after ultrasound that showed a gestational sac without a yolk sac or 7–10 days after an ultrasound that showed a gestational sac with yolk sac; empty amnion; enlarged yolk sac > 7 mm; small gestational sac in relation to size of embryo; absence of embryo for ≥ 6 weeks after last menstrual period.

Evaluation of First-Trimester Bleeding

The initial goal in evaluating a woman with first-trimester bleeding is to rule out life-threatening conditions such as active hemorrhage or ectopic pregnancy. **Table 22-8** reviews the history and physical components of evaluation for a woman with first-trimester bleeding. A midwife is frequently involved in the initial assessment of women who have bleeding early in pregnancy. However, ectopic pregnancy requires physician care, and depending on ultrasound and laboratory findings, close monitoring and repeat laboratory tests may be needed for several days before the diagnosis is clearly established. **Figure 22-1** presents one algorithm for evaluation and management of first-trimester bleeding that will help the midwife assess for the initial differential diagnoses which include: non-uterine source of bleeding (e.g., cervical polyp, cervicitis), viable pregnancy, nonviable pregnancy, uncertain pregnancy, pregnancy of uncertain location, or ectopic pregnancy.[15] Knowledge of all possible diagnoses and likely progression of assessment and care allows the midwife to obtain physician consultation and referral at the appropriate time. Once a plan of management is established, the midwife can support the woman with anticipatory guidance as needed.

Table 22-8 Evaluation of First-Trimester Bleeding

Data	Description and Differential Diagnosis
History	
Diagnosis pregnancy	Obtain LMP, LNMP, result of pregnancy test, and ultrasound results. If IUP is identified, ectopic pregnancy is unlikely.[a]
Reproductive history	Risk of miscarriage is increased in women who have previous miscarriages. Risk of ectopic pregnancy is increased in women with previous ectopic pregnancies.
Bleeding pattern and associated events	Profuse bleeding with clots suggests incomplete miscarriage or ectopic pregnancy. Intermittent spotting associated with intercourse or vaginal penetration indicates a cervical or vaginal source of bleeding. Chlamydia or gonorrhea can cause cervicitis that manifests as vaginal spotting. Intermittent bleeding can also suggest miscarriage.
Passage of any tissue	Passage of tissue suggests miscarriage.

Pain	Abdominal pain and referred pain to the neck or shoulder suggests intra-abdominal bleeding from a ruptured ectopic pregnancy, or possibly an ovarian cyst (a nonpregnancy cause).
Physical Examination	
Vital signs and blood pressure	Determine if the woman is hemodynamically stable.
Abdominal examination	Palpate for tenderness/pain, fundal height or other masses, and rebound tenderness (appendicitis).
	Perform Doppler fetal monitoring for FHR if gestational age is 10 weeks or greater (rule out ectopic pregnancy, hydatidiform mole, and missed abortion).
	Palpate for CVAT (pyelonephritis can present with referred pelvic pain).
Speculum examination	Inspect for lacerations, lesions, vaginitis, cervicitis, hemorrhoids, and varicosities.
	Perform a wet prep if indicated.
	Inspect the cervical os for polyps, dilation, presence of fluid, blood, clots, pus, or fetal parts or membranes.
Bimanual examination	Size of uterus.
	Cervical effacement, dilation, CMT.
	Adnexal masses or pain.
Laboratory Tests and Ultrasound	
	Obtain hemoglobin/hematocrit if indicated.
	Perform ultrasound if indicated.
	Serial serum quantitative beta-hCG or progesterone measurement may be indicated.

Abbreviations: beta-hCG, beta human chorionic gonadotropin; CMT, cervical motion tenderness; CVAT, costovertebral angle tenderness; FHR, fetal heart rate; IUP, intrauterine pregnancy; LMP, last menstrual period; LNMP, last normal menstrual period.

[a] Rarely a woman will have both an intrauterine and extrauterine pregnancy—a condition called a heterotropic pregnancy. In this situation, visualization of an intrauterine pregnancy does not rule out an ectopic pregnancy.

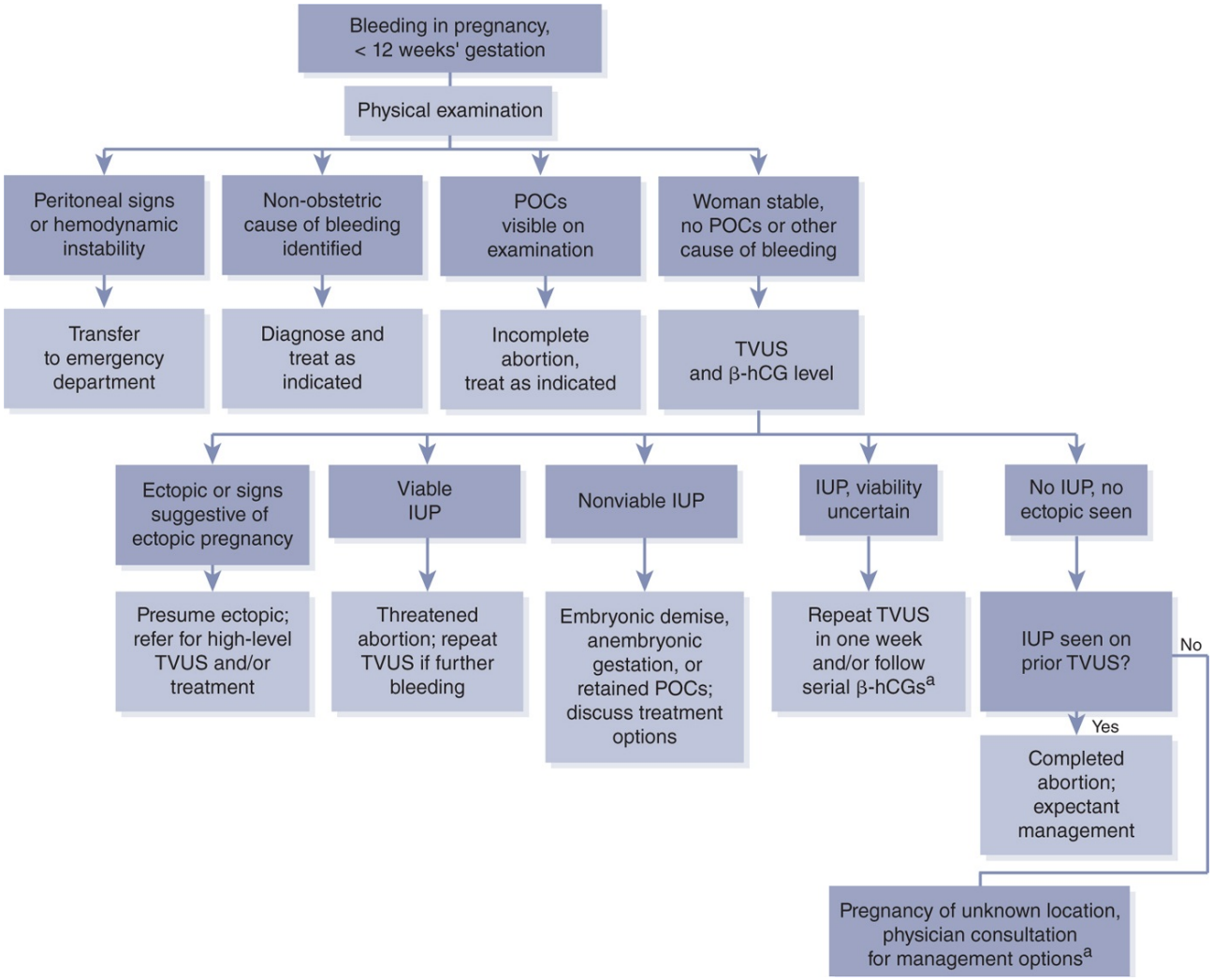


Figure 22-1 First trimester bleeding algorithm.

Abbreviations: **β**-hCG, human chorionic gonadotropin; IUP, intrauterine pregnancy; POCs, products of conception; TVUS, transvaginal ultrasound.

[a] Care of women with a pregnancy of unknown location requires physician consultation for close supervision and monitoring of beta-hcG levels. Ectopic pregnancy cannot be ruled out. The woman's desire for continued pregnancy in combination with assessment of the rise or fall in beta-hcG levels over several days will guide management.

Modified with permission from Reproductive Health Access Project. First trimester bleeding algorithm [Internet]. Available at: https://www.reproductiveaccess.org/resource/first-trimester-bleeding-algorithm/. Accessed December 6, 2017.[15]

Physical Examination

The physical examination can sometimes determine the origin of the bleeding. Cervical polyps, cervicitis, hemorrhagic cystitis, perineal lesions, and vulvar varicosities are sources of bleeding that may be perceived as coming from the vagina. Thus, the initial evaluation is to rule out non-uterine sources of bleeding.

Laboratory Tests

The normal values for beta human chorionic gonadotropin (beta-hCG) in early pregnancy are described in the *Anatomy and Physiology of Pregnancy* and *Prenatal Care* chapters. Serial quantitative measurements of beta-hCG are frequently used to help establish the presence and viability of a pregnancy during the early period before a pregnancy is visible on ultrasound. If the woman is hemodynamically stable, serum beta-hCG levels can be obtained every 2 to 3 days. These values should double approximately every 1.5 to 3 days in the first 5 to 6 weeks of gestation and every 3 to 3.5 days before plateauing at approximately 8–10 weeks' gestation.[14-18] Once the beta-hCG blood level reaches 1500 mIU/mL, a gestational sac should be visible during a transvaginal ultrasound examination. Sometimes, however, transabdominal ultrasound may not be able to visualize a gestational sac until after the beta-hCG reaches 6000 mIU/mL. Expected ultrasound findings have been linked to beta-hCG levels, although each institution will have defined cut-off values for correlating beta-hCG values with ultrasound findings, as there is significant variability in how beta-hCG is measured.[14-18]

Progesterone levels can also be used to discriminate between failing intrauterine pregnancy and viable intrauterine pregnancy. The primary limitation of this biomarker is that it does not help in determining the site of pregnancy. For this reason, assessment of a progesterone level is not a routine component of most clinical management protocols, but it may be of value in individual cases. Progesterone is produced by the corpus luteum in the first trimester of pregnancy. If the serum progesterone value is 20 ng/mL or more, the pregnancy is likely viable. Similarly, a value of 5 ng/mL or less is consistent with an abnormal or failing pregnancy. When the serum progesterone level is between 5 and 20 ng/mL, further evaluation of the pregnancy is indicated.[19] Another potential problem with the use of progesterone as a biomarker for pregnancy is that some laboratories take several days to return results, which limits the clinical utility of this test as a diagnostic tool.

Ultrasound in the first trimester can be diagnostic for most of the uterine causes of first-trimester bleeding. It can support assessment of intrauterine versus extrauterine pregnancy, gestational age (accurate within 5–7 days), viability, number of fetuses, and presence of adnexal masses, uterine fibroids, or fluid in the cul-de-sac. Ultrasound can also identify subchorionic hemorrhage, incomplete miscarriage with retained products of conception, and complete miscarriage. Such an examination can be diagnostic for hydatidiform mole via the appearance of a cluster of grape-like material, a honeycomb effect, or a "snowstorm" on the screen. However, point-of-care ultrasounds commonly performed in an outpatient setting do not have the resolution needed to make many of these diagnoses.[14] If findings of a point-of-care ultrasound performed by a midwife or other clinician are inconclusive, the woman should receive a Level 1 ultrasound under the guidance of a trained radiology technician.

Ectopic Pregnancy

An old adage states, "You have only one chance to miss an ectopic." A woman with subjective symptoms or physical findings consistent with an ectopic pregnancy, or a woman with a positive pregnancy test and no intrauterine pregnancy on ultrasound (pregnancy of unknown location), needs immediate medical assessment.

Signs and symptoms of a ruptured ectopic pregnancy vary widely. The classic presentation

of tubal rupture is a woman who may or may not realize she is pregnant who is having no bleeding or slight irregular spotting. Sudden sharp, stabbing, severe, lower abdominal pain is followed by hypotension and signs of shock. Physical examination findings include tender abdomen, painful vaginal examination, cervical motion tenderness, and a possible adnexal mass. The cul-de-sac may be full of blood, causing the posterior vaginal fornix to bulge outward. Pain in the neck or shoulder, especially on inspiration, may be present as a result of diaphragmatic irritation from blood in the peritoneal cavity. A woman with this profile is experiencing a medical emergency and requires immediate medical assistance.

Once the diagnosis of ectopic has been made, and physician care has been established, management can be surgical or medical if the woman is hemodynamically stable. Surgical removal of the pregnancy and fallopian tube has historically been the standard treatment. Currently, for women who meet specific criteria, oral administration of methotrexate (Trexall) can resolve the ectopic pregnancy, thereby avoiding a surgical procedure.[18,20] After methotrexate administration, the body slowly resorbs all products of conception and serial beta-hCG levels are monitored carefully to make certain the medication was effective in stopping the pregnancy.

Ovarian and abdominal pregnancies are rare. Abdominal pregnancy is usually the result of an early ectopic pregnancy that ruptured into the peritoneal cavity early enough that the fertilized ovum remains viable. In this scenario, the fertilized ovum implants in the abdomen. Signs and symptoms include an inability to outline the uterus and the sensation that fetal parts are just “under the skin” of the woman. The woman may have severe gastrointestinal symptoms that do not resolve with standard management. Ultrasound examination does not always suffice for making the diagnosis, especially later in pregnancy. Abdominal pregnancy is a life-threatening condition due to adverse effects of placental implantation on abdominal organs such as the liver. Birth must be by cesarean, and requires a tertiary setting wherein multiple subspecialists in maternal–fetal medicine and surgery are involved.

Pregnancy of Unknown Location

Approximately 8% to 31% of women will have a positive pregnancy test, yet an intrauterine pregnancy cannot be seen during transvaginal ultrasound. In this situation, neither intrauterine nor ectopic pregnancy can be determined.[14] This situation can resolve in one of four ways: (1) visualized ectopic pregnancy; (2) visualized intrauterine pregnancy; (3) spontaneous resolution to a nonpregnant state (no embryo is found and all biomarkers of pregnancy resolve); or (4) persistent pregnancy of unknown location. The most common outcome is spontaneous resolution. Although the site of pregnancy was never determined, it is assumed that most of these women experienced very early miscarriages.

Women with a pregnancy of unknown location may be managed expectantly or offered either an exploratory dilatation and curettage (D & C) or methotrexate.[14] Thus, an essential first determination must be made regarding the woman’s desire for ongoing pregnancy.[14] However, the recommendation of expectant management versus interventions that cause abortion is based on multiple individual clinical factors as well as the woman’s preference, and physician consultation/referral is needed. Once a plan of management is made, midwives may participate

in monitoring and supporting the woman through the process.

Miscarriage (Spontaneous Abortion)

Miscarriage is the spontaneous termination of pregnancy by expulsion of the products of conception prior to 20 weeks' gestation.[21] Approximately 10% to 15% of clinically recognized pregnancies end in miscarriage, and 50% of miscarriages are attributed to chromosomal abnormalities.[22] If a woman is hemodynamically stable when she is diagnosed with miscarriage, less than 14 weeks' gestation, with no evidence of an incomplete or septic miscarriage, she can choose expectant management, medical management, or surgical management via D & C.[23,24] A woman's choice of management is made after a review of the risks and benefits of each option within a shared decision-making process. All midwives in clinical maternity care will, at some point, need to help a woman through this process.

Regardless of the choice of management, follow-up includes administration of Rho(D) immune globulin (RhoGAM) for women who are Rh (D) negative, with this medication being given within 72 hours of the completed miscarriage. A post-miscarriage visit includes a review of the events, targeted history and physical examination, urine pregnancy test, and discussion of contraception or future pregnancy planning. If the woman appears to have significant grief or emotional difficulty, it may be appropriate to continue seeing her or to refer her for counseling.

Expectant, medical, and surgical management are equivalent with regard to the risk of infection and overall effectiveness.[23,24] Compared to surgical management, the expectant and medical management options are associated with an increased incidence of incomplete miscarriage that requires subsequent surgical intervention, as well as a higher risk of hemorrhage.[23] Approximately 25% to 30% of women with an incomplete, inevitable, missed, or anembryonic miscarriage will spontaneously expel the products of conception within 1 week. Approximately 52% to 60% will expel the products of conception by 2 weeks.[25] After 2 weeks, the additional number of women who spontaneously pass products of conception increases slowly, with a total of 85% to 90% of miscarriages being completed by 6–8 weeks. Based on these data, it is common practice to wait 2 weeks before offering medical or surgical intervention to women who chose expectant management. Some women may want to continue expectant management beyond 2 weeks, and there are no clear health risks to doing so, as long as the woman understands the warning signs and is able to seek treatment if needed.

If the woman chooses expectant management, she is counseled to call her healthcare provider if she has signs of infection (fever, chills, foul odorous vaginal discharge) or heavy bleeding (passing clots frequently and/or soaking a sanitary pad in less than an hour). If she has experienced two or more miscarriages, she may want to collect the products of conception in a container for genetic studies once the miscarriage is completed. The products of conception can also be sent to a laboratory to identify passage of placental villi, thereby confirming complete expulsion of an intrauterine pregnancy. A follow-up urine pregnancy test performed a week or more after the miscarriage can confirm that the miscarriage is complete. Ultrasound is not necessary if the history and/or physical examination findings are normal but may be employed to confirm the uterus is empty if needed.

Medical management of miscarriage involves vaginal administration of 800 mcg of misoprostol (Cytotec). The dose may be repeated up to 7 days later if there is no response to the first dose. Pain medication may be prescribed concomitantly. The success of misoprostol is 80% to 90% within 7 days in women who are up to 10 weeks' gestational age at the time of administration.[24] The success of misoprostol treatment varies somewhat by diagnosis, with slightly lower success rates being seen in women with anembryonic pregnancies and the highest success rates observed in women who have an incomplete or inevitable miscarriage. When misoprostol is the chosen management for miscarriage, the midwife counsels the woman about expected symptoms and warning signs. It is also essential to determine that the woman has access to emergency care should she need it.

Surgical management of miscarriage may be advised for women who prefer this option, after failed expectant management, in women who are more than 10 weeks' gestation, or if a woman has risk factors contraindicating medical management. Risks of surgical management include cervical trauma, uterine perforation, and intrauterine adhesions. These risks are reviewed by the provider prior to the procedure.

Pregnancy of Uncertain Viability

Management of a woman having a miscarriage depends on a definitive diagnosis of miscarriage. A woman who has expelled products of conception, has resolution of symptoms, an empty uterus on transvaginal ultrasound, and falling beta-hCG levels has had a completed miscarriage. Nevertheless, passage of tissue is not always noted, symptoms are not diagnostic, and beta-hCG values or transvaginal ultrasound findings can be inconclusive. Criteria that are diagnostic for pregnancy failure include the following:[24,26]

- Crown rump length of 7 mm or greater without a fetal heart rate
- Mean sac diameter of 25 mm or greater without an embryo
- Absence of an embryo with a heartbeat 2 weeks or more after a scan that showed a gestational sac without a yolk sac
- Absence of an embryo with a heartbeat 11 days or more after a scan that showed a gestational sac with a yolk sac
- Other findings on transvaginal ultrasound may suggest the diagnosis of pregnancy loss but are not conclusive.[22]

Recurrent Miscarriage

When a woman has experienced three or more miscarriages, genetic counseling and an endocrine evaluation should be considered. The standard evaluation for recurrent miscarriage includes ultrasound to rule out congenital abnormalities of the genital tract (e.g., uterine septum, bicornuate uterus), genetic testing, and tests for coagulation disorders, autoimmune disorders, and thyroid abnormalities. Often no direct etiology is able to be determined. Progesterone supplementation and hCG supplementation have been investigated as interventions to prevent miscarriage in women with a history of recurrent pregnancy loss but

evidence for these therapies is inconclusive.[27,28] In some settings, midwives may order initial laboratory tests before referring to fertility or perinatology specialists.

Subchorionic Hemorrhage

Subchorionic hemorrhage is the term for bleeding that occurs between the chorion and the myometrium, or between the chorion and the placenta. This finding is noted in approximately 18% to 30% of women who present with first-trimester bleeding. Its etiology is unclear, and no consensus exists regarding a specific course of treatment. Many of these hemorrhages resolve spontaneously without adverse pregnancy outcomes. Larger subchorionic hemorrhages, however, have been associated with miscarriage.

Gestational Trophoblastic Disease and Hydatidiform Mole

Gestational trophoblastic disease refers to conditions that develop from abnormal placental (trophoblastic) tissue, the most common of which is hydatidiform mole. Gestational trophoblastic neoplasms can develop from gestational trophoblastic disease and include gestational choriocarcinoma, invasive moles, and placental site trophoblastic tumor. All of these conditions can progress to become malignant and life-threatening.[29]

The term *hydatidiform mole* is derived from the combination of the Greek word *hydatisia*, which means “drop of water,” and the Latin word *mola*, which means “false conception.” A hydatidiform mole develops following an abnormal union of sperm and egg. No or limited fetal tissue develops, while abnormal trophoblastic tissue proliferates and fills the uterine space. Placental villi become edematous grape-like structures that can be identified on ultrasound.[29] Hydatidiform mole occurs in approximately 1 in 15,000 pregnancies. Risk factors include younger maternal age (< 16 years) and older maternal age (> 40 years). The risk is higher for women who have had a previous molar pregnancy.[29]

A molar pregnancy is classified as either complete or partial. A complete mole occurs when an ovum that has lost its DNA is fertilized by a sperm, and the resulting products of conception have sperm chromosomal DNA only. No embryonic tissue develops—only overgrowth of trophoblastic tissue. Complete molar pregnancy is characterized by marked enlargement of the uterus and extreme elevations of hCG as the placental tissue synthesizes large amounts of this hormone. Women with a complete mole may have hyperemesis, hyperthyroid symptoms, and often early-onset hypertensive disorders.

A partial mole occurs when two sperm fertilize a normal ovum, and the result is usually a triploid chromosomal complement. Products of conception consist of nonviable fetal tissue and a smaller amount of smaller abnormal trophoblastic tissue. Partial moles have a smaller amount of trophoblastic tissue compared to complete moles, and women’s symptoms are correspondingly less severe.[29]

A woman with molar pregnancy needs referral to a consulting physician for care and follow-up. The plan of care usually includes dilatation and evacuation (D & E) rather than dilatation and curettage (D & C), because suction lessens the chance of uterine perforation and spread of trophoblastic tissue into the abdominal cavity. Hysterectomy is also an option. Medical

abortion with methotrexate and misoprostol is not advised due to the rare but severe risk of trophoblastic embolism with uterine contractions.[29]

After resolution of molar pregnancy, a woman will be advised to avoid becoming pregnant for 6–12 months. A pregnant woman who has a history of a molar pregnancy is offered a first-trimester ultrasound, and depending on the gestational age, she may be monitored with serial quantitative beta-hCG levels until a viable pregnancy is confirmed.

Fetal Conditions

Today, the availability of genetic testing in the first trimester and standard ultrasound examination at 18–20 weeks' gestation can provide information about fetal abnormalities that could not be detected antenatally in the past. Fetal abnormalities range from relatively mild (e.g., cleft lip) to lethal (e.g., anencephaly) and are often first diagnosed in the first or early second trimester.

Congenital Anomalies

When a severe congenital anomaly is diagnosed prenatally, the woman and family may face a range of decisions, including pregnancy termination, carrying the pregnancy, accessing fetal therapy/fetal surgery, the degree of interventions desired in labor, and post-delivery care of the newborn (including perinatal hospice for lethal conditions). Depending on the condition of the newborn at birth, the woman and family may have continued decision making about parenting the child, placing the newborn in foster care, or relinquishing the child for adoption. This complex decision-making process, as well as management of prenatal care and birth, involves an interdisciplinary team of providers—including genetic counselors, pediatricians, and maternal–fetal medicine experts. The midwife can play an important role in helping women and their families identify important questions they want to ask and in interpreting some of the information they are given. It is most important to provide time for the woman and her family during this process, and to recognize that information may need to be repeated a few times. It is not uncommon for a woman with a fetus with an anatomic anomaly to request periodic reconfirmation that the anomaly still exists. A repeat ultrasound should be provided if requested. Nondirective counseling is difficult and may need to be practiced ahead of time so that the midwife can use this approach competently.[30,31]

If the anomaly is lethal, and pregnancy termination is chosen, the plan regarding procedures such as fetal heart rate monitoring and viewing the infant after birth will be detailed ahead of time. Fetal therapy may be an option for women who have a fetus with specific anomalies. The North American Fetal Therapy Network—a collaborative group of 24 medical centers—provides specialized therapy for fetuses with neural tube defects, cardiac anomalies, dysrhythmias, ventral wall defects such as gastroschisis, and other anomalies as part of research protocols.

Incidental Ultrasound Findings

Several anatomic variants in the fetus can be identified during second-trimester ultrasounds. In the majority of cases, they are normal variations in fetal anatomy that are not associated with adverse effects. A few of these findings are more often seen in fetuses with trisomy 21 or trisomy 18, but studies have shown that most of these ultrasound markers do not modify the pretest risk for trisomy sufficiently to warrant further examination.

Table 22-9 reviews the prevalence of each variant and its association with aneuploidy and/or other fetal pathology.[32-34] Because the risk of trisomy 21 or trisomy 18 is generally low,

even if the likelihood ratio increases that risk by 1.5- or even 5-fold, it rarely increases the absolute risk significantly. In addition, the risk of miscarriage associated with amniocentesis is generally higher than the post-test risk for aneuploidy following one of these ultrasound findings. Providing support and clarifying factual information can help minimize adverse psychological impacts following the detection of a soft marker on ultrasound.

Table 22-9 **Incidental Ultrasound Findings**

Ultrasound Marker	Prevalence in General Population	Association with Aneuploidy	Description and Clinical Implications[a]
Markers Not Significant in the Absence of Aneuploidy[a]			
Choroid plexus cysts	0.1–3.6%	1% of identified cases are trisomy 18; no association with trisomy 21	Small fluid-filled sacs in the neuroepithelial folds within the choroid plexus. Often transient and considered a normal developmental variant.
Echogenic intracardiac focus	3–4%; as high as 30% in Asian women	LR 1.4–1.8 for trisomy 21	Small echogenic brightness in the left ventricle thought to be a microcalcification. Usually disappears later in pregnancy, and has no association with cardiac dysfunction. Increases risk of trisomy 21 by 1.5-fold, which usually leaves women in the low-risk category. Targeted ultrasound may be recommended if other risks for trisomy are present.
Markers Associated with Other Fetal Pathologic Conditions			
Echogenic bowel	0.2–1.8%	LR 5.5–6.7 for trisomy 21	Sonographic finding in which the fetal bowel appears bright. Associated with increased risk for trisomy 21, cystic fibrosis, fetal growth restriction, and infection. Genetic counseling is recommended to recalculate trisomy 21 risk; recommend parental screening for cystic fibrosis and evaluation for CMV.
Pelviectasis	0.5–5%	LR 1.5–1.8 for trisomy 21	Mild hydronephrosis; most often a transient state but may indicate renal dysfunction. Physician consultation and comprehensive ultrasound to rule out other structural anomalies and follow-up ultrasound at 32 weeks recommended. If still present, schedule postnatal renal ultrasound.
Nuchal fold thickening	0.5%	LR 8.6–49 for trisomy 21	Measurement between the outer edge of the occiput and the outer margin of the skin. Present in 40% of fetuses with trisomy 21. Also associated with Noonan syndrome and cardiac defects. Genetic counseling and fetal echocardiogram recommended.
Shortened humerus and femur	5%	LR 2.5–7.8 (shortened humerus) and 1.2–3.72 (shortened femur) for trisomy 21	May be a normal genetic predisposition, or an early indicator of fetal growth restriction or skeletal dysplasia. Physician consultation recommended. LR for humerus only should be used to adjust risk for trisomy 21, as it is more sensitive and specific than the LR for shortened humerus. One follow-up ultrasound is recommended in the third trimester to assess fetal growth.

Markers Associated with Other Fetal Pathologic Conditions			
Single umbilical artery	0.2–1.9% in single gestation; 4.9% in twin gestation	40% with trisomy 21 if other anomalies present	Single umbilical artery is an isolated finding associated with FGR and increased risk of perinatal morbidity in some studies, though not all. More research is needed. Physician consultation for management plan if an isolated finding. Refer for genetic counseling and additional diagnostic testing if associated with other markers.
Ventriculomegaly			Larger than expected ventricles in the fetal brain, often idiopathic or a normal variant. May be associated with infection, hydrocephalus, or other chromosomal abnormalities. Physician consultation is recommended; perform comprehensive ultrasound to look for other abnormalities.

Abbreviations: CMV, cytomegalovirus; FGR, fetal growth restriction; LR, likelihood ratio.

[a] These findings are not clinically significant for a woman who has a low risk for aneuploidy according to the results of first- and second-trimester screening, or if the woman had a diagnostic test to rule out chromosomal abnormalities.

Based on Norton ME. Follow-up of sonographically detected soft markers for fetal aneuploidy. *Semin Perinatol.* 2013;37:365-369[32]; American College of Obstetricians and Gynecologists. Practice Bulletin No. 175: ultrasound in pregnancy. *Obstet Gynecol.* 2016; 128:e241-e256[33]; Rao R, Platt LD. Ultrasound screening: status of markers and efficacy of screening for structural abnormalities. *Semin Perinatol*. 2016;40(1):67-78.[34]

Fetal Growth: Large for Gestational Age

Serial measurement of fundal height is used to monitor fetal growth and as an initial screen for excessive growth or fetal growth restriction.[35] Although fundal height measurements are not highly sensitive, serial recordings following use of a standard technique is standard prenatal practice. When fundal heights are greater than expected, possible diagnoses include multifetal gestation, polyhydramnios, and macrosomia. Ultrasound can determine the number of fetuses and amount of amniotic fluid present, but does not reliably detect macrosomia. The fetus whose estimated weight is greater than the 90th percentile for gestational age is considered large for gestational age (LGA). A fetus estimated to weigh more than 4000 grams is considered macrosomic.

Risk factors for macrosomia include maternal diabetes, maternal weight gain, multiparity, Hispanic race, history of macrosomia in a prior pregnancy, and post-term gestation.[36] Macrosomia is highly associated with maternal diabetes. Fetuses of women who have diabetes develop more body fat that is preferentially centered in the shoulders and upper body. These fetuses are at increased risk for shoulder dystocia, birth trauma, and cesarean section. Because the ultrasound diagnosis of macrosomia is imprecise, the American College of Obstetricians and Gynecologists does not recommend induction of labor for suspected macrosomia. An elective cesarean section may be offered if the estimated fetal weight is more than 5000 grams in a woman without diabetes or if the estimated fetal weight is more than 4500 grams in a woman with diabetes.[36]

Fetal Growth: Fetal Growth Restriction

When serial fundal heights do not increase as expected, an ultrasound for fetal growth should be ordered. Fetal growth restriction is diagnosed when the fetus has an estimated weight less than the 10th percentile for gestational age.[37] Although some fetuses who are less than the 10th percentile for their gestational age are constitutionally small without compromise, others have some cause for their growth restriction.

Historically, fetuses who were more than two standard deviations smaller than average were termed "intrauterine growth retarded." This term was changed to "intrauterine growth restriction" (IUGR) and in recent practice has been adjusted to "fetal growth restriction" (FGR). The term "small for gestational age" (SGA) is reserved for an infant born at term who is below the 10th percentile for weight for his gestational age.[37] When FGR is related to an abnormal condition, perinatal morbidity and mortality are significantly increased. The etiology of FGR can be categorized as fetal, maternal, or placental (**Table 22-10**).[38]

Table 22-10 Etiology of Fetal Growth Restriction

Fetal Factors	Maternal Factors	Placental Factors
Chromosome abnormalities	Extremes of maternal age	Abnormal placental attachment to the uterus
Genetic syndromes	Maternal disease (e.g., hypertension, pregestational diabetes, cardiac, renal insufficiency, sickle cell anemia, systemic lupus erythematosus)	Reduced placental perfusion secondary to maternal vascular disease
Intrauterine infections: particularly viral	Malnutrition	Umbilical cord abnormalities
Multifetal gestation	Previous history of fetal growth restriction	
	Substance use	
	Teratogen exposure	
	Uterine malformation	

Based on Nardozza LM, Caetano ACR, Zamarian AC, et al. Fetal growth restriction: current knowledge. *Arch Gynecol Obstet.* 2017;295:1061-1077.[38]

Fetal growth restriction is generally subdivided into three categories: symmetrical, asymmetrical, or a combination of the two. A fetus with symmetrical fetal growth restriction will have both the fetal body and head equally smaller than expected. The most common cause of symmetrical growth restriction is congenital anomalies. Symmetrical growth restriction that occurs early in gestation and is progressively severe—that is, the delay becomes more pronounced as pregnancy continues—is associated with severe maternal malnutrition, low prepregnancy weight/no weight gain, multifetal gestation, chromosomal abnormalities, perinatal infections, and exposure to drugs or environmental teratogens. The prognosis for this fetus is poor.

The second category of fetal growth restriction is asymmetrical growth restriction. This condition is the result of compromise later in fetal life, typically after 30 weeks' gestation. Asymmetric growth restriction can be caused by any condition that causes decreased placental blood flow or decreased oxygenation of the fetus. In this case, the fetus's estimated weight is below the 10th percentile but the head circumference is larger than the 10th percentile. Factors

associated with asymmetric fetal growth restriction include maternal hypertension, renal disease, collagen vascular disease, microvascular disease of diabetes, cyanotic heart disease, and hemoglobinopathies.

The third subcategory of fetal growth restriction is a combination of symmetrical and asymmetrical growth restriction. The etiology of this type is often either maternal infection (e.g., rubella, cytomegalovirus) or exposure to toxins (e.g., medications, illicit drugs).

Perinatal complications for fetuses with growth restriction include complications associated with the etiology, such as intrauterine infection or maternal hypertension. Growth restriction from causes associated with inadequate placental perfusion places the fetus at increased risk for poor oxygenation, fetal compromise, and fetal death. Fetal growth restriction with absent or reversed end-flow Doppler velocimetry indicates a fetus at great risk.

Once fetal growth restriction is identified on ultrasound, prompt physician consultation or maternal–fetal medicine referral can provide the comprehensive fetal survey ultrasound and other evaluations that may be needed. Ongoing monitoring and determination of the best time for induction of labor will depend on the underlying etiology, gestational age, pattern of fetal growth, and results of fetal surveillance tests; therefore, physician consultation is required.

Multifetal Gestation

Multifetal gestation refers to a pregnancy in which two or more fetuses are present. The incidence of multifetal gestation in the United States increased 76% between 1980 and 2009, largely because of the increased use of assisted reproductive technologies, including ovulation-inducing medications. Twin pregnancies can develop secondary to implantation of two fertilized ova, or when one fertilized ovum cleaves and develops into two fetuses (**Figure 22-2**). Monozygotic twins (identical) develop from one egg and one sperm; dizygotic twins (fraternal) develop from two eggs and two sperm. Monozygotic twins may be monochorionic or dichorionic. Dizygotic twins will always be dichorionic and, therefore, diamniotic. Chorionicity in monozygotic twins depends on when, during the early gestational period, the zygote splits. It includes the following possibilities:

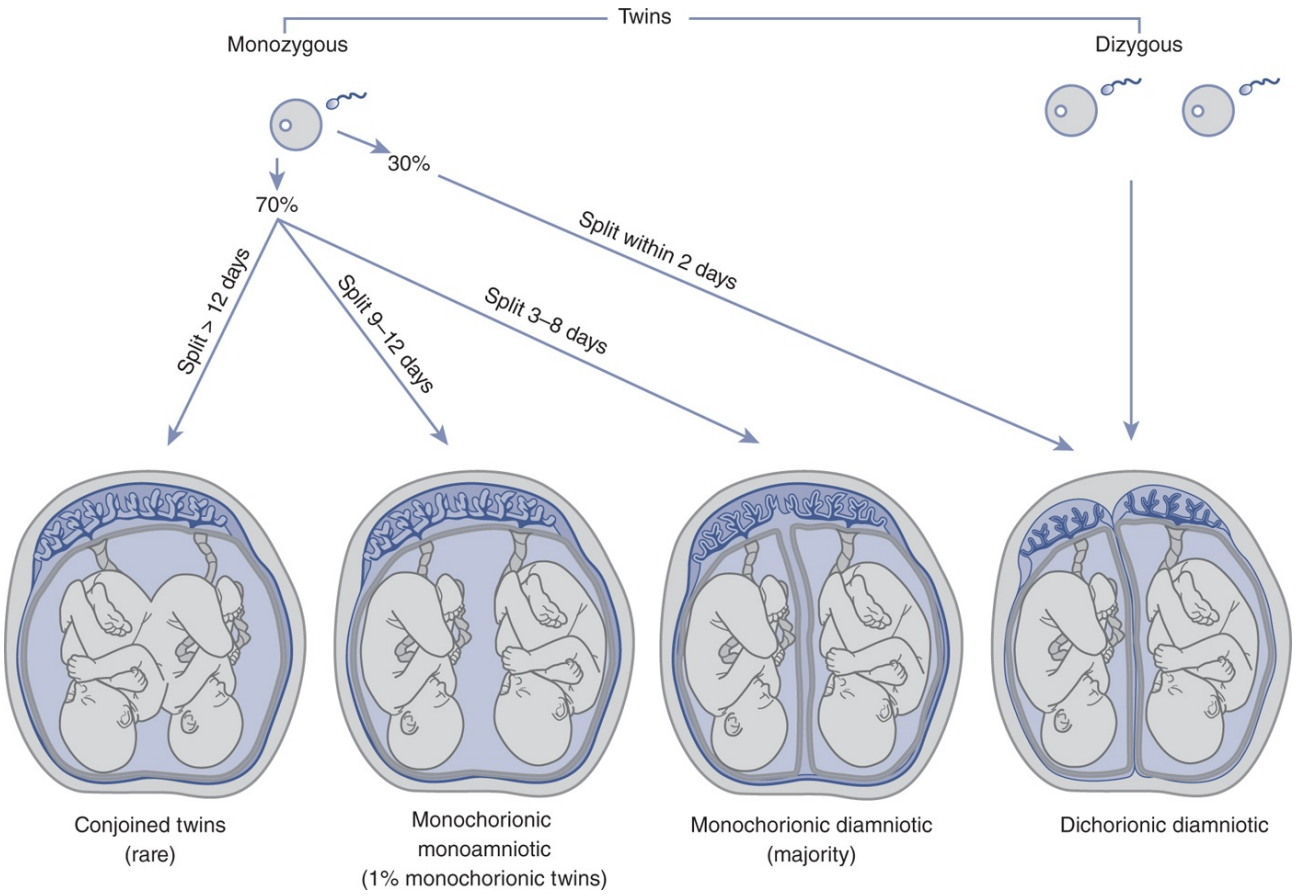


Figure 22-2 Zygosity of twin gestations.

- Diamniotic/dichorionic, which occurs if cleavage happens by day 3 after fertilization
- Diamniotic/monochorionic, which occurs if the zygote splits between days 4 and 8
- Monoamniotic/monochorionic, which occurs if cleavage happens between days 8 and 13

Zygosity cannot be determined prenatally in all cases. When both fetuses are the same gender and each has a separate placenta, the pregnancy could be either monozygotic or dizygotic. Monoamniotic/monochorionic chorionicity is rare and increases the risk for perinatal mortality, secondary to the high likelihood of cord entanglement and cord strangulation. If the zygote splits after day 13, the embryos have not had time to separate and they develop as conjoined twins.

It is common for a woman with a multifetal gestation who receives prenatal care in the first trimester to have an initial uterine size that is consistent with her dates. The uterus starts to become larger than expected in the early second trimester. Diagnosis is confirmed via ultrasound, which will determine fetal number, fetal anatomy, and chorionicity of placentation.

Once the diagnosis of multiple pregnancy is made, the woman is referred to a physician for care, although a midwife may participate in conducting prenatal visits as part of an interdisciplinary team approach. Prenatal care management includes more frequent prenatal visits, increased surveillance for complications, serial ultrasounds to monitor fetal growth, dietary counseling, iron supplementation, and earlier changes in home and work responsibilities. Fetal growth and development is monitored with ultrasound examinations that

are generally performed every 3 to 4 weeks from 20 weeks' gestation until term. Discordant growth or suggestion of lagging growth in either or both fetuses requires urgent physician evaluation.

Women with twin gestations may benefit from extra counseling and anticipatory guidance that address several topics unique to multifetal gestation. In these areas, midwifery care can complement medical care for women who are pregnant with twins. For example, women with twins need more protein and calories. The recommended weight gain with a twin gestation is 40–45 pounds for women with a normal body mass index (BMI), 42–50 pounds for underweight women, and 30–35 pounds for overweight women. Folic acid supplementation remains at 1 mg daily, as with singleton pregnancy. A daily prenatal multivitamin can provide the needed micronutrients, including 30 mg of elemental iron. Supplementation with calcium and Vitamin D is recommended and docosahexaenoic acid (DHA) should be considered for women with a twin pregnancy.[39]

Women with multifetal gestations are at increased risk for several conditions. Management is first based on chorionicity, as monochorionic twins have a higher rate of complications than do dichorionic twins. Twin-to-twin transfusion syndrome is one of the most serious complications associated with multifetal gestation; it occurs more frequently in women who have monochorionic placentation. Twin-to-twin transfusion syndrome can occur in monochorionic twins when two placentas are conjoined. Abnormal blood vessels form between the two placentas such that one twin receives an excess of blood (recipient) and the other twin (donor) receives too little blood. The donor twin may become significantly anemic and growth restricted, and may develop oligohydramnios. The recipient twin may have polyhydramnios, as well as volume overload leading to cardiomegaly and cardiac failure. The development of discordant growth in monochorionic twins with oligohydramnios in one twin and polyhydramnios in the other is diagnostic for twin-to-twin transfusion syndrome. Management of twin-to-twin transfusion syndrome requires maternal–fetal medicine and other subspecialized physicians who can consider procedures to maximize the outcome for the pregnancy.

For all women with multifetal gestations, major fetal complications are preterm birth and fetal growth restriction. Maternal complications may include placenta previa, gestational diabetes, preeclampsia, malpresentation, and dysfunctional labor. Less common complications include acute fatty liver disease, pruritic urticarial papules and plaques of pregnancy (PUPPP), and pulmonary embolus.

Prevention of preterm labor is a major priority in caring for women with a multifetal gestation. Management may include limiting activity and increasing rest periods throughout pregnancy. Bedrest has not been found to be an effective means to prevent preterm labor in multifetal gestations. Likewise, prophylactic cerclage and progesterone therapy have not been found to be effective in preventing preterm birth in such gestations. Instruction regarding sexual activity is based on cervical findings, previous obstetric history, and frequency of Braxton Hicks contractions. Careful attention to signs of hypertension, preeclampsia, and gestational diabetes is especially important for women with a multifetal gestation, and development of these signs warrants early intervention. Maternal morbidity will increase the already elevated risk of preterm birth by increasing the need to induce birth for maternal indications.

Plans should be made for a physician to attend the birth, and the mode of birth will depend on the position of both fetuses and physician expertise. The most common presentation is vertex/vertex. Next most common is breech with vertex or breech, and the third most common presentation is vertex/breech. The breech/vertex presentation creates a risk that the fetal heads may become locked. A cesarean section is recommended in that situation. Depending on the skills of the physician, a second twin breech may be delivered vaginally by the physician.

Fetal Malpresentations at Term

The three possible fetal presentations, as reviewed in the *Anatomy and Physiology of Pregnancy* chapter, are cephalic, breech, and shoulder. Subcategories of cephalic presentation include face, brow, and occiput; the latter is further categorized as occiput anterior, occiput transverse, or occiput posterior positioning. The breech presentation can present as a complete breech, frank breech, or footling breech.

Cephalic Malpresentations

Cephalic malpresentations, which include vertex, sinciput, brow, face, or chin, are diagnosed during labor and, therefore, are reviewed in more detail in the *Complications During Labor and Birth* chapter. Fetuses that are in a brow or face presentation usually convert to occiput presentation during labor and birth, but may be born in one of these positions if the fetus is very small. Risk factors for brow and face presentation include prematurity, grand multiparity, polyhydramnios, multiple nuchal cords, and congenital anomalies such as anencephaly or hydrocephalus.

Breech Presentation

By 35 to 36 weeks' gestation, the majority of fetuses will spontaneously settle into a vertex presentation. In approximately 3% to 4% of cases, however, at term the fetus will be in a breech presentation with the buttocks or feet presenting.[40] There are three types of breech presentation: frank, complete, or footling. In a *frank* breech presentation, the hips are flexed and the knees extended, with the infant's feet near the head. The buttocks (breech) are presenting at the pelvic inlet. A *complete* breech is similar, with the buttocks as the presenting part in the pelvis. The lower knees are flexed, however, and the infant is in a crossed-legged position. If one or both feet are extended down below the buttocks, this is referred to as a single or double *footling* breech. When assessing position in a fetus in a breech presentation, the fetal sacrum is used as the point of reference—for example, *left sacrum anterior* (*LSA*) (**Figure 22-3)**.

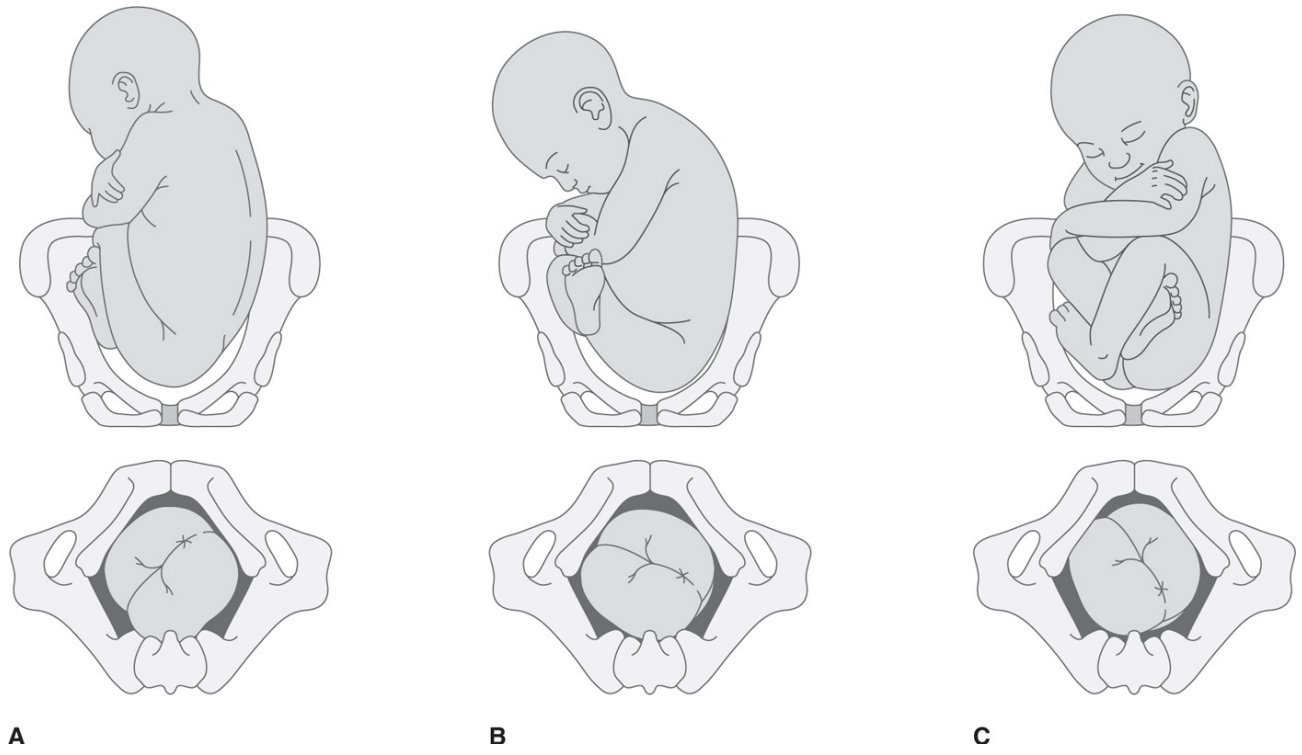


Figure 22-3 Breech positions. **A.** Left sacrum anterior. **B.** Left sacrum transverse. **C.** Left sacrum posterior.

Prematurity, multifetal gestation, multiparity with lax uterine tone, polyhydramnios or oligohydramnios, previous breech presentation, and placenta previa have all been associated with an increased risk of breech presentation.[40] In addition, uterine anomalies such as a bicornuate uterus or the presence of large fibroids in the lower uterine segment may impede a normal vertex presentation. Breech presentation is also associated with an increased incidence of fetal anomalies such as hydrocephaly or anencephaly.[40]

A breech presentation is often identified during a prenatal visit using Leopold's maneuvers when the firm ballottable vertex is palpated in the upper fundal region. The woman may report feeling increased movement in the lower pelvic area and the fetal heart tones may be found in the upper abdomen. Prior to 35 weeks' gestation, this finding is normal but should be noted in the prenatal chart, especially if the woman has any of the known predisposing risk factors. Approximately 10% of breech fetuses will spontaneously revert to vertex presentation after 36 weeks' gestation.[40] The midwife should, therefore, have a low threshold for ordering an ultrasound at 35 to 36 weeks' gestation to confirm the presentation if breech presentation is suspected.

Vaginal birth of a fetus in a breech position is associated with increased risks for perinatal morbidity and mortality. The head is the largest part of the fetus. When the fetal head passes through the cervix and vagina, the rest of the body follows without difficulty. In a breech presentation, however, the body can pass through a partially dilated cervix and cephalopelvic disproportion may not be detected until the head becomes entrapped after the body is born. If a breech presentation is diagnosed after 35 weeks' gestation but before the onset of labor, the midwife should consult with a physician to determine whether the woman is a candidate for an

external cephalic version. During this procedure, external pressure is manually exerted on the woman's abdomen to physically rotate the fetus to a vertex presentation. External cephalic version is successful in approximately 58% of cases.[41] Factors that decrease the success of external cephalic version include oligohydramnios, anterior placenta, breech engaged in the pelvis, fetal back located posteriorly ("back down"), and maternal obesity.[42] External cephalic version should not be performed if the woman has any contraindication to vaginal birth (e.g., placenta previa) or if any of the following conditions are present: ruptured membranes, fetal or uterine anomalies, nonreassuring fetal heart rate patterns, hyperextended fetal head, placental abruption, or multifetal gestation.[42]

External cephalic version is performed only in a hospital setting, ideally between 36 and 37 weeks' gestation. In addition to a prerequisite reassuring fetal heart rate assessment, the woman may be given a dose of subcutaneous terbutaline (0.25 mg) to induce uterine relaxation before the procedure is attempted.[41] External cephalic version is done under direct ultrasound guidance with continuous monitoring of the fetal heart rate (by an assistant). Women can experience considerable discomfort during this procedure, so external cephalic version is sometimes attempted under epidural or spinal anesthesia. The most common fetal side effect of external cephalic version is a transient nonreassuring fetal heart rate pattern, such as a prolonged deceleration.[41] In rare circumstances, external cephalic version has been reported to cause vaginal bleeding, placental abruption, fetal compromise, fracture of fetal limbs, and fetal death.[41] An emergency cesarean may be required if fetal compromise is noted. Midwives often assist during external cephalic version by providing ultrasound guidance during the procedure and/or offering emotional support and comfort to the woman. After the procedure, fetal heart rate and contraction monitoring should continue for at least 1 hour depending on institutional policies.

Various maternal position change exercises that might turn breech presentations to vertex have been proposed (e.g., resting with hips elevated and knee–chest positions), but research has not shown these methods to be successful.[43] Women with persistent breech presentations at 36 to 37 weeks' gestation will need a referral to an obstetrician for birth planning.

Placental, Amniotic Fluid, and Umbilical Cord Abnormalities

Placental abnormalities fall into three categories: (1) abnormal placental size or structure, (2) abnormal placement within the uterus, and (3) abnormal attachment. Some morphologic placental variations can be detected prenatally via ultrasound. Some are associated with adverse fetal effects, or may cause maternal or fetal bleeding. In general, the placental size correlates with the size of the fetus. An abnormally small placenta is associated with intrauterine infection and fetal growth restriction, whereas an abnormally large or edematous placenta is associated with conditions such as maternal diabetes and fetal hydrops.

Circumvallate Placenta

Abnormal placenta configurations are shown in **Figure 22-4**. Circumvallate placenta is a placenta with a double layer of membrane (chorion and amnion) that circles the edge of the placenta on the maternal side and appears as a whitish ring.[44] The fetal vessels do not extend beyond this ring, but extrachorionic placental tissue does. The etiology of circumvallate placenta is not well known. Retrospective studies have found an association with circumvallate placenta and an increased incidence of second-trimester bleeding, preterm birth, and preterm premature rupture of membranes.[45] In contrast, a circummarginate placenta has a ring of membrane that is flat and close to the edge of the placental disc. Circummarginate placentas are not associated with adverse outcomes.

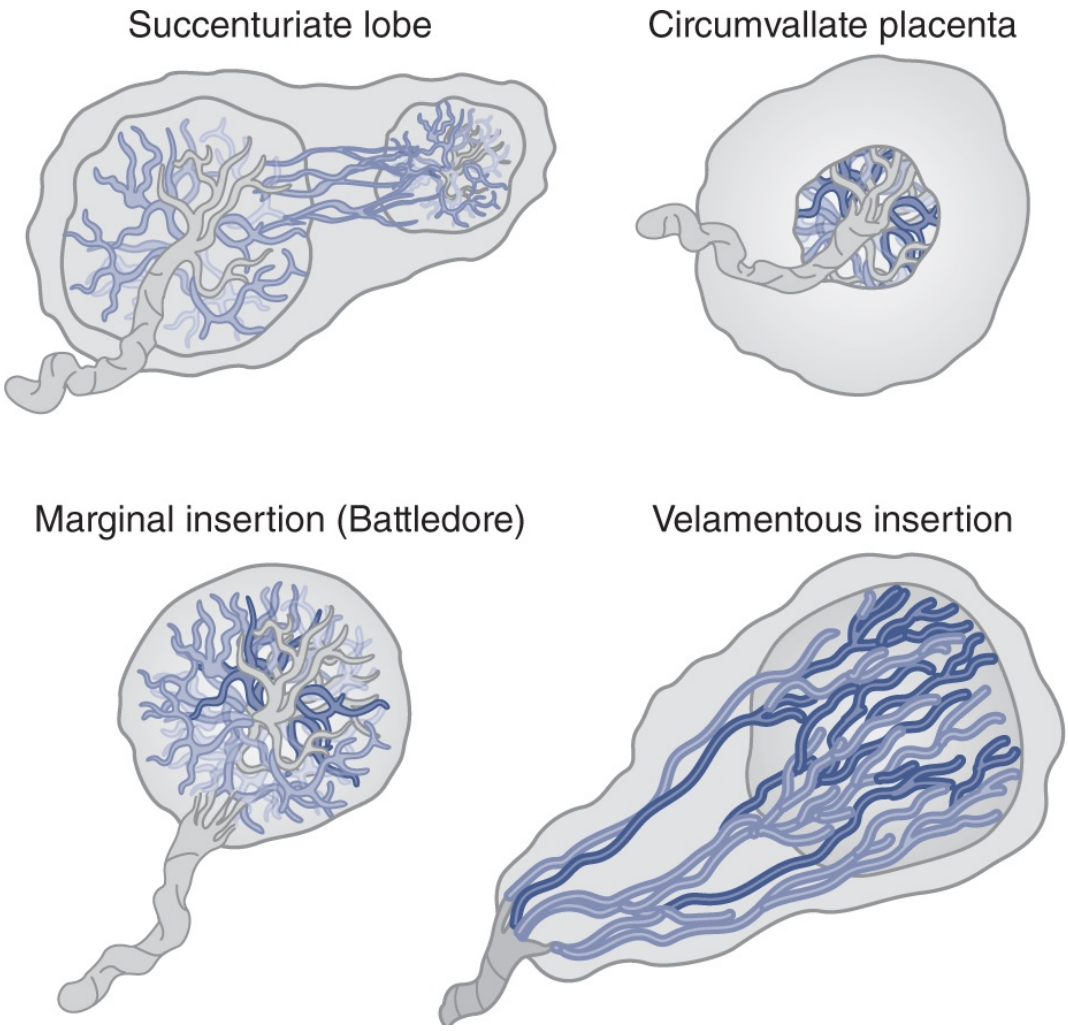


Figure 22-4 Abnormal placental configurations.

Succenturiate Lobe

A placenta with a succenturiate lobe has an accessory lobe or cotyledon separate from the primary placental mass. The succenturiate lobe, which is connected to the primary mass by a fetal artery and vein, functions normally. Succenturiate lobes are often clinically insignificant but may be associated with other placental abnormalities such as velamentous cord insertion. The most common problem associated with this placental variation is retained placenta after birth, which is one of the reasons the placental edge is examined after birth to look for torn vessels that extend beyond the placental margin.

Umbilical Cord Abnormalities

Umbilical cord variations include, unusual insertions, single umbilical artery, and excessive or low coiling. The umbilical cord is normally inserted centrally on the fetal side of the placenta. *Marginal insertion*, also called *battledore placenta*, refers to insertion of the umbilical cord on the edge of the placental mass. Fewer than 10% of placentas exhibit a marginal insertion, though this morphologic variation is not associated with adverse effects.

Umbilical Cord Insertion

Velamentous cord insertion is a structural developmental abnormality of the placenta in which the cord is inserted in the membrane some distance from the placental tissue, leaving a span of vessels not protected with Wharton's jelly that traverse the membrane before being integrated into the placenta. A velamentous cord insertion can occur with a normal placenta or between succenturiate lobes of a placenta. If the velamentous vessels cross the internal cervical os, the condition is referred to as *vasa previa*. Risk factors for a velamentous cord insertion and vasa previa include placenta previa, multifetal gestation, history of assisted reproductive technology, obesity, and smoking.[46] Doppler ultrasound may or may not identify the abnormal vessels; indeed, the diagnosis is often made at birth.

Any umbilical cord vessels that are not protected within the structure of the umbilical cord are vulnerable to rupture and compression during labor and birth.[46] Rupture of the velamentous vessels can cause rapid fetal exsanguination. These abnormalities are also associated with an increased risk for preterm birth, fetal growth restriction, and perinatal morbidity/mortality, although the absolute risk for these events is low. Notably, studies that have correlated velamentous insertion of perinatal outcomes may be confounded by other complications such as multifetal gestation or placenta previa. Nonetheless, physician consultation is indicated for a woman who has a velamentous cord insertion diagnosed prenatally. Additional antepartum surveillance may be recommended, and the woman is counseled to come into a hospital setting as soon as labor starts for continuous fetal heart rate monitoring. A note is placed in the woman's health record where the intrapartum care providers can see it, because delivery of the placenta needs to be performed gently to avoid tearing the cord off.

Labor and cervical dilation in the presence of vasa previa poses a risk for life-threatening fetal hemorrhage. Women with a vasa previa require physician management in a Level III or Level IV setting. Recommendations for care include weekly fetal surveillance, administration of corticosteroids to stimulate fetal lung maturity, and planned preterm cesarean birth that is scheduled for 34 weeks' gestation but may be considered earlier if needed.[47]

Single Umbilical Artery

The umbilical cord usually has two arteries and one larger vein. Occasionally, a fetus will have only one artery and one vein. The mechanism is presumed to be secondary atresia of one of the arteries. The prevalence of one umbilical artery is less than 1%. However, the prevalence is higher in fetuses with congenital anomalies, and those who are stillborn. When this finding is noted, a comprehensive ultrasound is performed to assess for other anatomic abnormalities, in particular cardiac and renal abnormalities. The incidence of aneuploidy is not increased and therefore invasive genetic diagnosis is not indicated although non-invasive testing for fetal DNA to screen for trisomy 21, 18, or 13 may be offered if not already performed.

If the fetus does not have other abnormalities, a single umbilical artery is associated with a small increase in small for gestational age. Other complications such as preeclampsia, preterm birth, and perinatal mortality have been reported but studies on these outcomes are inconclusive. In some settings, serial ultrasounds to monitor growth and fetal testing in the third

trimester is recommended but these recommendations are not universal so physician consultation is obtained. Most women with this isolated finding can be reassured that there are no additional risks for the fetus.

Umbilical Cord Coiling

One interesting umbilical cord finding is the degree of umbilical coiling present. Umbilical cords that are hypo- or hyper-coiled are associated with increased perinatal morbidity. Attempts to correlate the actual degree of coiling with perinatal outcomes have not been fruitful.[48] Umbilical cord coiling is not a standard component of an ultrasound examination, and changes in prenatal management are not recommended if either variation is noted. Nevertheless, hypo- or hyper-coiling is occasionally reported. In such a case, a detailed examination and review of the woman's history may be of value and physician consultation is indicated.

Placenta Previa

When the placenta implants over the internal cervical os, the condition is referred to as placenta previa. Placenta previa occurs in approximately 1 in 200 births. It is often diagnosed during routine ultrasonography in the second trimester.[46,49] Approximately 2% of women will have placenta previa identified during the second-trimester standard ultrasound, but 90% of these placentas will no longer be located over the cervical os later in pregnancy as the placenta grows toward the fundus.

In the past, placenta previa was categorized by the distance from the placental edge to the internal cervical os and classified as complete, partial, marginal, or low-lying (**Figure 22-5**). The availability of transvaginal ultrasound has allowed for a high degree of accuracy in measuring the distance between the placental edge and the cervix and has resulted in a change in terminology.[49] All placentas covering the os to any degree are now given the diagnosis of placenta previa; placentas close to but not covering the os are referred to as *low-lying placenta*.[49]

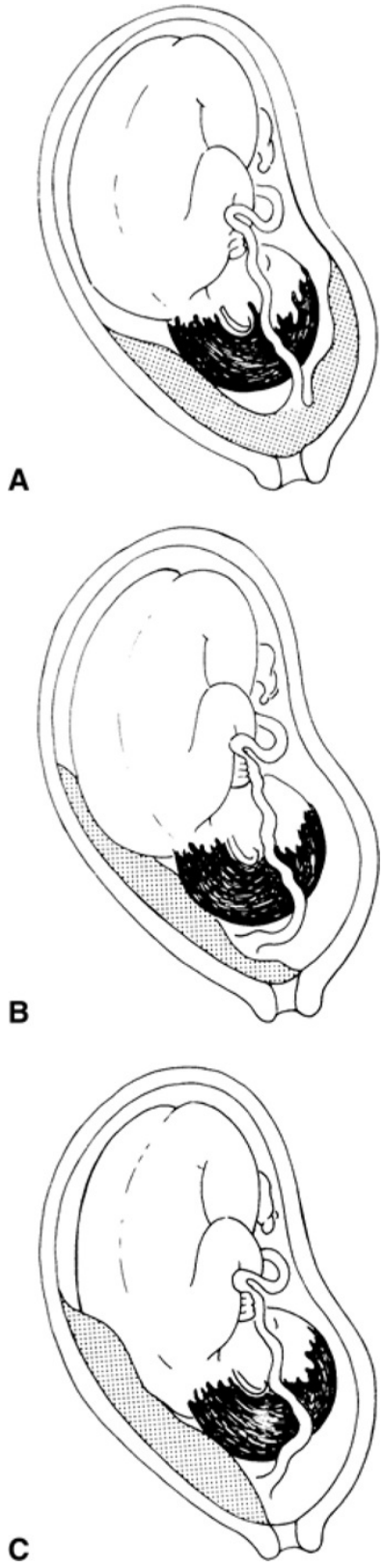


Figure 22-5 Placenta previa. **A.** Complete. **B.** Partial. **C.** Low-lying.

The primary risk factor for development of placenta previa is previous uterine scarring secondary to cesarean section or uterine surgery.[50] Additional risk factors are maternal age, parity, multifetal gestation, male fetus, smoking, cocaine usage, multiple prior pregnancy

terminations, and use of assisted reproductive technology.[49] This risk increases with repeat cesarean sections.[51]

The greatest risks associated with placenta previa are maternal hemorrhage and premature birth. When the cervix starts to remodel (effacement and dilation) in preparation for labor and birth, the portion of the placenta resting over the cervix separates, which causes bleeding. The gestational age at which vaginal bleeding first occurs is unpredictable. In the majority of women, the initial bleeding episode presents as a painless gush of blood (the amount is also unpredictable) prior to 36 weeks' gestation.

Once placenta previa is diagnosed, repeat ultrasound is recommended at 32 weeks, and then again at 36 weeks (if the placenta is still a previa at the 32-week examination). If the placenta remains encroaching over the cervix at 36 weeks' gestation, vaginal birth is contraindicated. Women are advised to avoid sexual intercourse with vaginal penetration and orgasm so as to avoid direct trauma to the cervix or uterine contractions, and to call or come to the hospital immediately if vaginal bleeding occurs. Physician consultation or referral is indicated, as the management plan for ongoing prenatal care may include administration of corticosteroids to enhance lung maturity as well as plans for cesarean birth. Acute care of women who present with vaginal bleeding is reviewed in the *Complications During Labor and Birth* chapter.

Placenta Accreta

Placenta accreta, increta, and percreta are abnormalities of placental implantation in which the anchoring placental villi attach to the myometrium instead of the decidua (endometrium). *Placenta accreta* is diagnosed when the cytotrophoblast attaches to the myometrium, *placenta increta* occurs when the cytotrophoblast invades into the myometrium, and *placenta percreta* occurs when the cytotrophoblast tissue extends through the myometrium and serosa to attach to adjacent maternal organs (**Figure 22-6**).[50,51] The term "placenta accrete" is often used to describe all three variants of abnormal placental implantation.

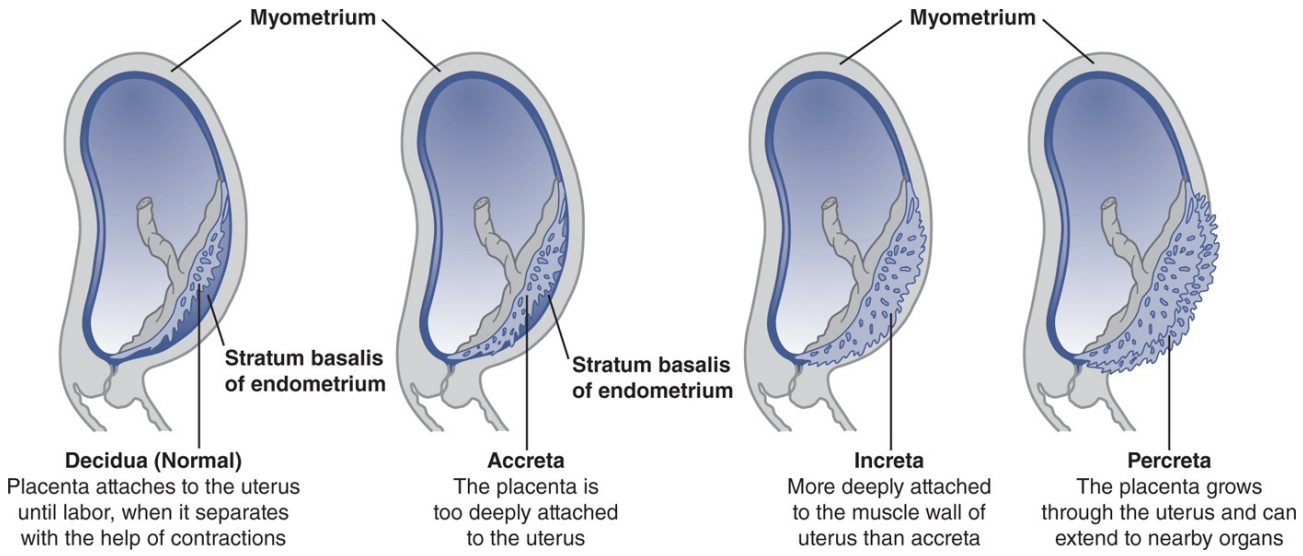


Figure 22-6 Placenta accreta.

Placenta accreta occurs when the placenta implants over an area of the uterus that is scarred or damaged. The incidence of placenta accreta is increasing as a result of the rising rate of cesarean birth and uterine procedures associated with assisted reproductive technology (such as myomectomy or resection of the uterine septum). Placenta accreta is also more common in women who have placenta previa. In women with prior cesarean births who develop placenta previa in a subsequent pregnancy, 3% will develop placenta accreta if they have a history of one prior cesarean and current previa. The incidence of accreta increases to 11% in women with placenta previa who had two prior cesareans, and to 40% in women with placenta previa who had three prior cesareans.[52]

Hemorrhage at birth is anticipated in all cases of placenta accreta. The severity of hemorrhage and associated complications are related to the type of placenta accreta. Placenta increta and certainly placenta percreta are life-threatening situations for the woman and frequently require hysterectomy for control of hemorrhage because the placenta cannot be removed.

Although placenta accreta has characteristic findings on ultrasound, ultrasound is not able to diagnose this condition in all cases. The role of the midwife is to be alert to risk factors such as an ultrasound that shows an anterior low-lying placenta in a woman who had a previous cesarean section. Consultation with or referral to maternal–fetal medicine is indicated in such cases. Specialized ultrasound and magnetic resonance imaging (MRI) can aid in determining the diagnosis. If placenta accreta is diagnosed, planned cesarean birth in a Level III or Level IV center is needed. For women with placenta increta or percreta, care in a Level IV site is recommended. Massive transfusion protocols and subspecialist resources are essential to manage this serious and often life-threatening complication of pregnancy.

Abnormal Amniotic Fluid Volume

Amniotic fluid is primarily composed of fetal urine and lung fluid. The amount of amniotic fluid increases throughout pregnancy until it reaches approximately 1000 milliliters by the beginning of the third trimester; it then gradually decreases to approximately 800 milliliters at term. Amniotic fluid volume can be measured on ultrasound in two ways: by the amniotic fluid index (AFI) or as a single maximum vertical pocket of amniotic fluid. The AFI is a composite value taking into account the maximum vertical pocket in each quadrant of the uterus. Particularly when screening for low amniotic fluid volume, using a maximum vertical pocket, rather than AFI, to diagnose abnormal volume is associated with fewer interventions without an increase in poor pregnancy outcomes.[53-55]

Polyhydramnios (Hydramnios)

Polyhydramnios, also called hydramnios, is an excessive amount of amniotic fluid. It is characterized as an AFI of 24 cm or more, or a maximum vertical pocket that is 8 cm or more.[56] This condition is idiopathic in 50% to 60% of women and most likely to become evident in the third trimester. Several conditions are associated with a higher incidence of polyhydramnios, including fetal structural anomalies, multifetal gestation (especially

monozygotic twins, twin-to-twin transfusion syndrome), diabetes, fetal infection, and fetal chromosomal abnormalities. Severe polyhydramnios (e.g., AFI > 35 cm) is more likely to be associated with fetal structural anomalies, such as tracheoesophageal fistula, anencephaly, and meningomyelocele, whereas milder forms are more likely to be idiopathic.[56] Complications of polyhydramnios include preterm labor secondary to uterine distension, premature rupture of membranes, malpresentation of the fetus, umbilical cord prolapse, abruptio placenta, dysfunctional labor, and postpartum hemorrhage.

Polyhydramnios is initially suspected when uterine enlargement, maternal abdominal girth, and fundal height are larger than expected for the fetus's gestational age. It may be difficult to auscultate fetal heart tones and to palpate the fetal outline and fetal parts. The fetus may have an unstable lie—and a change in lie may be detected during Leopold's maneuvers. In severe cases of polyhydramnios, the woman may experience dyspnea; lower-extremity and vulvar edema; pressure in the back, abdomen, and thighs; heartburn or nausea; and vomiting.

An ultrasound will confirm the diagnosis and identify any coexisting fetal or placental abnormalities. At this point, consultation with a physician is needed. Additional laboratory evaluation may include rescreening for gestational diabetes, antibody titers to detect isoimmunization, or TORCH (toxoplasmosis, other, rubella, cytomegalovirus, and herpes) titers to assess for infection depending on the degree of polyhydramnios and other findings. Of note, polyhydramnios alone is not an indication for aneuploidy screening by karyotyping.[57] Likewise, uncomplicated polyhydramnios is not an indication for delivery. The diagnosis warrants increased fetal surveillance and attention to the potential mechanical difficulties of dyspnea and edema for the woman. On rare occasions, the polyhydramnios is so severe that fluid must be removed via controlled amniocentesis to relieve maternal respiratory distress.

Oligohydramnios

Oligohydramnios is an abnormally low volume of amniotic fluid, adjusted for the gestational age of the pregnancy. This condition may be suspected on clinical examination but needs to be confirmed via ultrasound. Clinical signs include a fetus that is easily outlined during abdominal palpation, a fetus that is not ballottable, and lagging fundal height measurements. The diagnosis of oligohydramnios is made when the amniotic fluid index in the third trimester is less than 5 cm in a woman with a singleton pregnancy.

A number of fetal conditions are associated with oligohydramnios, including congenital anomalies (e.g., renal agenesis, Potter's syndrome), viral diseases, fetal growth restriction, uteroplacental insufficiency, preterm premature rupture of membranes, response to indomethacin as a tocolytic, and post-term pregnancy.

Oligohydramnios can develop at any time in pregnancy, but its clinical significance in the first trimester is not clear. The fetus normally begins to urinate and swallow fluid in the second trimester, so disorders of the fetal renal/urinary system become problematic at this time. If oligohydramnios is present between 17 and 26 weeks' gestation, the fetus is at risk for developing hypoplastic lungs.[58] In this condition, the fetal lungs do not develop to full size because the fetus is unable to breathe amniotic fluid, a process that normally causes chest expansion and growth.

Oligohydramnios that appears in the third trimester is often associated with uteroplacental insufficiency and fetal growth restriction or preterm premature rupture of membranes. Thus, evaluation of oligohydramnios focuses on physical, ultrasound, and laboratory examinations that can determine the underlying etiology.

Management of oligohydramnios is directed toward management of the underlying etiology, and physician referral is indicated to establish the plan of care. Oligohydramnios without ruptured membranes in an indication for frequent surveillance of fetal well-being, including fetal movement counts, nonstress tests (NST), a biophysical profile (BPP), and possibly umbilical Doppler studies. The timing of birth depends on the evidence for fetal well-being during frequent surveillance. When uncomplicated oligohydramnios occurs in a post-term pregnancy, and tests of fetal well-being are reassuring, there is limited high-quality evidence to support induction over expectant management.[59]

Cervical and Uterine Abnormalities

Two anatomic variations of the cervix can have adverse consequences during pregnancy. First, the cervix that is unusually short is associated with an increased risk for preterm birth. Second, cervical insufficiency refers to a structural weakness in the cervix that results in painless cervical dilation and miscarriage in the early second trimester of pregnancy. Uterine abnormalities such as bicornuate uterus can also adversely affect the course of pregnancy.

Cervical Insufficiency

Cervical insufficiency typically presents with vaginal bleeding, pelvic pressure, or ruptured membranes in the early second trimester. On examination, the cervix is effaced and dilated. Fetal parts or bulging membranes may be detected through the cervix. Risk factors for cervical insufficiency are listed in **Table 22-11**.

Table 22-11	Risk Factors for Cervical Insufficiency
Congenital Factors	
Congenital anomalies	
Uterine anatomic anomalies	
Obstetric History Factors	
History of fetal loss at 14 weeks' gestation or more	
History of cervical laceration following vaginal or cesarean birth	
Multiple first- or second-trimester pregnancy terminations	
Gynecologic History Factors	
Mechanical dilation from dilatation and curettage or hysterosalpingogram	
Previous cervical conization with a large amount of tissue removed	

A woman with a history of two or more second- or third-trimester losses is referred to a physician for an evaluation for cervical insufficiency. Depending on her current clinical situation and reproductive history, she may be a candidate for placement of a suture around the cervix, called *cervical cerclage*, that holds the cervix closed.

Historically, the management of a woman with two or more second-trimester losses included a placement of a cerclage between 14 and 18 weeks' gestation without consideration of the woman's cervical condition. The ability to monitor cervical length has provided new management options. Currently, the trend is to monitor the cervical length and perform cerclage only if the cervix becomes shorter than 25 mm before 24 weeks' gestation.[60] This shift in practice is due to recognition that cerclage has significant risks, including infection, rupture of membranes with or without infection, and subsequent pregnancy loss.

If a woman presents with signs or symptoms that suggest possible cervical insufficiency, such as increased discharge, watery discharge, or vaginal bleeding, in the early second trimester, an initial speculum examination will rule out common causes of symptoms such as

vaginitis. Observable cervical dilatation or the presence of visible membranes indicates a need for immediate physician consultation. Bimanual and digital examination will be needed but are best performed by the physician to avoid potentially rupturing membranes and introducing infection.

If the membranes are intact and the cervix is amenable, cervical cerclage may be recommended. Several types of cerclage are used, with the precise technique selected based on the clinical situation, the cervical length and dilation, and the practice experience and preference of the physician. Women who have a cerclage placed may also be offered progesterone therapy. Removal of the cerclage suture is done at the direction of the physician between 36 and 37 weeks' gestation.[61]

Short Cervix

Until a few years ago, identification of a "short cervix" on ultrasound was an incidental finding of unknown importance. Subsequent research has shown there is a strong positive association between cervical length and preterm labor (**Table 22-12**).[62,63] Ultrasound measurement of cervical length is recommended for women who have a history of preterm birth.[64,65] The routine transvaginal ultrasound measurement of cervical length in women who do not have a history of preterm birth is not universally recommended at this time, but is being increasingly adopted in many clinical settings as meta-analyses have demonstrated it to be a cost-effective screening strategy.[64,65]

Table 22-12 Predicted Probability (%) of Preterm Birth by Cervical Length (mm) and Gestational Week at Time of Measurement

Weeks' Gestation	Cervical Length (mm)							
	10	15	20	25	30	35	40	45
16	53.3	45.2	37.3	30.1	23.7	18.3	13.9	10.5
18	50.7	42.6	34.9	27.9	21.8	16.8	12.7	9.6
20	48.1	40.1	32.5	25.8	20.1	15.4	11.6	8.7
22	45.4	37.6	30.3	23.9	18.5	14.1	10.6	7.9
24	42.8	35.1	28.1	22.0	16.9	12.8	9.6	7.5
26	40.5	32.8	26.0	20.3	15.5	11.7	8.7	6.5
28	37.8	30.5	24.0	18.6	14.2	10.6	7.9	5.9

Modified with permission from Berghella V, Roman A, Daskalakis C, Ness A, Baxter JK. Gestational age at cervical length measurement and incidence of preterm birth. *Obstet Gynecol.* 2007;110:311-317.[63]

Cervical length measurement to screen for a risk of preterm labor is performed using transvaginal ultrasound to measure the distance between the internal and external os; determine the shape of the canal, such as "funneling"; and observe for the presence of membranes bulging into the cervical canal. Abdominal ultrasound is not a reliable means of measuring the cervical length because of the difficulty in achieving the appropriate angle as the ultrasound is performed. The confounding factors of maternal habitus and the need to fill the bladder may result in a false lengthening of the measurement.

Cervical length measurement for screening should be performed after 16 weeks' gestation and before 24 weeks' gestation. Prior to 16 weeks' gestation, the lower uterine segment is not well developed and measurement is not reliable. There is insufficient data about the value of interventions such as cerclage when instituted after 24 weeks' gestation.[65] Cervical length measurements may be obtained after 24 weeks' gestation as part of the evaluation for women who have signs and symptoms of preterm labor, which is reviewed in more detail in the *Complications During Labor and Birth chapter.*

The diagnosis of short cervical length is made when the cervix is less than 20 to 30 mm after 16 weeks' gestation and before 24 weeks' gestation.[65] The range of 20 mm to 30 mm that defines "short cervix" differs depending on the population studied and gestational age range of study participants. Women who are found to have a short cervix on ultrasound screening are referred to a physician for consultation and discussion of therapeutic options.

Management of women with a short cervix who have a history of preterm labor differs from

the management of women without a history of preterm labor, as depicted in **Figure 22-7**.[64] In women without a history of preterm birth, the cervical length measurement at which treatment should be instituted to prevent preterm labor has not been conclusively determined. Two major randomized controlled trials (RCTs) examining the impact of vaginal progesterone treatment for short cervix included only women with cervical length less than 15 mm or in the range of 10–20 mm. Based on these studies, the the Society for Maternal-Fetal Medicine and the American College of Obstetricians and Gynecologists recommends that women without a history of preterm birth who have a short cervix be offered treatment with vaginal progesterone if the cervical length is less than 20 mm.[64] If the decision is made to initiate vaginal progesterone for a woman who has a short cervix, this therapy consists of suppositories (200 mg) or gel (90 mg) inserted once daily from 16 weeks through 36 completed weeks of gestation.

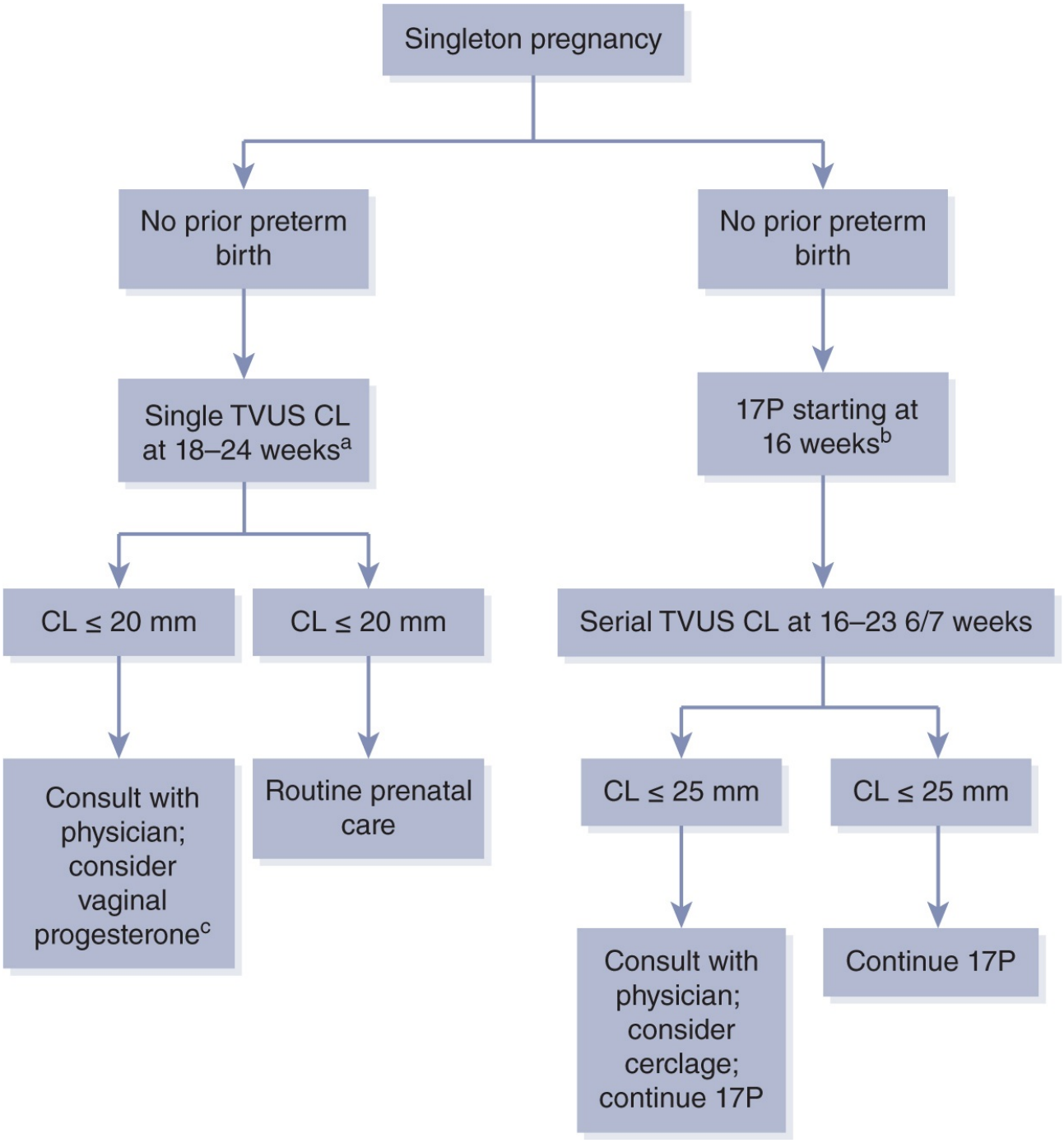


Figure 22-7 Assessment and management of women with a short cervix.

Abbreviations: CL, cervical length; TVUS, transvaginal ultrasound, 17P, 17-hydroxyprogesterone.

[a] If transvaginal ultrasound for screening is performed.

[b] 17P 250 mg intramuscularly given weekly from 16–20 weeks to 36 weeks' gestation.

[c] Daily 200-mg suppository or 90-mg gel from time of diagnosis of short cervical length to 36 weeks' gestation.

Modified with permission from Society for Maternal-Fetal Medicine Publications Committee, Berghella V. Progesterone and preterm birth prevention: Translating clinical trials data into clinical practice. *Am J Obstet Gynecol*. 2012;206(5), 376-

386.[64]

Uterine Anomalies

Uterine anomalies, also known as Müllerian duct anomalies, are congenital variants of the female reproductive tract (as reviewed in the *Gynecologic Disorders* chapter). Uterine anomalies have an estimated prevalence of 0.5% to 5% in the general female population.[66] The most common uterine anomalies are septate uterus, bicornuate uterus and arcuate uterus.

Uterine anomalies are associated with infertility. Once pregnancy is achieved, such anomalies are associated with increased risks of miscarriage, fetal growth restriction, preterm labor/birth, malposition, and placental abruption.[67] Some Müllerian duct anomalies are also associated with the rare but serious risk of uterine rupture in a rudimentary horn. A rudimentary horn is often noncommunicating with the body of the uterus and has an underdeveloped myometrium that limits the horn's ability to expand with a growing pregnancy. It may rupture either catastrophically or silently. Catastrophic rupture has symptoms similar to ruptured tubal pregnancy; silent rupture can lead to a secondary abdominal pregnancy.

Uterine anomalies may be identified for the first time during pregnancy, such as during first-trimester ultrasound, or on clinical examination when the uterine outline is displaced to one side of the abdomen. Depending on the gestational age of the fetus and type of anomaly suspected, 3-D ultrasound or magnetic resonance imaging may be needed to confirm the diagnosis.

Management of a woman who has a uterine anomaly requires consultation with a physician or specialist group. Depending on the anomaly and plan for care, midwives may participate in prenatal care under the guidance of the consulting specialists.

Preterm Birth

Preterm birth is defined as birth that occurs before 37 completed weeks of gestation. Because numbers are not assigned until the end of a completed week, 37 completed weeks of gestation occurs when the fetus is 37 weeks and 0 days gestational age. Approximately 10% of births in the United States occur at a preterm gestational age.[68] Overall, congenital anomalies are the leading cause of infant mortality and preterm birth is the second most common cause.[69] Globally, preterm birth is the leading cause of neonatal deaths, accounting for 35% of all neonatal deaths. Survivors of preterm birth are at risk for health complications that extend into adulthood. Preterm birth is more common in non-Hispanic black women compared to women from all other racial and ethnic backgrounds.[70] This racial disparity remains even after accounting for other risk factors such as smoking, socioeconomic background, or educational level. Although the reasons for the racial disparities in the incidence of preterm birth are not fully known, contributing factors include a mix of environmental, genetic, and social factors.[70] Furthermore, many of these risk factors are interrelated. For example, some of the genetic differences such as changes in telomere length maybe related to environmental influences.

Table 22-13 presents subcategories of preterm gestation based on a combination of the definitions used by the United States Centers for Disease Control and Prevention and the World Health Organization. For those infants who survive preterm birth, short-term adverse outcomes include respiratory distress syndrome, intraventricular hemorrhage, and infection. Long-term adverse outcomes following preterm birth include neurodevelopmental disabilities, altered pulmonary function, increased risk for metabolic and cardiovascular disorders, and decreased long-term survival. Morbidity and mortality rates increase as the gestational age at the time of birth decreases. Infants born in the periviable period have the lowest survival rates and highest rates of long-term morbidities among those who do survive, whereas infants born in the moderate or late preterm gestational age categories have a high chance of surviving without long-term complications.

Table 22-13 Gestational Age Categories and Associated Mortality Rates

Category	Definition
Periviable gestation	20 0/7–25 6/7 completed gestational weeks
Extremely preterm	26 0/7–27 6/7 completed gestational weeks
Very preterm	28–31 6/7 completed gestational weeks
Moderate preterm	32 0/7–33 6/7 completed gestational weeks
Late preterm	34 0/7–36 6/7 completed gestational weeks

This chapter presents an overview of preterm labor, including risk factors, diagnostic criteria, and prenatal care for women who have a history of preterm labor. Some of the initial work-up for preterm labor is often completed in the outpatient setting, but the definitive diagnosis requires uterine monitoring for a period of time and possibly hospitalization. For this reason, the steps involved in a thorough evaluation of a woman with suspected preterm labor

are presented in the *Complications During Labor and Birth* chapter.

Pathophysiology of Preterm Labor

Labor is caused by a release from inhibitory mechanisms that sustain uterine quiescence throughout the pregnancy. At least five discrete mechanisms have been identified that initiate premature uterine activity in pregnant women:[71-77]

- *Premature activation of the maternal or fetal hypothalamic–pituitary–adrenal (HPA) axis.* Physical or psychological stress activates the maternal HPA axis and leads to subsequent release of high levels of corticotropin-releasing hormone (CRH), which in turn increases prostaglandin production and directly stimulates the uterus. Preterm activation of the maternal HPA axis can be caused by maternal stress (depression, post-traumatic stress disorder [PTSD], anxiety) or physical stress.[71,72] Premature activation of the fetal HPA axis can occur secondary to genetic factors, an inhospitable uterine environment, or inflammation (e.g., intra-amniotic infection).[73]
- *Inflammation/infection.* Microorganisms in the genital tract may cause inflammation at the choriodecidual interface, which in turn stimulates contractions.[73]
- *Decidual hemorrhage (placenta abruption or subchorionic hemorrhage).* Localized uteroplacental ischemia can cause necrosis of the decidua. This necrosis is a type of oxidative stress that releases thrombin. Thrombin, in turn, stimulates uterine contractility.[74]
- *Overdistension of the uterus (e.g., polyhydramnios, multifetal gestation).* Overstretching of the myometrium induces formation of gap junctions and upregulation of oxytocin receptors.
- *Genetics.* Genetic factors have long been suspected as an etiology in preterm birth, but that relationship has only recently been confirmed. Four genetic loci have been identified as being associated with length of gestation, and variants in three of those loci are associated with preterm birth: *EBF1*, *EEFSEC*, and *AGTR2*.[75,76]

Risk Factors for Preterm Labor

Known risk factors are listed in **Table 22-14**.[77-86] However, approximately 50% of women who give birth prematurely do not have an identified risk factor for preterm birth,[77] so with a few exceptions, risk factors do not reliably predict preterm labor. The most significant risk factor for preterm birth is a history of preterm birth: Women with such a history are significantly more likely to experience a subsequent preterm birth.[78,84]

Table 22-14 Risk Factors for Preterm Labor

Maternal Demographic and Lifestyle Factors	Fetal Factors
Age < 17 years or > 35 years	Congenital anomalies
African American ethnicity	Fetal growth restriction

Genetic variants	Infection
Low prepregnancy maternal body weight (BMI < 19.8)	Isoimmunization with hydrops
Low socioeconomic status	
Short interpregnancy interval (< 18 months)	
Stress (depression, anxiety, post-traumatic stress disorder)	
Smoking during pregnancy	
Substance abuse (cocaine, crack, heroin, tobacco)	
Maternal Medical and Reproductive History	**Current Pregnancy Factors**
Previous preterm birth	Cervical insufficiency/short cervix
Pregnant woman born preterm	Infections during pregnancy
Prior cervical surgery (cone, dilatation and curettage)	Intrauterine infection
	Hydramnios (polyhydramnios)
Müllerian tract abnormalities	Multifetal gestation
Short cervical length measured on transvaginal ultrasound	Maternal disorders (e.g., pregnancy-induced hypertension/HELLP syndrome, placenta previa)
	Pyelonephritis
	Vaginal bleeding during > one trimester
Factors with Conflicting Evidence Regarding Risk for Preterm Birth	**Preventable Iatrogenic Factors**
Asymptomatic bacteriuria, lower UTIs	Unknown gestational age
Genital tract infections	Elective induction of labor before term
Periodontal disease	
Vaginal bleeding	

Abbreviations: BMI, body mass index; HELLP, hemolysis, elevated liver enzymes, and low platelet count; UTIs, urinary tract infections.

Based on Adams MM, Elam-Evans LD, Wilson HG, Gilbertz DA. Rates and factors associated with recurrence of preterm delivery. *JAMA.* 2000;283:1591-1996[78]; Mercer BM, Goldenberg RL, Das A, et al. The Preterm Prediction Study: a clinical risk assessment system. *Am J Obstet Gynecol.* 1996;174:1885-1893[79]; Muglia LJ. The enigma of spontaneous preterm birth. *N Engl J Med.* 2010;362:529-535[80]; Dekker GA, Lee SY, North RA, McCowan LM, Simpson NA, Roberts CT. Risk factors for a preterm birth in an international prospective cohort of nulliparous women. *PLoS One.* 2012;7(7);e39154[81]; Ward K, Argyle VA, Meade M, Nelson L. The heritability of preterm delivery. *Obstet Gynecol*. 2005;106(6):1235-1239[82]; Manns-James L. Bacterial vaginosis and preterm birth. *J Midwifery Womens Health.* 2011;56(6):575-583.[83]

Evaluation and Diagnosis of Preterm Labor

The choice of site for an evaluation of a woman who might be in preterm labor will depend on the institution, role of the midwife in that setting, severity of the

woman's symptoms, and gestational age of the fetus. The adverse outcomes of preterm birth can be profound, so rapid referral to physician care is indicated for a woman whose fetus is at an early gestation or a woman with significant symptoms. In all settings, the primary role of the midwife is to recognize significant risks for preterm labor and signs of preterm labor.

The diagnosis of preterm labor can be challenging. Before preterm labor is well established and clearly active, it can be difficult to distinguish normal pregnancy symptoms from early preterm labor. Indeed, many early symptoms of preterm labor are quite common during pregnancy. Most women with symptoms of preterm labor do not go on to have a preterm birth,[87,88] and over-intervention is a concern. Delays in treatment, however, have the potential to adversely affect outcomes. Symptoms of preterm labor, listed in **Table 22-15**, are not predictive of ongoing preterm labor. For example, contraction frequency is a poor predictor of subsequent preterm birth, although the risk of preterm birth does increase with frequency of contractions.[87] For this reason, it is important to evaluate a woman with any symptoms of preterm labor carefully by reviewing her history for risks for preterm labor, asking her about any additional symptoms from the list in **Table 22-15**, and observing her over time if needed.

Table 22-15	Signs and Symptoms of Preterm Labor[a]
Symptoms	**Signs**
Abdominal tightness or “menstrual-like cramps”	Short cervix
Diarrhea	Change in cervical effacement or dilation
Fetus dropping low into the pelvis before 36 weeks	Positive fetal fibronectin test
Increased vaginal discharge (clear, pink, or slightly bloody mucus)	Ruptured membranes
Lower back pain	
Pelvic pressure	
Vaginal bleeding	
Uterine contractions that are increasing in frequency, duration and intensity	

[a] Individual clinical signs and symptoms of preterm labor are not highly predictive.

Diagnostic criteria for active preterm labor include regular uterine contractions plus a change in cervical effacement and/or cervical dilation. Rupture of membranes or vaginal bleeding increases the diagnostic certainty. Some institutions use specific criteria such as regular painful contractions plus cervical dilation of 3 centimeters or more, or cervical length less than 20 millimeters, or cervical length between 20 and 30 millimeters with a positive test for fetal fibronectin. The March of Dimes recommends six or more contractions per hour as the threshold prompting evaluation for preterm labor.[89] If active preterm labor is suspected, in-patient obstetric triage/emergency department assessment is warranted.

Prevention of Preterm Birth

Urinary tract infections, periodontal disease, and several urogenital infections—group B *Streptococcus* (GBS), *Chlamydia trachomatis*, bacterial vaginosis, *Neisseria gonorrhea*, syphilis, and *Trichomonas vaginalis*—are associated with preterm labor. Even so, a causal relationship between these infections and preterm labor has not been established. Furthermore, treatment is not associated with a lowered incidence of preterm labor,[83,90-95] and a few studies have found an increase in preterm labor associated with treatment of bacterial vaginosis or *T.*

vaginalis infection with metronidazole (Flagyl).[83] Although asymptomatic bacteriuria is not associated with preterm birth per se, treatment of asymptomatic bacteriuria is recommended to decrease the incidence of pyelonephritis—a condition that is associated with preterm birth. Women with human immunodeficiency virus (HIV) should be screened and treated for *T. vaginalis* infection to reduce the risk of vertical transmission of HIV. All sexually transmitted infections should be treated per guidelines reviewed in the *Reproductive Tract and Sexually Transmitted Infections* chapter. Finally, women with symptomatic infections and women with periodontal disease should be treated per standard protocols for general health promotion.

For women who have a history of preterm birth use of progesterone therapy (17-alpha hydroxyprogesterone caproate [17-OHPC]) to prevent preterm labor has demonstrated effectiveness in multiple RCTs and subsequent systematic reviews.[90] Although the mechanism of action is not well understood, progesterone is thought to have an anti-inflammatory effect that curtails the inflammatory process associated with preterm labor. Another theory suggests that exogenous progesterone increases the level of progesterone in maternal tissue and prevents the drop in progesterone level associated with the onset of labor.

The safety of progesterone for the developing fetus and child has been studied in children exposed to progesterone in utero. The studies conducted to date indicate that children 2 to 4 years old have no difference in health status or cognitive skills when compared to a similar cohort of children who were not exposed to this drug in utero.[91],[92] Longer-term follow-up is needed before safety can be fully assured. Nonetheless, the balance of unknown long-term risks to the child versus benefits of avoiding preterm birth is such that progesterone is recommended for selected women at risk for preterm labor.

The dose of 17-OHPC is 250 mg, administered via weekly injections from 16 to 36 weeks' gestation. The injections are continued until 36 completed weeks of pregnancy. 17-OHPC is not effective for prevention of preterm birth in women with a multifetal gestation. Likewise, it is not effective for women who have current preterm rupture of membranes or as a tocolytic for women in active preterm labor. This agent has not been shown to prolong gestation in women with a cerclage who did not have a prior preterm birth. Women with short cervix alone and no history of preterm birth may benefit from vaginal progesterone, but no value has shown for injected 17-OHPC in this group. Similarly, for women who had a prior preterm birth and are receiving 17-OHPC for prevention of preterm labor in the current pregnancy, no benefit has been found if vaginal progesterone is added to treat the additional finding of a short cervix.

Premature Prelabor Rupture of Membranes

Spontaneous rupture of membranes prior to the onset of labor is termed prelabor rupture of membranes (PROM). If the rupture occurs before 37 weeks' gestation, it is called *premature prelabor rupture of membranes* (PPROM). In the United States, PPROM occurs in approximately 3% of all pregnant women.[96] Risk factors for PPROM are the same as the risk factors for preterm labor. Infection can be both a cause of PPROM and a consequence of PPROM. Suspected PPROM is an indication for physician consultation and referral.

Ruptured membranes may present as a "gush" that the woman notices or as "a trickle" or constant wetness. In rare cases, a small tear may seal over during the course of a few days or weeks. In that situation, pregnancy is expected to progress normally, although careful monitoring for any additional significant fluid loss is needed. A woman who reports possible PPROM is best evaluated in a labor and delivery inpatient setting. The procedure for assessment and diagnosis is reviewed in the *First Stage of Labor* chapter.

Post-Term Pregnancy

A post-term pregnancy is one that has reached or continues beyond 42 0/7 weeks (294 days) of gestation, which is 42 completed weeks.[97] Historically, term pregnancy was defined as a gestation of 37 0/7 weeks to 41 6/7 weeks. In an effort to address the variation in perinatal outcomes in the period between 37 0/7 and 41 6/7 weeks, a 2012 work group recommended the following terminology for term pregnancy:[98]

- Early term: 37 0/7 weeks through 38 6/7 weeks
- Full term: 39 0/7 weeks through 40 6/7 weeks
- Late term: 41 0/7 weeks through 41 6/7 weeks
- Post term: 42 0/7 weeks or more

Management of late term and post-term pregnancy is reviewed here. The term "postdates" is imprecise and should be avoided when discussing late term or post-term pregnancy management.

In the United States, births during the 41st week of gestation account for 6.5% of all births; births at 42 weeks of gestation and beyond account for 0.4%.[68] Beyond an error in the dating, the causes of a truly prolonged pregnancy are unknown. In many cases, maternal or fetal genetic influences on initiation of labor may play a role. The incidence is higher among women who are older, who are nulliparous, and who have a history of a prior post-term birth.[99] Caucasian women are more likely to have a post-term pregnancy than are women of other racial/ethnic groups. Rare causes of prolonged pregnancy include placental sulfatase deficiency, fetal adrenal insufficiency, and fetal anencephaly.[99]

Pregnancy at 41 weeks' gestation and greater is associated with increased incidence of fetal and maternal adverse outcomes, most of which are the result of excessive fetal growth or placental insufficiency. Maternal risks include severe perineal lacerations, infection, postpartum hemorrhage, operative vaginal birth, and cesarean birth.[99] Study results vary

somewhat regarding specific risks, but include an increased fetal risk of oligohydramnios, meconium aspiration syndrome, macrosomia, shoulder dystocia, fetal dysmaturity syndrome, and perinatal mortality.[100]

The term *fetal dysmaturity syndrome* is used to describe a fetus/newborn exposed to chronic malnutrition. Such a fetus is small for gestational age, with little body fat, and is at increased risk for oligohydramnios, umbilical cord compression, and abnormal fetal heart rate patterns. As a newborn, the fetus is also at greater risk for hypoglycemia, polycythemia, meconium aspiration, and perinatal asphyxia. There is a clear increased risk of perinatal mortality (which includes stillbirth and early neonatal death) for women with a late term and post-term gestation.[101] A large retrospective study involving more than 3 million women found that the risk of stillbirth was 4.2 cases per 10,000 women at 40–41 weeks, 6.1 cases per 10,000 women at 41–42 weeks, and 10.8 cases per 10,000 women at 42–43 weeks.[102]

Women without medical or obstetric complications who are in the late term gestational period are managed expectantly with watchful waiting. At 41 0/7 weeks, the woman is offered expectant management with fetal surveillance twice weekly (antepartum testing) or induction of labor. After 42 0/7 weeks and by 42 6/7 weeks, the recommendation is to induce labor.[98]

During the week between 41 0/7 and 42 0/7 gestational weeks, induction is associated with a lower risk of perinatal death (relative risk [RR], 0.31; 95% confidence interval [CI], 0.12–0.88), cesarean delivery (RR, 0.89; 95% CI, 0.81–0.97), and meconium aspiration syndrome (RR, 0.50; 95% CI, 0.34–0.73).[103] However, the perinatal death rate is low and a high number of inductions (n = 410) are needed to avoid one perinatal death. There are no significant differences in birth asphyxia or neonatal intensive care unit admissions when induction is compared with expectant management for women in this gestational age range.[103] Thus, the choice of management during this period of time should follow a process of shared decision making that includes assessment of biologic factors such as gravidity, cervical status, and fetal well-being, and the woman's preferences.

Membrane Sweeping

Membrane sweeping is a common intervention performed by both midwives and physicians in the outpatient setting at term in pregnancy to try to hasten the onset of labor. Evidence shows a reduced rate of pregnancy continuing beyond 41 weeks when membrane sweeping is done, with no increase in adverse outcomes.[104] Most women find this procedure uncomfortable but say they would choose to do it again in a subsequent pregnancy. This elective procedure is done only after the woman is informed of the risks and benefits and gives her consent for the procedure. It should not be offered or performed prior to 40 weeks' gestation. Membrane sweeping, historically called stripping of the membranes, involves gently dislodging the membranes from where they attach to or lay over the cervix; this process releases prostaglandins, which aid in the preparation and initiation of labor. Numerous studies have demonstrated that membrane sweeping increases the likelihood of spontaneous labor in conjunction with oxytocin.[104] Evidence has also found membrane sweeping to be safe with no increased risks of fetal or maternal infection.[104] Women may experience discomfort during the examination, and they may experience slight vaginal bleeding and irregular contractions after membrane sweeping. Accidental rupture of the membranes could also occur but is rare.

The technique for performing membrane sweeping is as follows: The examining fingers of the practitioner's sterile gloved hand are introduced into the vagina, enter the cervical os, and swing circumferentially around the internal os, separating the membranes from the uterine wall. If the cervical os is closed and membrane sweeping cannot be performed, some clinicians recommend massage of the cervix at the vaginal fornices, as this intervention may cause release of prostaglandins. Membrane sweeping can be repeated in 36 to 48 hours.

Dermatoses of Pregnancy

Pruritus is a common problem for pregnant women. Skin conditions related to or caused by the unique physiologic aspects of pregnancy are classified into four categories: (1) atopic eruption of pregnancy, (2) intrahepatic cholestasis of pregnancy, (3) pemphigoid gestationis, and (4) polymorphic eruption of pregnancy.[105,106] Because both intrahepatic cholestasis and pemphigoid gestationis are associated with serious adverse fetal outcomes, early recognition and differentiation is important to minimize potential fetal risks and provide adequate relief for the woman. These four dermatoses can be differentiated by appearance, location on the woman's body, and time of onset during pregnancy (**Table 22-16**).[105-109]

Table 22-16 **Dermatoses in Pregnancy**

Characteristics	Atopic Eruption of Pregnancy	Intrahepatic Cholestasis of Pregnancy	Pemphigoid Gestationis	Polymorphic Eruption of Pregnancy	Pustular Psoriasis of Pregnancy
Nomenclature: Historical term	Eczema in pregnancy[a] Pruritic folliculitis of pregnancy[a] Papular dermatitis of pregnancy	Obstetric cholestasis Icterus gravidarum Pruritus gravidarum	Herpes gestationis	PUPPP[a] Erythema multiforme of pregnancy	Impetigo herpetiformis
Associated conditions		Hepatitis C	Multiparity, hydatidiform mole (rare)		Rare variant of psoriasis triggered by pregnancy
Trimester(s) of onset	First and second	Late second or third	Second and third	Third and postpartum	Third
Symptoms	Eczematous rash	Pruritus without rash, jaundice	Urticarial papules, plaques, bullae	Urticarial papules, plaques	Symmetric erythematous plaques with sterile pustules. Enlarge from the periphery as center becomes crusted over. Systemic symptoms but pruritus is often absent
Location	Entire body, including palms/soles	Entire body; may be most intense on palms/soles	Begins on abdomen, then body; spares the face	Abdominal striae, then body; spares the face, palms, soles, and periumbilical area	Begins in flexural areas and spreads centrifugally
Diagnosis	Clinical symptoms and appearance	Clinical symptoms and elevated bile acids	Skin biopsy, ELISA	Clinical symptoms and appearance	Clinical symptoms and appearance
Maternal risks	No known risks	Cholelithiasis, steatorrhea, hemorrhage	Increased risk for Graves' disease; recurrence in future pregnancy	No known risks	None
Fetal risks	No known risks	Meconium, preterm birth, stillbirth	Preterm birth, low birthweight, IUGR, neonatal lesions	No known risks	Placental insufficiency, miscarriage, fetal growth restriction, stillbirth
Management	Topical corticosteroids	Ursodeoxycholic acid, possible induction; screen for hepatitis C; consider delivery at 37 weeks or later Antenatal fetal testing, fetal movement counts	Topical and systemic corticosteroids, antihistamines Antenatal fetal testing, fetal movement counts	Topical corticosteroids, antihistamines, emollients	Systemic corticosteroids
Resolution after birth	Soon after	2–3 weeks	Weeks or months	7–10 days	Soon after birth
Recurrence in future pregnancies	Possible	Possible	Frequent (50–70%)	Low	Not known

Abbreviations: ELISA, enzyme-linked immunosorbent assay; IUGR, intrauterine growth restriction; PUPPP, pruritic urticarial papules and plaques of pregnancy

[a] Acceptable term for this condition.

Atopic Eruption of Pregnancy

Atopic eruption of pregnancy is a benign dermatosis of pregnancy characterized by intense itching. This group of dermatoses includes eczema, prurigo of pregnancy, and pruritic folliculitis of pregnancy. It is considered to be the most common skin disorder during pregnancy, accounting for almost 50% of all rashes experienced by women when pregnant.[105]

The presentation of atopic eruption of pregnancy includes intense pruritus with papular, eczematous lesions primarily appearing in the first or second trimester. The rash usually involves the face, neck, and antecubital and popliteal fossae, but can occur on all parts of the body, including the palms and soles. Approximately one third of women develop the rash on the trunk and the abdomen.[105,107]

This condition resolves early in the postpartum period, but recurrence in subsequent pregnancies is possible.[107] There are no known adverse effects of atopic eruption of pregnancy on maternal or fetal outcomes.[105] Treatment involves topical corticosteroids for maternal relief. Moisturizers and lukewarm showers with mild soap can also provide symptom relief.[107]

Intrahepatic Cholestasis of Pregnancy

Intrahepatic cholestasis of pregnancy is a hepatic condition specific to pregnancy that features intense itching without rash, elevated serum bile acids, and increased liver enzymes.[105,108] Slowed emptying of bile from the gallbladder underlies the development of this condition. Multifetal gestation, a personal history of hepatitis C, cholelithiasis, and dyslipidemia are associated with increased risk for this disorder.[105] Environmental, dietary, genetic, and ethnic influences also contribute to the relative risk of developing intrahepatic cholestasis of pregnancy. There is wide variation in the incidence of this dermatosis, with rates ranging from 2% or less in the United States and Europe to as high as 28% in Chile.[105] The reoccurrence rate of intrahepatic cholestasis of pregnancy in subsequent pregnancies is approximately 60%.[105,108]

Symptoms of intrahepatic cholestasis of pregnancy develop in the second and third trimesters, although in as many as 10% of women, the symptoms manifest in the first trimester.[105,108] Intense itching, without rash, occurs all over the body; it may be most severe on the palms and soles, and intensifies at night. In addition, some women have diarrhea or steatorrhea.

Fetal risks include increased incidence of meconium-stained fluid (20–30%), preterm birth (20–60%), and stillbirth (1–2%).[105,108] Stillbirth occurs suddenly among women with intrahepatic cholestasis of pregnancy. The underlying etiology appears to be related to sudden vasospasm of placental veins secondary to exposure to toxic bile acids that the immature liver cannot clear.[105,108]

The differential diagnoses include the hemolysis, elevated liver enzymes, and low platelet count syndrome (HELLP), preeclampsia, hepatitis, choledocholithiasis, and other liver disorders. The diagnosis is based on the presence of pruritus without rash and laboratory tests that indicate liver dysfunction in the absence of any other dermatologic diagnosis. Jaundice develops in 10% to 25% of women with intrahepatic cholestasis of pregnancy.

Aminotransferase levels rise dramatically, conjugated bilirubin is increased, and bile acids become elevated to more than 10 mmol/L.[105,108] Elevated bile acids are the most accurate biomarker of intrahepatic cholestasis.[105,108] Nevertheless, bile acids are not markedly elevated in all women with this disorder, perhaps because bile acid elevation is a later sign in the natural course of the disorder. Because the risk of stillbirth is high, severe pruritus without rash is sufficient to make a presumptive diagnosis and start treatment, before laboratory values are available.

Maternal complications of intrahepatic cholestasis of pregnancy are not common. Theoretically a woman with intrahepatic cholestasis can develop vitamin K deficiency secondary to fat malabsorption when bile acids do not function normally. Some, but not all, physicians who care for these women recommend supplemental vitamin K. The risk of postpartum hemorrhage does not appear to be higher, and coagulation studies are not usually indicated but may be considered in individual circumstances.

Physician consultation and antenatal fetal testing should be initiated promptly. Maternal treatment with ursodeoxycholic acid (Ursodiol, Actigall) is the most effective treatment. The usual dose is 10 to 15 mg/kg body weight daily. Additional treatments include cholestyramine (Questran), a bile acid sequestrant; hydroxyzine (Vistaril) to treat pruritus; and occasionally dexamethasone (Decadron). Antepartum fetal surveillance will be initiated but its value is uncertain, as stillbirth happens rapidly and without warning in women with this disorder. Induction of labor is planned at approximately 36 weeks' gestation to prevent stillbirth.

Pemphigoid Gestationis

Pemphigoid gestationis is a rare autoimmune disorder that is most common among Caucasians and occurs in approximately 1 in 50,000 pregnancies. Due to the similar appearance of the lesions, pemphigoid gestationis was originally called herpes gestationis; in reality, it is not related to the herpes viruses. This dermatosis is most common in multigravidas in the third trimester and often occurs in subsequent pregnancies. However, presentation in the first trimester is possible.[105,107]

A prodrome of malaise, fever, nausea, and headache may precede the skin eruptions. Pruritus begins initially with urticarial and annular plaques that make it difficult to distinguish pemphigoid gestationis. The initial symptoms are followed by vesicobullous eruptions that begin on the abdomen and spread to the extremities, sparing the face. Enzyme-linked immunosorbent assay (ELISA) is an effective method for diagnosing pemphigoid gestationis. This technology's high sensitivity and specificity allow for differentiation between pemphigoid gestationis and polymorphic eruption in pregnancy.[105] Skin biopsy is also used for diagnosis.

Pemphigoid gestationis has been linked to several adverse effects in the fetus. Low birth weight and prematurity are associated with earlier onset of this dermatosis and the formation of blisters.[107] In addition, there is an estimated 5% to 10% maternal–fetal transfer rate.

Maternal risks include a long-term increase in the risk of Graves' disease. Remission may occur in the latter part of the third trimester. Pemphigoid gestationis may flare in the postpartum period, then resolve without causing scars. It recurs in most, but not all, subsequent pregnancies and tends to worsen with recurrence.

Treatment in the early stages of pemphigoid gestationis includes potent topical steroids, topical or oral antihistamines, and oral prednisolone. If pemphigoid gestationis progresses to the bullous stage, systemic corticosteroid therapy is required. Antenatal fetal testing is initiated in the third trimester, including weekly biophysical profiles, as well as Doppler studies if placental insufficiency is suspected. Infants born with the disease do not require treatment and the condition typically resolves spontaneously.[105]

Polymorphic Eruption in Pregnancy

Polymorphic eruption in pregnancy (PEP) is also known as pruritic urticarial papules and plaques of pregnancy (PUPPP). Most common in primigravid women, this condition typically occurs in the third trimester. Excessive stretching of abdominal skin causing tissue damage and high levels of estrogen and progesterone may be related to the development and severity of the outbreaks. The rash resolves spontaneously approximately 4 to 6 weeks after its onset. Recurrence in subsequent pregnancies is rare.[105,109] The red, painfully itchy urticarial papules initially present in the striae, but then spread to cover the trunk and extremities. The abdomen and proximal thighs are the most common sites of involvement. The face, palms and soles, and periumbilical area are not involved.[105,109] Except for the discomfort, polymorphic eruption in pregnancy is a benign condition with no adverse effects on the mother or fetus. Low-potency topical steroids and antihistamines can be used to treat the discomfort. Severe cases may require systemic corticosteroids.

Peripartum Cardiomyopathy

Peripartum cardiomyopathy is a rare form of heart failure that develops in the last month of pregnancy or the first 5 months postpartum in the absence of heart disease or another identifiable cause for failure. The majority of women who develop peripartum cardiomyopathy do so in the postpartum period; thus, this disorder is reviewed in more detail in the *Postpartum Complications* chapter.

Risk factors for peripartum cardiomyopathy include African American race, obesity, maternal age older than 30 years, multiparity, and chronic hypertension/preeclampsia. The disparity in the prevalence of this condition is dramatic: Compared to white women, African American women have a 4- to 6-fold increase in the incidence and mortality of peripartum cardiomyopathy.[110] The reasons for the disparity are unclear, but may reflect an increased rate of underlying cardiovascular disease, known risk factors, and less access to care.

Signs and symptoms of peripartum cardiomyopathy may be inaccurately attributed to normal physiologic changes in pregnancy and postpartum and women with this diagnosis frequently report being given an incorrect diagnosis initially.[111] The role of the midwife is to maintain a high index of suspicion. Signs and symptoms of peripartum cardiomyopathy include all the signs of pulmonary edema: shortness of breath that is particularly noticeable when the woman reclines, cough, tachycardia, palpitations, and chest pain. Women with symptoms should be seen immediately, and a complete history and physical examination performed. Care of women with peripartum cardiomyopathy requires a multidisciplinary team of cardiologists, intensive care physicians, maternal–fetal medicine specialists, and respiratory specialists.

Red Cell Alloimmunization

Alloimmunization (also referred to as isoimmunization) is the process by which a woman develops antibodies against human tissue antigens not present in her own body. Events that can lead to isoimmunization include pregnancy and birth, wherein the woman is exposed to fetal red blood cells, blood transfusion, and tissue/organ transplant. The topic of isoimmunization is introduced in the *Prenatal Care* chapter and interventions to prevent Rh (D) sensitization are addressed there.

In addition to the Rh (D) antigen, red cell isoimmunization can develop secondary to other red cell membrane proteins. Red cell isoimmunization affects approximately 1.5% to 2.5% of women during pregnancy, although only a small proportion of fetuses and newborns of women with alloantibodies to non-Rh (D) red blood cell antigens will develop hemolytic disease of the fetus and newborn (HDFN).[112] Although Rh (D) alloimmunization is no longer the most common antibody identified among pregnant women, Rh (D) remains the most common cause of isoimmunization-associated pregnancy complications.[112]

The pathophysiology of red cell alloimmunization in pregnancy involves maternal exposure to the foreign antigen secondary to exposure to the fetal red blood cells, development of antibodies to that antigen, and finally transport of those antibodies across the placenta to the fetus, where they attack and lyse fetal red blood cells that carry the specific antigen.[113,114] The resulting hemolytic disease of the fetus and newborn can lead to mild to severe fetal anemia, depending on the type of antibody–antigen complex causing disease and on the level of immune response a woman mounts to the antigen in question. If severe fetal anemia develops, a fetus may also develop hydrops fetalis, a condition associated with hepatosplenomegaly, third-space fluid in the abdomen, and heart failure. Newborns who survive may have anemia and hyperbilirubinemia. The common course of this process occurs over two subsequent pregnancies. First, a woman is exposed during one birth to a fetus with Rh (D) antigen as fetal cells enter the maternal blood stream at the time of birth and placental separation. The woman develops antibodies, and in a subsequent pregnancy with an Rh (D) positive fetus, those antibodies cross the placenta to attack fetal red blood cells.

In addition to the Rh (D) antigen, the non-Rh (D) antigens most likely to cause hemolytic disease of the fetus and newborn are Rh G(c), Rh (E), Kell, and Duffy (**Table 22-17**).[112,114] The Lewis antigen is common but not associated with hemolytic disease of the fetus and newborn because it is an immunoglobulin M (IgM) antibody and, therefore, too large to cross the placenta.

Table 22-17 **Red Cell Antibodies and Relationship to Hemolytic Disease of the Fetus and Newborn**

Red Cell Antigen	Association with Hemolytic Disease of the Fetus and Newborn
Diego D1^{a}, Di^{b}	Mild to severe
Duffy Fy^{a}	Mild to severe
Duffy Fy^{b}	Not associated with HDFN
Duffy By[3]	Mild to severe HDFN

Kell K	Mild to severe HDFN
Kell k, Ko, Kp^a, Kp^b, Js^a, Js^b	Mild HDFN
Kidd Jk^a	Mild to severe HDFN
Kidd Jk^b, Jk^3	Mild HDFN
Lewis	No association with HDFN
Lutheran Lu^a, Lu^b	Mild HDFN
MSS Mt^a	Moderate HDFN
MSS Vw, Mur, Hil, Hut	Mild HDFN
Rh (D)	Mild to severe HDFN
Rh (non-D), E, C	

Abbreviation: HDFN, hemolytic disease of the fetus and newborn.

Note: This list is not an exhaustive list of atypical antibodies that can cause HDFN. Women with any atypical antibody is referred for physician consultation and management.

Based on American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 75: management of alloimmunization during pregnancy. *Obstet Gynecol.* 2006;108:457[112]; De Haas M, Thurik FF, Koelewijn JM, van der School CE. Haemolytic disease of the fetus and newborn. *VOX Sanguinis*. 2015;109:99-103.[114]

Alloimmunization is diagnosed via a maternal antibody screen (indirect Coombs test) that is one of the routine tests offered to all pregnant women. Results from red blood cell (RBC) antibody screens are reported as positive or negative. A positive antibody screen identifies the antibody and if it is one that is associated with alloimmunization, titers are performed that measure how much antibody-positive maternal serum is required to agglutinate a standardized sample of antigen-positive RBCs. Titers are reported as dilutions; that is, 1:1 means one full quantity of maternal serum agglutinates a sample of antigen-positive RBCs, whereas 1:16 means 1/16 the standard quantity of maternal serum agglutinates a sample of antigen-positive RBCs. A critical titer level depends on the risk for hemolytic disease of the fetus and newborn. Titer levels associated with onset of pregnancy complications vary between 1:8 and 1:32, depending on the antibody–antigen complex in question.[114]

Management of isoimmunization begins with identification of women who are alloimmunized. Women who have a positive antibody screen are referred for additional testing and if the resultant antibody and titer are associated with alloimmunization, physician consultation is obtained. Depending on the titer, the antibody titer maybe repeated every 4 weeks until 28 weeks, at which time screening frequency increases to every 2 weeks.[113-115] If a woman's titers reach critical levels for the antibody in question [1:32 for Rh (D), 1:8 for Kell], Doppler studies are used to screen for evidence of fetal anemia. Follow-up monitoring may involve serial amniocentesis to monitor bilirubin levels in the amniotic fluid. In addition, ultrasound monitoring for hydrops fetalis may be initiated.

Population-Specific Special Considerations During Pregnancy

Adolescent Pregnancy and Advanced Maternal Age

Pregnancy at both ends of the age spectrum, younger and older, is associated with increased risks of adverse outcomes. The age range for adolescent or teen pregnancy is defined as 13 to 19 years. Advanced maternal age (AMA) describes women who will be 35 years or older at the time of birth. The incidence of pregnancy in young women age 15 to 19 years is approximately 22 pregnancies per 1000 women in the United States; although this incidence is decreasing, the United States still has one of the highest rates of adolescent pregnancy among all developed nations.[116] By comparison, approximately 9% of pregnant women in the United States are 35 years or older when they give birth.[117]

Adolescent Pregnancy

Adolescent pregnancy is associated with increased rates of adverse maternal and fetal health outcomes, more social risks, and living in poverty. Approximately 80% of adolescent pregnancies are unplanned.[118] Worldwide, complications related to pregnancy and birth are the second leading cause of death in women between ages 15 and 19 years.[118] Studies that evaluate the effects of pregnancy during adolescence often subcategorize this population as younger (less than or equal to age 15 years) or older (age 16 to 19 years) because they have different profiles and different risks. The prevalence of adolescent pregnancy and risks associated with adolescent pregnancy are also highly related to race/ethnicity, geographic setting, family support, financial status, and educational attainment. Likewise, the social perceptions of early parenting differ greatly among different cultures. It is important to view the individual within the framework of her environment and support structure.

Overall, adolescent pregnancy is associated with increased risks for anemia, preterm birth, low birth weight, preeclampsia, postpartum hemorrhage, stillbirth, sexually transmitted infections, and substance use disorder.[118,119] Younger women are also less likely to receive adequate prenatal care as compared to older women.

Prenatal care for adolescents requires increased awareness and screening for social risks such as tobacco use, substance use, intimate-partner violence, food insecurity, and financial instability. In addition, adolescents are minors and subject to different regulations with regard to mandatory parental involvement.[120] Younger women often need detailed health education regarding nutrition, sexuality, and preparing for labor, birth, and parenting.[121] The skills needed to care for adolescents during pregnancy are within the scope of practice for independent midwifery care. Midwives have a long history of providing care to adolescents, and in many settings, they lead teen clinics. In many settings, an interdisciplinary team can provide the best care for this population. The benefits of the CenteringPregnancy group prenatal care model is most promising for adolescents.[122] The Resources section at the end of this chapter includes reviews and guidelines that address the specific prenatal needs of

adolescents.

Advanced Maternal Age

Women who are older than 35 years when they give birth have increased risks for miscarriage, aneuploidy, gestational diabetes, hypertensive disorders of pregnancy, placenta previa, preterm labor/birth, and stillbirth. There is also an age-related increase in maternal morbidity/mortality once pregnancy complications arise, particularly regarding the severity of cardiovascular complications.[123]

The association between advanced maternal age and these conditions is widely recognized, but little evidence exists regarding their causal mechanisms. A key question emerging from the available data is how much of the association between advanced maternal age and pregnancy complications can be accounted for by parity, comorbid conditions, use of assisted reproductive technology, and/or socioeconomic status, all of which commonly change as a woman ages. Another question is whether obstetric risks apply to all women older than 35 years, or whether these risks increase at specific age increments, as is seen in younger versus older adolescents. Screening for conditions associated with advanced maternal age should occur according to general guidelines, and management of each condition should be guided by the details of the woman's condition rather than by her age.

Pregnancy After Assisted Reproductive Technology

There is a positive association between assisted reproductive technology and certain pregnancy complications, including multifetal gestation, miscarriage, ectopic pregnancy, congenital anomalies, preterm birth, low birth weight, preeclampsia, placenta previa, placental abruption, and cesarean birth.[124,125] The increased incidence of multifetal gestation is related to the procedure, whereas some of the identified complications are related to multifetal gestation. Other pregnancy-related complications are more likely in women with singleton pregnancies. It is not clear whether the process of assisted reproductive technology or the underlying infertility is the etiologic factor that explains these associations.

There are no specific guidelines for prenatal care in the absence of signs or symptoms of a complication or other indications for increased surveillance. Some institutions recommend antenatal fetal surveillance in the late third trimester for women who conceived via assisted reproductive technologies, but the evidence for this recommendation is not substantial.

Female Genital Mutilation

In Western medical practice, female circumcision or "cutting" is called female genital mutilation (FGM). This procedure is practiced in parts of Africa, Asia, and the Middle East, affecting more than 90% of all women in some countries.[126] As women from these regions have immigrated to Europe and the United States, practitioners in areas previously unfamiliar with this practice have found themselves caring for pregnant women with FGM.

The World Health Organization classifies the degree of FGM as type I, involving the removal of the prepuce and clitoral tissue; type II, which includes removal of the prepuce,

clitoris, and labia minora; and type III, called infibulation, in which the clitoris, labia minora, and inner surface of the labia majora are excised. The sides of the labia majora are then stretched toward each other and stitched together, creating a false hood of skin over the urethra and anterior part of the vaginal orifice. Infibulation narrows the vaginal opening and leaves only a small opening at the introitus through which menstrual blood and urine can exit.[126] Long-term complications of infibulation include urinary tract infections, menstrual irregularities and retention, dyspareunia, perineal and pelvic pain, recurrent vaginal infections, infertility, pelvic infections, inclusion cysts, and abscess formation.

Although women with type I and type II FGM do not usually experience obstetric complications, type III FGM can cause obstruction from the resulting anatomy and scar tissue, necessitating the need for deinfibulation or "anterior episiotomy" prior to birth.[127] Deinfibulation generally does not injure underlying structures, as the scar tissue that forms following infibulation does not fuse with tissue of the female genitalia. Discussion of deinfibulation is best initiated early in pregnancy. Counseling includes information about the medical and obstetric risks associated with birth and the options for deinfibulation. Counseling about deinfibulation can take several visits, and physician consultation and involvement early in pregnancy will allow this process to take place over time. Deinfibulation may occur prenatally or during labor.

Substance Use In Pregnancy

Tobacco, alcohol, and illicit and/or prescription medications all have fetotoxic properties, as summarized in **Table 22-18**.[128] A midwife's role in caring for a woman with substance use disorder during pregnancy includes identification of substance being used at the earliest opportunity, provision of counseling resources, accessing medically assisted treatment, and referral to social services to address housing, employment, parenting, and transportation issues.

Table 22-18 **Substances and Associated Harm During Pregnancy**

Pregnancy-Related Complication	Opioids	Cocaine	Amphetamines	Cannabis	Tobacco	Alcohol	Marijuana
Miscarriage	X	X		X	X		
Placental disruption		X			X		
Preterm birth	X	X	X	X	X		X
Fetal growth restriction		X		X	X	X	
Low birth weight	X	X	X	X	X	X	X
Fetal death		X	X	X			
Respiratory disorders/failure	X				X		
Fetal alcohol spectrum						X	
Neonatal abstinence syndrome	X				X	X	
Behavioral and cognitive dysfunction in childhood							X

Based on Foeller ME, Lyell D. Marijuana use in pregnancy: concerns in an evolving era. *J Midwifery Womens Health.* 2017;62(3): 363-367[12]; Finnegan L. Licit and illicit drug use during pregnancy: maternal, neonatal and early childhood consequences. Canadian Centre on Substance Abuse. 2013. Available at: http://www.ccsa.ca/Resource%20Library/CCSA-Drug-Use-during-Pregnancy-Report-2013-en.pdf. Accessed October 6, 2017.[128]

Women with substance use disorders have an increased risk for sexually transmitted infections (STIs), hepatitis, and HIV. Rescreening for STIs in the second and/or third trimester may be indicated and should be considered on an individual basis. Fetal risks are related to the substance and amount of exposure as noted in **Table 22-18**.

Screening for substance use is recommended for all women early in pregnancy. Women may not self-disclose substance use during pregnancy out of fear of intervention by Child Protective Services and because of the potential to have their child taken away at birth. Providing a positive, compassionate, and nonjudgmental prenatal setting can encourage disclosure, although it may take time to build a trusting relationship.

The recommended process of screening, brief intervention, and referral to treatment is referred to as SBIRT. This process has been validated by the U.S. Preventive Services Task Force and other professional organizations.[129] Screening should occur via use of a validated questionnaire such as the 4P's which screens for any substance and asks the woman about past, present, parents, or partner use of substances. The goal of the screening portion of SBIRT is to identify women at low, moderate, or high risk for substance use disorder.

Drug testing is not recommended for universal screening.[129] Urine drug tests have many limitations including the number of substances detected (these tests do not detect alcohol metabolites for example). Urine drug testing requires the woman's permission. In addition, some states require reporting of substance abuse in pregnancy and in the hospital if a drug screen is positive on admission and/or neonatal stool is positive for drugs.

Brief intervention is a woman-centered form of counseling that uses the principles of motivational interviewing. The purpose is to identify the desire to change behavior. Women who are at high risk are referred to specialty treatment.[129] Training in SBIRT is available and resources are listed at the end of this chapter.

Treatment of the substance use disorder is most effective if it involves a multidisciplinary

group of professionals. Social workers, psychologists, substance abuse counselors, and medical addiction specialists are all needed. Referral to a structured program for pregnant women is ideal. Medical addiction specialists in a facility registered with the Substance Abuse and Mental Health Administration will be able to prescribe opioid agonist therapy (OAT) when indicated. Such therapy comprises substitution of an illicit or prescription opioid being overused with an opioid agonist that helps relieve cravings and prevent withdrawal symptoms. OAT medications used during pregnancy are methadone or buprenorphine. Because OAT reduces or eliminates risks associated with dramatic changes in blood opioid levels, perinatal outcomes are improved for both the woman and her fetus/newborn.

Finally, prescription opiate overdose is emerging as an important cause of maternal death in many states within the United States. Coordination of prescriptions within a practice group, reviewing the state prescription registry of controlled substances, and urging referral to pain management specialists may help reduce the rate of prescription misuse. Opioid agonist therapy which is provided by specialists is available for women with opioid use disorders but may be difficult to access in some locales.

Stillbirth

After 20 weeks (or 350 grams if gestational age is uncertain), fetal death is classified as a stillbirth. This term, rather than “fetal death” or “demise,” is preferred. The overall stillbirth rate in the United States is approximately 5.6 per 1000 fetal deaths and live births.[130] Half of all stillbirths occur between 20 and 27 weeks’ gestation, and the other half occur at 28 weeks or later. Of particular concern is the racial disparity in the rate of stillbirth, which is twice as high in non-Hispanic black women (11.3 per 1000 fetal deaths and live births) compared to non-Hispanic white women (4.79 per 1000 fetal deaths and live births). Factors known to contribute to this disparity include social stress, access to health care, and genetic responses to infection, but the full picture of why non-Hispanic black women are at increased risk for stillbirth compared to non-Hispanic white women is not fully known.[131]

The determination of the cause of the stillbirth is extremely difficult. The causes of stillbirth can be maternal, fetal, or placental. In the United States, more than half of all stillbirths have no identifiable cause, and approximately 20% are related to infection (which causes preterm labor and/or preterm premature rupture of membranes). In low-resource countries, infection accounts for more than half of all stillbirths. Other causes of stillbirth include congenital or genetic anomalies, fetomaternal hemorrhage, hydrops fetalis secondary to alloimmunization, umbilical cord abnormalities, maternal disorders such as diabetes or hypertension, and maternal substance use disorder. It is best to refrain from discussing any possible cause for the loss until objective facts are clear and post-delivery studies are completed.

Often the first sign of a stillbirth is the woman’s perception of loss of fetal movement. Inability to detect the fetal heart tones with a Doppler fetal monitor indicates the need for immediate ultrasound to confirm the diagnosis. The midwife should accompany the woman to the ultrasound appointment if at all possible, and notify the ultrasound staff ahead of time that fetal heart tones could not be found. Once the diagnosis is made, immediate physician consultation is recommended. In many states, a physician must make the determination of death,

including fetal death. After birth, midwives may be able to complete the required death certificate, but these regulations vary by state.

The decision to induce labor or wait for labor to occur spontaneously will depend on the woman's choice, her cervical status, and any concomitant medical issues that need to be addressed.[132] Although most women choose immediate induction, expectant management is a viable option for a short period of time. The majority of women (80–90%) will go into labor spontaneously within 2 weeks following a fetal demise. An unusual chronic form of disseminated intravascular coagulation (DIC) can occur if the fetus is retained in utero for more than 4 to 5 weeks; this DIC occurs secondary to slow release of tissue factor from the fetal tissue. If expectant management continues beyond a week or two, coagulation studies measuring prothrombin, partial prothrombin, fibrinogen, and platelets may be performed to screen for DIC prior to induction and at intervals.

Pregnancy After Prior Stillbirth

When a woman has experienced stillbirth, intensified surveillance is indicated in a subsequent pregnancy. Prior to pregnancy or at the first prenatal visit, a detailed history is obtained to review all information available about the prior stillbirth. Medical records can be ordered if needed and physician consultation is recommended to determine a management plan. Plans can be made to mitigate any modifiable factors such as smoking, cocaine use, or glycemic control for women with diabetes. Genetic counseling, as well as thrombophilia and antiphospholipid antibody assessment, may be considered. A detailed anatomic ultrasound (Level II) at 18 to 20 weeks' gestation may be ordered to screen for fetal anomalies and provide reassurance to the woman and her family if the ultrasound findings are normal.

The third trimester is likely to be a time of special anxiety for the woman, her family, and the providers. Although there is no evidence that heightened surveillance lowers the risk of a recurrent stillbirth, many providers offer women frequent prenatal visits or serial nonstress tests, and elective delivery at 38 weeks' gestation. Serial ultrasounds for fetal surveillance including fetal growth are recommended every 2 weeks beginning at 28 weeks.[132]

Perinatal Mood Disorders, Post-traumatic Stress Disorder, and Previous Birth Trauma

Perinatal mood disorders include depression, anxiety disorders, post-traumatic distress disorder (PTSD) and others. Women who have a mental health disorder during pregnancy are at increased risk for perinatal mood disorders in the postpartum period. Compassionate midwifery care can be very helpful for these women as midwives work in concert with other specialists as needed. Screening and treatment of perinatal mood disorders are reviewed in the *Mental Health Conditions* chapter.

Trauma specifically related to childbirth is a recognized cause of PTSD. A compassionate question—"How did your birth go for you?"—is a good approach to initiating assessment for PTSD related to prior traumatic birth (e.g., emergency cesarean, hemorrhage, newborn illness). Factors that increase the risk of PTSD after traumatic birth include history of mental

health disorders, sexual abuse, family violence, loss of a child or spouse, or other emotional/physical trauma. The addition of a traumatic birth can initiate new-onset PTSD symptoms or exacerbate symptoms in cases of preexisting PTSD.

Just as midwives recognize the need to take special care during examinations for women who have experienced abuse or rape, they should appreciate that the same caution applies to women who had a previous traumatic birth. Time needs to be provided to make plans to avoid triggers and reminders of the event in the current pregnancy and birth. Referral to therapists or counselors is indicated to provide the depth and ongoing treatment for PTSD.

Vaginal Birth After Cesarean

Cesarean birth rates have been increasing for two decades in the United States, with repeat cesarean section as the leading cause. Both repeat cesarean birth and vaginal birth after cesarean (VBAC) have health benefits and health risks (**Table 22-19**).[133-135] The decision of whether to perform a repeat cesarean birth or a trial of labor after cesarean (TOLAC) involves balancing several considerations including: (1) the chance of success versus (2) the risks for the woman associated with each option and (3) the risks for the fetus/newborn associated with each option.

Table 22-19 **Risks Associated with Trial of Labor After Cesarean Versus Elective Repeat Cesarean**

Outcome	Trial of Labor After Cesarean	Elective Repeat Cesarean	Analysis
Short-Term Effects: Maternal			
Risk of uterine rupture	7 per 1000 women	2.6 per 10,000 women	4/5/1000 more uterine rupture in women undergoing TOLAC
Maternal mortality	3.8 per 100,000 women at term	13.4 per 100,000 women at term	Approximately 9/100,000 fewer maternal deaths in women undergoing TOLAC
Hysterectomy	1.57 per 1000 women	2.8 per 1000 women	No significant difference
Blood transfusion	6.6 per 1000 women at term	4.6 per 1000 women at term	No significant difference
Operative injury	4.0–5.1 per 1000 women	2.5–4.4 per 1000 women	Differing study methodologies make this comparison difficult.
Infection	46 per 1000 women	32 per 1000 women	Differing study methodologies make this comparison difficult. No significant difference
Hospital stay	2.5 days	3.92 days	Longer for ERCD but longest for women who have failed TOLAC that results in repeat ERCD
Urinary incontinence	No data	No data	Inconclusive evidence
Pelvic floor disorders	No data	No data	Inconclusive evidence
Short-Term and Long-Term Effects: Infant			
Perinatal mortality	13 per 10,000	5 per 10,000	Approximately 1 more death per 1000 TOLAC
Hypoxic ischemic encephalopathy if uterus ruptures	8.9 per 1000	3.2 per 1000	Strength of evidence is low secondary to methodologic differences in studies
Respiratory problem in the first days after birth	5.4 per 100	2.5 per 100	Insufficient studies to assess differences
Effects on Subsequent Pregnancy Following Cesarean Birth			
Placenta previa in subsequent pregnancy	1% in women with 1 prior cesarean 1.7% in women with 2 prior cesareans 2.8% in women with ≥ 3 prior cesareans		
Placenta accreta in subsequent pregnancy with placenta previa	5% in women with 1 prior cesarean 24% in women with 2 prior cesareans 47% in women with 3 prior cesareans		

Abbreviations: ERCD, elective repeat cesarean delivery; TOLAC, trial of labor after cesarean.

Based on Guise J-M, Eden K, Emeis C, Denman MA, Marshall N, Fu R, et al. Vaginal birth after cesarean: new sights. Vol. No. 10-E001. Rockville, MD: Agency for Healthcare Research and Quality, 2010.[134]

A woman's chance of having a successful VBAC is affected by several individual factors, such as the indication for the prior cesarean, history of a vaginal birth, and current obstetric factors such as spontaneous onset of labor versus induced labor. For example, although the overall chance of success is approximately 70%, women who have had one prior vaginal birth in addition to a cesarean have a much higher chance of success. Conversely, women who had one prior cesarean for possible cephalopelvic disproportion have a much lower chance of success.

The risks associated with repeat cesarean birth include excessive blood loss, post-operative

infection, accidental tissue laceration such as bladder nicking, anesthesia reaction, and increased risk for placenta previa and placenta accreta in future pregnancies.[133-135]

The risks associated with TOLAC include unplanned repeat cesarean, which is associated with more morbidity than a repeat cesarean and uterine rupture. A complication unique to vaginal birth after cesarean, uterine rupture has significant adverse sequelae for both the woman and the infant.[133-135]

Women approaching this decision require a detailed review of the benefits and risks of each option based on their individual history as part of a shared decision making process.[133-136] Birth in an out-of-hospital setting is not recommended given the most recent analyses of outcomes following labor and/or birth at home.[137,138] Women considering TOLAC should also be informed about available hospital and provider resources. Online VBAC success rate calculators can assist the midwife in providing guidance. Although these calculators have limitations with regard to prediction of adverse outcomes, they can be helpful for estimating an individual's success of a trial of labor. (See the Resources section at the end of this chapter.)

Pregnancy After Bariatric Surgery

Midwives increasingly provide care for women after bariatric surgery as this procedure becomes more common for treatment of diabetes and infertility. Bariatric surgery is recommended when lifestyle, diet and medication therapies have failed and BMI exceeds 35 kg/mm^2 or when BMI exceeds 30 kg/mm^2 and there are comorbid conditions such as diabetes or hypertension.[139] There are two common types of bariatric surgery procedures: (1) restrictive (laparoscopic gastric banding or LAP-BAND) and (2) restrictive/malabsorptive (Roux-en-Y gastric bypass). Current recommendations advise women to wait between 12 and 18 months postbariatric surgery so that weight loss is stabilized before starting a pregnancy.[140]

Pregnancy following bariatric surgery is safer than pregnancy when BMI exceeds 40 kg/mm^2. Reviews to assess the advantages of bariatric surgery prior to pregnancy found that bariatric surgery reduced the risk for gestational diabetes, preeclampsia, hypertension, and large-for-gestational-age neonates, with most women experiencing no complications during pregnancy.[139,141,142]

Knowing the type of bariatric procedure done allows the midwife to provide appropriate nutritional advice and risk screening. Pregnancy weight gain recommendations are based on pregravid BMI even after bariatric surgery. LAP-BAND slippage and movement can occur during pregnancy and result in severe vomiting.[142] Some advocate for deflating the band prior to pregnancy to allow for adequate nutrition, though there are no national guidelines.

Malabsorptive surgeries reduce food, micronutrient and medication absorption. Deficiencies in iron, vitamin B_{12}, folate, vitamin D, calcium, and protein can be common. Baseline vitamin and mineral status is evaluated at the first prenatal visit with supplements recommended as needed. A complete blood count and measurement of B_{12}, iron, ferritin, calcium, and vitamin D levels every trimester should be considered.

Women are prone to dumping syndrome following malabsorptive surgeries. Gestational diabetes testing can be done by following fasting and postprandial blood glucose levels after

breakfast for 1 week.[140] Third trimester serial ultrasounds document fetal growth if maternal weight gain has been limited or abdominal adipose limits palpation.

The midwife providing prenatal care for women following bariatric surgery coordinates care among a network of clinicians including maternal-fetal medicine physicians, nutritionists, and ultrasonographers. Care within this team optimizes a woman's chance for a safe and satisfying birth.

Techniques for Fetal Surveillance

The terms “fetal surveillance” and “antenatal testing” refer to tests that assess fetal well-being in the late second or early third trimester. The goals of antenatal fetal testing include (1) prevention of stillbirth, (2) identification of the fetus whose oxygen status is compromised so as to allow intervention before irreversible metabolic acidosis ensues, and (3) avoidance of unnecessary interventions when other clinical parameters are equivocal. This section reviews the theory, indications, methodology, and normal versus abnormal findings for the following tests: fetal kick count, nonstress test (NST), biophysical profile (BPP), contraction stress test (CST), and Doppler indices. Antenatal testing is a classic “screening test” in that if the results are normal, fetal well-being is assured. In contrast, if results are abnormal, further assessment is indicated to determine fetal well-being or fetal compromise.

Tests for fetal well-being are clinically useful primarily because their negative predictive value is 99% or higher.[143,144] The incidence of false-positive tests varies between 50% and 60% depending on the test. Because the positive predictive values are much lower than the negative predictive values, clinical management of abnormal fetal surveillance tests varies depending on other clinical factors.[145] The interpretation of these testing methods is summarized in **Table 22-20**.

Table 22-20 Interpretation of Fetal Surveillance Tests

Name	Results	Criteria
Contraction stress test (CST) or breast stimulation test (BST)[a]	Negative	Normal FHR without late decelerations
	Equivocal suspicious	Intermittent late decelerations or variable decelerations
	Equivocal hyperstimulation	Decelerations that occur in presence of tachysystole
	Unsatisfactory	Unable to obtain a satisfactory FHR tracing
	Positive	Recurrent late decelerations following 50% or more of contractions even if fewer than 3 contractions in 10 minutes
Nonstress test (NST)[b]	Reactive	≥ 2 accelerations within 20 minutes (some settings extend to 40 minutes)
	Nonreactive	< 2 accelerations in 40 minutes
	Inconclusive	Unable to obtain a satisfactory FHR tracing
		Variable or late decelerations or other Category II FHR tracing
		Reactive NST with FHR decelerations
Biophysical profile (BPP)	Normal	≥ 8/10 or 8/8 if NST excluded
	Equivocal	6/10
	Abnormal	≤ 4/10
Modified NST	Normal	Reactive NST and AFI > 5 cm
	Abnormal	Nonreactive NST and/or AFI ≤ 5 cm

Abbreviations: AFI, amniotic fluid index; FHR, fetal heart rate.

[a] This test is interpreted once the woman has at least 3 contractions within 10 minutes.

[b] An acceleration must be 15 beats per minute above the baseline, and must last 15 seconds or more from onset to resolution, after 32 weeks' gestation. The acceleration must be 10 beats per minute above baseline and last 10 seconds in a preterm fetus.

Fetal Physiologic Indices and Factors That Affect Fetal Behavior

Prenatal surveillance of fetal well-being is based on the observation that specific fetal behavior states reflect adequate oxygenation. The fetal heart rate pattern, level of fetal activity, and degree of muscle tone are sensitive to hypoxemia and acidosis. Therefore, fetal oxygenation can be indirectly evaluated by assessing biophysical parameters, just as vital signs are used to assess well-being in an adult or child. Fetal biophysical behavior that is assessed in the various prenatal fetal tests includes fetal heart rate parameters, fetal movement, fetal breathing, and quantification of amniotic fluid (because the amount of amniotic fluid is an indication of fetal renal function and renal perfusion). Each of these parameters can be affected by a multitude of factors.

Maternal awareness of fetal movement begins in the second trimester; multiparous women usually start feeling the fetus between 16 and 18 weeks, whereas primiparous women usually start feeling the fetus between 18 and 22 weeks. Women who have an anterior placenta may begin detecting fetal movement somewhat later than women whose placenta is posterior. Fetal movement is initially slight and irregular, gradually becoming stronger and more frequent. Fetal movement maximizes around 34 weeks, but then appears to become less frequent. This pattern occurs because, as the central nervous system matures, the fetus begins to exhibit longer and cyclic sleep or quiet alert states wherein movement does not occur. Thus, the woman's perception can be one of decreased movement toward the end of the pregnancy. Women usually perceive approximately 50% of isolated limb movements and 80% of movements that involve both the trunk and the limb.[146] **Table 22-21** lists maternal factors that affect perception of fetal behavior.

Table 22-21	Factors That Influence Maternal Perception of Fetal Movement
Decreased Fetal Movement	
Obesity	
Maternal position (fetal movement is perceived best in a recumbent position as compared to sitting or standing)	
Anterior placenta	
Amniotic fluid volume (polyhydramnios and oligohydramnios)	
Drugs:	
Corticosteroids	
Sedatives and alcohol prolong quiet sleep states	
Tobacco (short period of time after smoking a cigarette)	
Hypoglycemia	
Fetal spine in an anterior position (occiput anterior)	
No Difference in Perception of Fetal Movement	
Parity	

Maternal anxiety
Increased Fetal Movement
Maternal meal or increase in blood glucose levels
Evening hours: fetal movement is the least evident in the morning

Fetal behavior—including fetal tone, movement, breathing, and fetal heart rate characteristics—changes as the fetus becomes hypoxic. The functions that appear first in gestation disappear last as hypoxia and acidosis increase. In general, loss of fetal heart rate reactivity, fetal heart rate decelerations, and reduced fetal activity are fetal responses to acute hypoxia, whereas oligohydramnios, fetal growth restriction, and altered umbilical artery blood flow are responses to chronic hypoxia. Additional information on the fetal heart rate responses to acute hypoxia can be found in the *Fetal Assessment During Labor* chapter.

Indications for Antenatal Testing

Although indications for antenatal testing vary between institutions, **Table 22-22** lists those that are most common to all settings.[145] Likewise, determining when to start testing will vary depending on the condition. Most antenatal testing is started at 34 to 36 weeks and is conducted either weekly or biweekly.

Table 22-22 Selected Indications for Antenatal Testing[a,b]

Maternal	Pregnancy Related	Fetal
Age > 40 years	Abnormal maternal serum screening (one or two abnormal values, unexplained)	Abruption in current pregnancy
Active substance use	Assisted reproductive technology	Congenital abnormalities
Antiphospholipid antibody syndrome	Cholestasis of pregnancy	Decreased fetal movement
Cyanotic heart disease	Late term or post-term pregnancy	Fetal growth restriction
Chronic hypertension	Multifetal gestation	Oligohydramnios
Diabetes requiring insulin	Pemphigoid gestationis	Polyhydramnios
Hemoglobinopathies	Preeclampsia	Congenital abnormalities
Hypertension		Decreased fetal movement
Hyperthyroidism, poorly controlled		Fetal growth restriction
Renal disease		Umbilical cord abnormalities
Stillbirth in previous pregnancy		
Seizure disorder, poorly controlled		
Sickle cell disease		
Systemic lupus erythematosus		

[a] This table presents a representative list of common indications. Individual practices will have indications for prenatal testing identified in collaboration with consulting physicians that may include additional conditions and that

may not include some conditions that appear in this table.

[b] Additional indications for women hospitalized in the prenatal period include conditions such as preterm premature rupture of membranes, Rh isoimmunization, and fetal surgery.

Fetal Movement Counting (Kick Counts)

The rationale for formal fetal movement counting is that 30% to 50% of stillbirths occur in women with low-risk pregnancies and structurally normal fetuses in whom there were no indications for prenatal testing. In addition, fetal movement starts to decrease several days prior to stillbirth, and women who report decreased fetal movement have a higher incidence of fetal growth restriction and stillbirth. These observations became the basis for introducing fetal movement counts for the purpose of preventing stillbirth.

More than 40 years of literature and research has now been accumulated on the relationship between fetal movement and adverse pregnancy outcomes. Formal fetal movement counting does not prevent all stillbirth in the general population, but women who have a heightened awareness of fetal movement will detect decreased movement and these women self-select into a higher risk category in which more intense evaluation is of value. The clinical question has long been, “Should all women perform formal fetal movement counts during the third trimester or just women who are at high risk for stillbirth?” This question has not been definitively answered, and the RCTs conducted on routine fetal movement counting have yielded mixed results.[146,147] Currently, some clinicians recommend formal fetal movement counting for all pregnant women, whereas others recommend formal fetal movement counting only for those women who are at increased risk for fetal growth restriction or stillbirth. The American College of Obstetricians and Gynecologists does not recommend formal fetal movement counting for women who are not at risk for chronic fetal hypoxia.[145]

The specific fetal movement count that is the best “alarm” or trigger for additional evaluation has not been determined. The method most commonly used is the “Count to 10” method, whereby a woman focuses attention on fetal movement and records how long it takes to document 10 fetal movements. If it takes longer than 2 hours, the woman is to call her midwife; she is then asked to come in for a formal nonstress test (**Figure 22-8**).

Name _____

At the same time each day, count the baby's movements. When you have felt 10 movements, record the length of time it took. All movements, even small ones, count toward the total. If you have not felt 10 movements in the usual amount of time, please call your midwife.

Week of _____

Day	Start Time	Hours taken to feel 10 movements of the baby: 1	2	3	4	5	6	7	8	9	10
M											
T											
W											
T											
F											
S											
S											

Figure 22-8 Fetal movement counting chart.

Women close to term often call their midwives to report decreased fetal movement. If the woman has no risks for uteroplacental insufficiency, it is reasonable to recommend that she eat something, rest in a semirecumbent position, and then count fetal movement. If she notes 10 movements in an hour, she can be reassured and the midwife may recommend that she repeat the formal fetal movement counts for a few days at the same time of day. If she notes fewer than 10 movements, the midwife can either have her count for another hour to see if 10 movements occur or have her be seen for a formal auscultated acceleration test or nonstress test. Because women who report decreased fetal movement have an increased risk for adverse outcomes, the midwife should have a low threshold for having a woman come for additional testing.

Contraction Stress Test

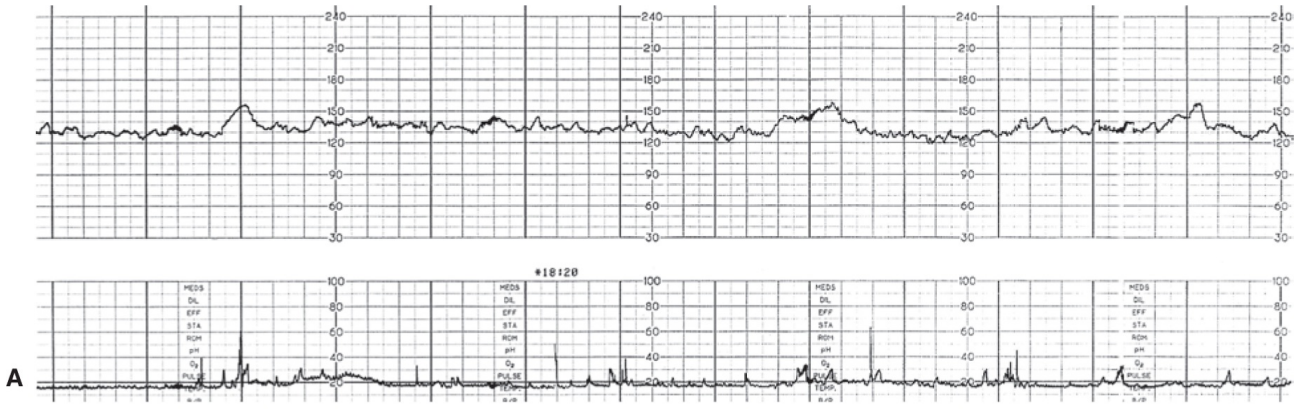
The CST was the first antenatal fetal test employed in clinical practice. It is based on the observation that uterine contractions transiently restrict blood flow to the intervillous space, thereby lowering oxygen availability to the fetus. The compromised fetus will respond with a late deceleration. (For more description of the pathophysiology of the fetal heart rate response to contractions, see the *Fetal Assessment During Labor* chapter.) The CST entails intravenous administration of oxytocin to initiate uterine contractions and continuous electronic fetal monitoring. A breast stimulation test (BST) may also be performed to initiate contractions, as an alternative to administering intravenous oxytocin. In the BST, the woman massages one nipple through her clothing for 2 minutes, followed by a rest for 5 minutes and then a repeat of the procedure on the other nipple. She should not stimulate the breast during a contraction. The BST and CST are performed in similar fashion, but the BST is associated with more uterine hyperstimulation. Interpretation of results is the same, however.

The fetal response to 3 contractions in a 10-minute window is measured during the test. The CST has the lowest false-negative rate of all antenatal fetal tests (0.04%) but a high false-

positive rate of approximately 30% and is expensive to perform. The NST and the BPP have largely replaced the CST except in inpatient settings, where it is important to establish the fetus's ability to tolerate uterine contractions prior to induction of labor.

Nonstress Test

The NST is the most common method of antenatal testing used in practice today. Its false-negative rate ranges from 0.3% to 0.65%, and the false-positive rate is approximately 55% to 90% depending on the population studied.[148] External fetal monitoring is initiated when the woman is in a side-lying semirecumbent position. The test is concluded when 2 fetal heart rate accelerations appear within a 20-minute window in a fetal heart rate tracing that has moderate variability, no decelerations, and a normal baseline, or when accelerations are unable to be elicited (**Figure 22-9**). If accelerations do not appear in the first 20 to 30 minutes, vibroacoustic stimulation may be applied. Vibroacoustic stimulation lowers the incidence of nonreactive results. The generation of accelerations via vibroacoustic stimulation appears to be as valid as the observation of spontaneous accelerations in predicting fetal well-being. The interpretive criteria for NST results are listed in **Table 22-20**. Management of inconclusive and nonreactive NSTs varies by practice and by the condition for which the NST was performed.



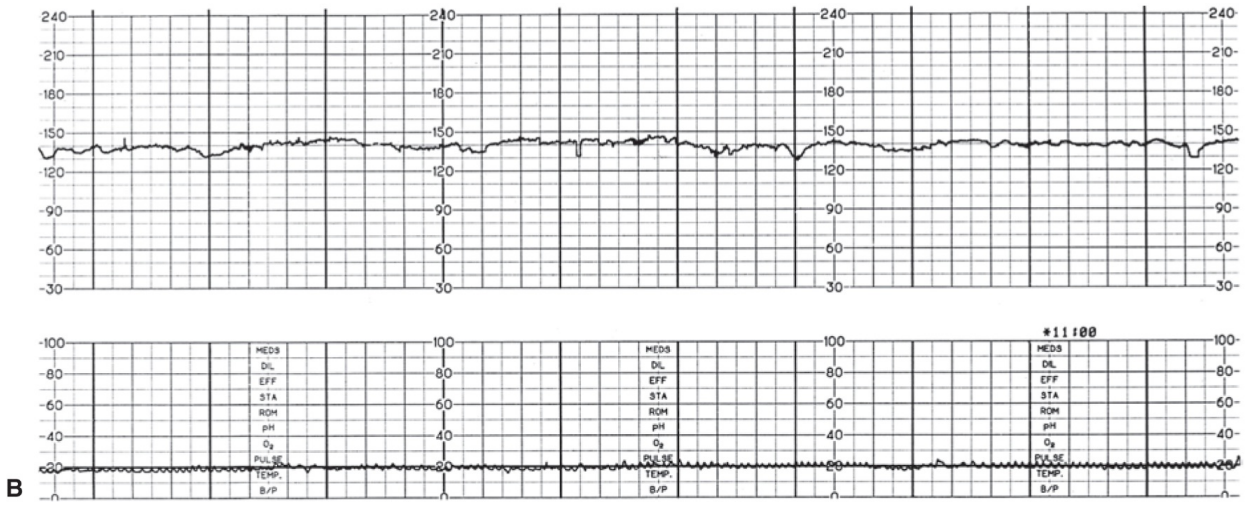


Figure 22-9 **A.** Reactive nonstress test. **B.** Nonreactive nonstress test.

Because a rise in blood glucose can initiate fetal movement, clinicians have traditionally believed that a sudden infusion of glucose via juice or candy would stimulate fetal accelerations. Studies of this practice, however, have found that glucose does not improve the results of antenatal fetal testing.[149]

Auscultated Nonstress Test

A nonstress test can be performed via auscultation with a fetoscope. During the 1980s, Paine et al. researched the use of auscultation of the fetal heart rate as a means of predicting fetal well-being prenatally.[150] From this work, a method of auscultation was devised as a simple alternative to the nonstress test and as a formal method of auscultation for antenatal testing. The auscultated acceleration test (AAT) has been validated as a predictor of both reactive and nonreactive NST results. The procedure and interpretation for an auscultated acceleration test are described in **Appendix 22A**. Anecdotal accounts indicate that use of low-technology techniques such as fetal movement counting and the AAT has led to appropriate management of compromised fetuses that would not otherwise have been identified as high risk by usual prenatal care services in midwifery practice.

Biophysical Profile

The BPP employs both ultrasound and NST. The ultrasound portion is an “intrauterine Apgar score” that includes observation of AFI, fetal breathing, fetal tone, and fetal movement. The NST provides the fifth component of the test. Each examined factor has a possible score of 2, for a maximum total score of 10 (**Table 22-23**). The BPP has a lower false-negative rate (0.08%) and fewer false-positive tests (60%) when compared to the CST or NST.[148]

Table 22-23 **The Biophysical Profile**

Variable	Adequate Score = 2	Inadequate Score = 0
Fetal breathing movement	≥ 1 episode of fetal breathing movements of ≥ 30 seconds in duration	< 30 seconds of sustained fetal breathing movements
Fetal movement	≥ 3 discrete body/limb movements (simultaneous limb and trunk movements are counted as a single movement)	≤ 2 movements
Fetal tone	≥ 1 episode of active extension with rapid return	Either slow extension with return to partial flexion or movement of limb in full trunk, or hand extension, or absent fetal movement
Fetal heart rate over 20 minutes	≥ 2 accelerations of ≥ 15 beats/min, peak amplitude lasting ≥ 15 seconds from the baseline with moderate variability in 20 minutes	< 2 accelerations or accelerations < 15 beats/min peak amplitude or accelerations < 15 seconds duration in 20 minutes Minimal or absent variability
Amniotic	Single deep pocket > 2 cm that does not include	No pocket of fluid that is > 2 cm

fluid volume[a]	umbilical cord or fetal extremities	
Amniotic fluid index (AFI)[a]	≥ 5.0 cm	< 5.0 cm, oligohydramnios
	5.1–8	Low normal
	8.1–24	Normal
	> 24	Polyhydramnios

[a] Amniotic fluid volume or AFI can be used as the fifth component of the biophysical profile. Evidence from randomized controlled trials has found the amniotic fluid volume of a pocket < 2 cm to be a reliable diagnosis of oligohydramnios and associated with fewer obstetric interventions when compared to the AFI.

Individual biophysical activities appear at different stages of fetal development. Fetal tone appears at approximately 8 gestational weeks, fetal movement at 9 gestational weeks, fetal breathing at 21 gestational weeks, and fetal heart rate reactivity by the late second trimester. The biophysical activity that appears first is also the last to disappear when acidemia is present. Thus, fetal heart rate variability is first to disappear; conversely, the absence of fetal tone predicts fetal acidemia 100% of the time.

The relationship between the BPP score and fetal acidemia has been studied extensively. A full-term fetus with a score of 8 to 10 has a risk of fetal asphyxia occurring within a week after the test of approximately 1 per 1300 tests, whereas the risk of fetal asphyxia for a fetus with a score of 1 to 4 is between 91/1000 and 600/1000, respectively.[148]

Modified Biophysical Profile

The full BPP can take some time to perform. The modified BPP, composed of an NST and AFI, has similar false-negative and false-positive rates as the full BPP, and has become the most common prenatal testing conducted. Nonstress testing can identify acute hypoxia, and decreased amniotic fluid is considered to reflect the presence of chronic hypoxia. The modified BPP has a predictive value similar to that of a full biophysical profile when the NST is reactive and the AFI is higher than 5.0 cm. If the NST is nonreactive or if the AFI is less than 5.0 cm, a complete BPP should be performed. If all of the ultrasound components of the BPP are reassuring, performing the NST is not necessary for confirmation of fetal well-being.

Doppler Indices

Doppler ultrasound uses the waveform properties of arteries or veins to analyze the passage of blood through that vessel. Evaluations of the Doppler velocimetry within the umbilical arteries, fetal aorta, and middle cerebral artery are recently introduced antenatal tests used to assess placental function in women who may have a growth-restricted fetus.

The end-diastolic flow in the uterine artery normally increases with advancing gestation secondary to decreased resistance in the placenta. As more tertiary vessels develop, the pressure within the artery drops slightly. The systolic/diastolic (S/D) ratio of the umbilical artery normally decreases as gestational age increases. This development takes place secondary to the higher diastolic velocity that occurs as the number of placental villi increase. Abnormal umbilical artery Doppler studies have been consistently linked to abnormal fetal

outcomes. It is estimated that no diastolic flow reflects a loss of 60% to 70% of placental vascularity. Abnormal elevation of the umbilical artery S/D ratio occurs before fetal heart rate variability decreases, which makes this assessment a useful data point when evaluating a fetus with fetal growth restriction. The rate of perinatal death is decreased when uterine artery Doppler studies are added to standard antepartum testing in the setting of FGR. Assessment of the umbilical artery S/D ratio is a standard component of care for the fetus with fetal growth restriction.

Prediction of fetal risks based on umbilical artery Doppler indices may be improved with the addition of assessments of umbilical vein Doppler indices, the middle fetal cerebral artery, and, most recently, the ductus venosus. For example, vasodilation and high diastolic flow in the middle fetal cerebral artery reflects the “brain-sparing” response to hypoxia, although tests using this observation are still being evaluated for clinical utility. Flow through the ductus venosus is being used to assess fetal cardiac function.

Conclusion

Although midwives primarily care for women without pregnancy-related conditions, an essential component of that care is careful risk screening and early detection of complications. In addition, when a woman does have a condition that is troubling or a complication, she can be reassured when given accurate information and emotional support. The midwife who understands the pathophysiology and the maternal, fetal, and newborn risks associated with a pregnancy-related condition, as well as the interventions for complications when they occur and risks associated with those interventions, will be best positioned to provide continuity of care and the information women need most for the current pregnancy, future pregnancy, and ongoing health.

Resources

Organization	Description	Webpage
Adolescent Pregnancy		
Geneva Foundation for Medical Education and Research (GFMER)	Guidelines for care of adolescents during pregnancy worldwide.	https://www.gfmer.ch/Guidelines/Adolescent_gynecology_and_contrac
Global Library of Women's Medicine (GLOWM)	Dopkins Broecker & Hillard's chapter about pregnancy in adolescence (doi:10.3843/GLOWM.10414).	http://www.glowm.com/section_view/heading/Pregnancy%20in%20Ad
Congenital Anomalies and Genetic Counseling		
Centers for Disease Control and Prevention (CDC)	Resources for clinicians and families.	https://www.cdc.gov/ncbddd/birthdefects/families-support.html
National Human Genome Research Institute (NHGRI)	The website has links to many genetic counseling organizations.	https://www.genome.gov/27533643/genetic-counseling-resources/
North American Fetal Therapy Network (NAFTNet)	Voluntary consortium of specialists and sites that provide fetal therapy as part of research protocols.	https://www.naftnet.org
Preterm Labor		
American College of Obstetricians and Gynecologists (ACOG)	Preterm labor resources: This website includes ACOG guidelines for risks, diagnosis, and treatments for preterm labor.	https://www.acog.org/Womens-Health/Preterm-Premature-Labor-and-
March of Dimes	The March of Dimes has multiple resources for clinicians and pregnant women.	https://www.marchofdimes.org
Vaginal Birth After Cesarean (VBAC) Calculator		
Maternal-Fetal Medicine Network	Calculator that predicts the change of VBAC using data available at entry to care.	https://mfmunetwork.bsc.gwu.edu/PublicBSC/MFMU/VGBirthCalc/vagl
	Calculator that predicts the change of VBAC using data available at time of labor and	https://mfmunetwork.bsc.gwu.edu/PublicBSC/MFMU/VGBirthCalc/vagl

	birth.
Screening, Brief Intervention and Referral to Treatment (SBIRT) Counseling	
SBIRT Training	Online training with continuing medical education and resources for developing SBIRT counseling skills https://www.sbirttraining.com

References

1. American College of Nurse-Midwives, American College of Obstetricians and Gynecologists. College statement of policy: joint statement of practice relations between obstetrician-gynecologists and certified nurse-midwives/certified midwives. 2011. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000224/ACNM.ACOG%20Join Accessed September 30, 2017.
2. American College of Obstetricians and Gynecologists. Obstetric Care Consensus No. 2: levels of maternal care. *Obstet Gynecol*. 2015:125:502-515.
3. McParlin C, O'Donnell A, Robson SC, et al. Treatments for hyperemesis gravidarum and nausea and vomiting in pregnancy: a systematic review. *JAMA*. 2016;316(13):1392-1401.
4. King TL, Murphy PA. Evidence-based approaches to managing nausea and vomiting in early pregnancy. *J Midwifery Womens Health*. 2009;54:430-444.
5. Mitchell-Jones N, Gallos I, Farren J, Tobias A, Bottomley C, Bourne T. Psychological morbidity associated with hyperemesis gravidarum: a systematic review and meta-analysis. *BJOG*. 2017;124(1):20-30.
6. Fejzo MS, Macgibbon K. Hyperemesis gravidarum: it is time to put an end to the misguided theory of a psychiatric etiology. *Gen Hosp Psychiat*. 2012:34(6):699-700.
7. Bustos M, Venkataramanan R, Caritis S. Nausea and vomiting of pregnancy: what's new? *Auton Neurosci*. 2017;202:62-72.
8. Lacasse A, Rey E, Ferreira AE, Morin C, Berard A. Validity of a modified Pregnancy-Unique Quantification of Emesis and Nausea (PUQE) scoring index to assess severity of nausea and vomiting of pregnancy. *Am J Obstet Gynecol*. 2008;198(1):71.31-71.37.
9. Slaughter SR, Hearns-Stokes R, van der Vulgt T, Joffe HIV. FDA approval of doxylamine-pyridoxine therapy for use in pregnancy. *N Engl J Med*. 2014; 320(12):1081-1083.
10. Tan PC, Norazilah MJ, Omar SZ. Dextrose saline compared with normal saline rehydration of hyperemesis gravidarum: a randomized controlled trial. *Obstet Gynecol*. 2013;121(2 pt 1):291-298.
11. Carstairs SD. Ondansetron use in pregnancy and birth defects: a systematic review. *Obstet Gynecol*. 2016; 127(5):878-883.
12. Foeller ME, Lyell D. Marijuana use in pregnancy: concerns in an evolving era. *J Midwifery Womens Health*. 2017;62(3):363-367.
13. Alaniz VI, Liss J, Metz TD, Stickrath E. Cannabinoid hyperemesis syndrome: a cause of refractory nausea and vomiting in pregnancy. *Obstet Gynecol*. 2015;125(6):1484-1486.
14. Fields L, Hathaway A. Key concepts in pregnancy of unknown location: identifying ectopic pregnancy and providing patient-centered care. *J Midwifery Womens Health*. 2017;62:172-179.
15. Reproductive Health Access Project. First trimester bleeding algorithm [Internet]. Available at: https://www.reproductiveaccess.org/resource/first-trimester-bleeding-algorithm/. Accessed December 6, 2017.
16. Connolly AM, Ryan DH, Stuebe AM, Wolfe HM. Reevaluation of discriminatory and threshold levels for serum-hCG in early pregnancy. *Obstet Gynecol*. 2013;121(1):65-70.
17. Seeber B. What serial hCG can tell you, and cannot tell you, about an early pregnancy. *Fertil Steril*. 2012;98:1074-1077.
18. Kriebs JM, Fahey JO. Ectopic pregnancy. *J Midwifery Womens Health*. 2006;51:431-439.
19. Verhaegen J, Gallos ID, van Mello NM, et al. Accuracy of single progesterone test to predict early pregnancy outcome in women with pain or bleeding: meta-analysis of cohort studies. *BMJ*. 2012;345-354.
20. Barash JH, Buchanan EM, Hillson C. Diagnosis and management of ectopic pregnancy. *Am Fam Physician*. 2014;90(1):34-40.
21. Regan L, Rai R. Epidemiology and the medical causes of miscarriage. *Baillieres Best Pract Res Clin Obstet Gynaecol*. 2000;14:839.
22. Goddijn M, Leschot NJ. Genetic aspects of miscarriage. *Baillieres Best Pract Res Clin Obstet Gynaecol*. 2000;14:855.
23. Nanda K, Lopez LM, Grimes DA, Peloggia A, Nanda G. Expectant care versus surgical treatment for miscarriage. *Cochrane Database Syst Rev*. 2012;3:CD003518. doi:10.1002/14651858.CD003518.pub3.
24. American College of Obstetricians and Gynecologists. Practice Bulletin No. 150: early pregnancy loss. *Obstet Gynecol*. 2015;125(5):1258-1267. [Reaffirmed 2017].
25. Casikar I, Bignardi T, Riemke J, Alhamdan D, Condous G. Expectant management of spontaneous first-trimester miscarriage: prospective validation of the "2-week rule." *Ultrasound Obstet Gynecol*. 2010;35:223-227.
26. Doubilet PM, Benson CB, Bourne T, et al. Diagnostic criteria for nonviable pregnancy early in the first trimester. *N Engl J Med*. 2013;369(15):1443-1451.
27. Haas DM, Ramsey PS. Progestogen for preventing miscarriage. *Cochrane Database Syst Rev*. 2013;10:CD003511. doi:10.1002/14651858.CD003511.pub3.
28. Coomarasamy A, Williams H, Truchanowicz E, et al. PROMISE: first-trimester progesterone therapy in women with a

history of unexplained recurrent miscarriages: a randomised, double-blind, placebo-controlled, international multicentre trial and economic evaluation. *Health Technol Assess*. 2016;20(41):1-92.
29. Monchek R, Wiedaseck S. Gestational trophoblastic disease: an overview. *J Midwifery Womens Health*. 2012;57:255-259.
30. Minogue JP, Reedy NJ. Companioning families in perinatal decision-making. *J Perinat Neonat Nsg*. 1988;1(3):25-35.
31. Chervanak F, McCullough LB. Responsibly counseling women about the clinical management of pregnancies complicated by severe fetal anomalies. *J Med Ethics*. 2012;38(7):397-398.
32. Norton ME. Follow-up of sonographically detected soft markers for fetal aneuploidy. *Semin Perinatol*. 2013;37:365-369.
33. American College of Obstetricians and Gynecologists. Practice Bulletin No. 175: ultrasound in pregnancy. *Obstet Gynecol*. 2016;128:e241-e256.
34. Rao R, Platt LD. Ultrasound screening: status of markers and efficacy of screening for structural abnormalities. *Semin Perinatol*. 2016;40(1):67-78.
35. Robert Peter J, Ho JJ, Valliapan J, Sivasangari S. Symphysial fundal height (SFH) measurement in pregnancy for detecting abnormal fetal growth. *Cochrane Database Syst Rev*. 2015;9:CD008136. doi:10.1002/14651858.CD008136.pub3.
36. American College of Obstetricians and Gynecologists. Practice Bulletin No. 173: fetal macrosomia. *Obstet Gynecol*. 2016;128:e195-e209.
37. American College of Obstetricians and Gynecologists. Practice Bulletin No. 134: fetal growth restriction. *Obstet Gynecol*. 2013;121:1122-1133.
38. Nardozza LM, Caetano ACR, Zamarian AC, et al. Fetal growth restriction: current knowledge. *Arch Gynecol Obstet*. 2017;295:1061-1077.
39. Goodnight W. Newman R. Optimal nutrition for improved twin pregnancy outcome. *Obstet Gynecol*. 2009;114:1121-1134.
40. Glezerman M. Planned vaginal breech delivery: current status and the need to reconsider. *Expert Rev Obstet Gynecol*. 2012;7:159-166.
41. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 161: external cephalic version. *Obstet Gynecol*. 2016;127:e54-e61.
42. Hofmeyr GJ, Kulier R. External cephalic version for breech presentation at term. *Cochrane Database Syst Rev*. 2012;10:CD000083. doi:10.1002/14651858.CD000083.pub2.
43. Hofmeyr GJ, Kulier R. Cephalic version by postural management for breech presentation. *Cochrane Database Syst Rev*. 2012;10:CD000051. doi:10.1002/14651858.CD000051.pub2.
44. Schuler-Maloney D. Placental triage of the singleton placenta. *J Midwifery Womens Health*. 2000;45:104-113.
45. Taniguchi H, Aoki S, Sakamaki K, et al. Circumvallate placenta: associated clinical manifestations and complications—a retrospective study. *Obstet Gynecol Int*. 2014;2014:986230. doi:10.1155/2014/986230.
46. Wiedaseck S, Monchek R. Placental and cord insertion pathologies: screening, diagnosis, and management. *J Midwifery Womens Health*. 2014;59:328-335.
47. Sinkey RG, Odibo AO, Dasche JS. #37: Diagnosis and management of vasa previa. *Am J Obstet Gynecol*. 2015;213(5):615-619.
48. Predanic M, Perni SC, Chasen ST, Baergen RN, Chervenak FA. Ultrasound evaluation of abnormal umbilical cord coiling in second trimester of gestation in association with adverse pregnancy outcome. *Am J Obstet Gynecol*. 2005;193(2):387-394.
49. Reddy UM, Abuhamad AZ, Levine D, Saade GR. Fetal imaging: executive summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, American Institute of Ultrasound in Medicine, American College of Obstetricians and Gynecologists, American College of Radiology, Society for Pediatric Radiology, and Society of Radiologists in Ultrasound Fetal Imaging workshop. *Obstet Gynecol*. 2014;123(5):1070-1082.
50. Silver RM. Abnormal placentation: placenta previa, vasa previa, and placenta accreta. *Obstet Gynecol*. 2015;126:654-668.
51. Rosenberg T, Pariente G, Sergienko R, Wiznitzer A, Sheiner E. Critical analysis of risk factors and outcome of placenta previa. *Arch Gynecol Obstet*. 2011:284(1):47-51.
52. Silver RM, Landon MB, Rouse DJ, et al. Maternal morbidity associated with multiple repeat cesarean deliveries. *Obstet Gynecol*. 2006;107:1226-1232.
53. Lim KI, Butt K, Naud K, Smithies M. Amniotic fluid: technical update on physiology and measurement. *J Obstet Gynaecol Can*. 2017;39(1):52-58.
54. Moore TR. Amniotic fluid dynamics reflect fetal and maternal health and disease. *Obstet Gynecol*. 2010;116(3):759-765.
55. Nabhan AF, Abdelmoula YA. Amniotic fluid index versus single deepest vertical pocket as a screening test for preventing adverse pregnancy outcome. *Cochrane Database Syst Rev*. 2008;3:CD006593. doi:10.1002/14651858.CD006593.pub2.
56. Pri Paz S, Khalek N, Fuchs KM, Simpson LL. Maximal amniotic fluid index as a prognostic factor in pregnancies complicated by polyhydramnios. *Ultrasound Obstet Gynecol*. 2012;39(6):648-653.
57. Sagi-Dain L, Sagi S. Chromosomal aberrations in idiopathic polyhydramnios: a systematic review and meta-analysis. *Eur J Med Genet*. 2015;58(8):409-415.
58. Kozinszky Z, Sikovanyecz J, Pásztor N. Severe midtrimester oligohydramnios: treatment strategies. *Curr Opin Obstet*

Gynecol. 2014;26(2):67-76.
59. Rossi AC, Prefumo F. Perinatal outcomes of isolated oligohydramnios at term and post-term pregnancy: a systematic review of literature with meta-analysis. *Eur J Obstet Gynecol Reprod Biol*. 2013;169(2):149-154.
60. Berghalla V, Mackeen AD. Cervical length screening with ultrasound-indicated cerclage compared with history-indicated cerclage for prevention of preterm birth. *Obstet Gynecol*. 2011;118(1):148-155.
61. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 142: cerclage for the management of cervical insufficiency. *Obstet Gynecol*. 2014;123:372-379. [Reaffirmed 2016].
62. Slager J, Lynne S. Assessment of cervical length and the relationship between short cervix and preterm birth. *J Midwifery Womens Health*. 2012;57:S4-S11.
63. Berghella V, Roman A, Daskalakis C, Ness A, Baxter JK. Gestational age at cervical length measurement and incidence of preterm birth. *Obstet Gynecol*. 2007;110:311-317.
64. Society for Maternal-Fetal Medicine Publications Committee, Berghella V. Progesterone and preterm birth prevention: Translating clinical trials data into clinical practice. *Am J Obstet Gynecol*. 2012;206(5):376-386.
65. Society for Maternal-Fetal Medicine; McIntosh J, Feltovich H, Berghella V, Manuck T. Society for Maternal-Fetal Medicine Consult Series #40: the role of routine cervical length screening in selected high- and low-risk women for preterm birth prevention. 2016. *Am J Obstet Gynecol*. 2016;15(3):B2-B7.
66. Chan YY, Jayaprakasan K, Tan A, Thornton JG, Coomarasamy A, Raine-Fenning NJ. Reproductive outcomes in women with congenital uterine anomalies: a systematic review. *Ultrasound Obstet Gynecol*. 2011;38(4):371-382.
67. Venetis CA, Papadopoulos SP, Campo R, Gordts S, Tarlatzis BC, Grimbizis GF. Clinical implications of congenital uterine anomalies: a meta-analysis of comparative studies. *Reprod Biomed Online*. 2014;29(6):665-683.
68. Martin JA, Hamilton BE, Osterman MJK, Driscoll AK, Mathews TJ. Births: final data for 2015. *National Vital Statistics Report*. 2017;66(1):1-68.
69. Heron M. Deaths: leading causes for 2014. *National Vital Statistics Report*. 2016;65(5):1-25. Available at: https://www.cdc.gov/nchs/data/nvsr/nvsr65/nvsr65_05.pdf. Accessed November 30, 2017.
70. Manuck TA. Racial and ethnic differences in preterm birth: a complex, multifactorial problem. *Semin Perinatol*. 2017;41(8):511-518.
71. Ding XX, Wu YL, Xu SJ, et al. Maternal anxiety during pregnancy and adverse birth outcomes: a systematic review and meta-analysis of prospective cohort studies. *J Affect Disord*. 2014;159:103-110.
72. Yonkers KA, Smith MV, Forray A, et al. Pregnant women with posttraumatic stress disorder and risk of preterm birth. *JAMA Psych*. 2014;71(8):897-904.
73. Goldenberg RL, Hauth JC, Andrews WW. Intrauterine infection and preterm delivery. *N Engl J Med*. 2000; 342:1500-1509.
74. Han CS, Schatz F, Lockwood CJ. Abruption-associated prematurity. *Clin Perinatol*. 2011;38(3):407-421.
75. Anum EA, Springel EH, Shriver MD, Strauss JF 3rd. Genetic contributions to disparities in preterm birth. *Pediatr Res*. 2009;65:1-9.
76. Zhang G, Feenstra B, Bacelis JL, et al. Genetic associations with gestational duration and preterm birth. *N Engl J Med*. 2017;377(12):1156-1167.
77. Iams JD, Goldenberg RL, Mercer BM, et al. The Preterm Prediction Study: can low risk women destined for spontaneous preterm birth be identified? *Am J Obstet Gynecol*. 2001;184:652-655.
78. Adams MM, Elam-Evans LD, Wilson HG, Gilbertz DA. Rates and factors associated with recurrence of preterm delivery. *JAMA*. 2000;283:1591-1596.
79. Mercer BM, Goldenberg RL, Das A, et al. The Preterm Prediction Study: a clinical risk assessment system. *Am J Obstet Gynecol*. 1996;174:1885-1893.
80. Muglia LJ. The enigma of spontaneous preterm birth. *N Engl J Med*. 2010;362:529-535.
81. Dekker GA, Lee SY, North RA, McCowan LM, Simpson NA, Roberts CT. Risk factors for a preterm birth in an international prospective cohort of nulliparous women. *PLoS One*. 2012;7(7):e39154.
82. Ward K, Argyle VA, Meade M, Nelson L. The heritability of preterm delivery. *Obstet Gynecol*. 2005;106(6):1235-1239.
83. Manns-James L. Bacterial vaginosis and preterm birth. *J Midwifery Womens Health*. 2011;56(6):575-583.
84. McManemy J, Cooke E, Amon E, Leet T. Recurrence risk for preterm delivery. *Am J Obstet Gynecol*. 2007;196:576.
85. Silver R, Bronwyn J, Guy RJ, Kaldor JM, Jamil MS, Rumbold A. *Trichomonas vaginalis* as a cause of perinatal morbidity: a systematic review and meta-analysis. *Sexually Transmit Dis*. 2014;41(6):369-376.
86. Folger AT. Maternal *Chlamydia trachomatis* infections and preterm birth: the impact of early detection and eradication during pregnancy. *Matern Child Health J. 2014;*18(8):1795-1802.
87. Iams JD, Newman RB, Thom EA, et al. Frequency of uterine contractions and the risk of spontaneous preterm delivery. *N Engl J Med*. 2002;346:250-255.
88. Chao TT, Bloom SL, Mitchell JS, McIntire DD, Leveno KJ. The diagnosis and natural history of false preterm labor. *Obstet Gynecol*. 2011;118:1301-1308.
89. Hedriana H, Byrne J, Campbell Bliss M, et al. *March of Dimes Preterm Labor Assessment Toolkit*. White Plains, NY:

March of Dimes; 2013.
90. Likis FE, Andrews JC, Woodworth AL, et al. *Progestogens for Prevention of Preterm Birth*. Comparative Effectiveness Review No. 74. AHRQ Publication No. 12-EHC105-EF. Rockville, MD: Agency for Healthcare Research and Quality; September 2012. Available at: https://effectivehealthcare.ahrq.gov/topics/progestogens-preterm-birth/research/. Accessed November 5, 2017.
91. Northern AT, Norman GS, Anderson K, et al.; National Institute of Child Health and Human Development (NICHD) Maternal–Fetal Medicine Units (MFMU). Network follow-up of children exposed in utero to 17**α**-hydroxyprogesterone caproate compared with placebo. *Obstet Gynecol*. 2007;110:865-872.
92. Norman JE, Marlow N, Messow C-M, et al. Vaginal progesterone prophylaxis for preterm birth (the OPPTIMUM study): a multicentre, randomised, double-blind trial. *Lancet*. 2016;387(10033):2106-2116.
93. Klebanoff MA, Carey JC, Hauth JC, et al. Failure of metronidazole to prevent preterm delivery among pregnant women with asymptomatic *Trichomonas vaginalis* infection. *N Engl J Med*. 2001;345:487.
94. Schwendicke F, Karimbux N, Allareddy V, Gluud C. Periodontal treatment for preventing adverse pregnancy outcomes: a meta- and trial sequential analysis. *PLoS One*. 2015;10:e0129060.
95. Cunnington M, Korsalioudaki C, Heath P. Genitourinary pathogens and preterm birth. *Curr Opin Infect Dis*. 2013;26:219-230.
96. Waters TP, Mercer B. Preterm PROM: prediction, prevention, principles. *Clin Obstet Gynecol*. 2011;54:307-312.
97. American College of Obstetricians and Gynecologists. Practice Bulletin No. 146: management of late term and postterm pregnancies. *Obstet Gynecol*. 2014;124:182-192. [Reaffirmed 2016].
98. American College of Obstetricians and Gynecologists, Society for Maternal-Fetal Medicine. ACOG Committee Opinion No. 579: definition of term pregnancy. *Obstet Gynecol*. 2013;122(5):1139-1140. [Reaffirmed 2016].
99. Norwitz ER, Snegovskikh VV, Caughey AB. Prolonged pregnancy: when should we intervene? *Clin Obstet Gynecol*. 2007;50:547-557.
100. Caughey AB, Stotland NE, Washington E, Escobar GJ. Maternal and obstetrical complications of pregnancy are associated with increasing gestational age at term. *Am J Obstet Gynecol*. 2007;196:155.e1-155.e6.
101. Alexander JM, McIntire DD, Leveno KJ. Forty weeks and beyond: pregnancy outcomes by week of gestation. *Obstet Gynecol*. 2000;96:291-294.
102. Rosenstein MG, Cheng YW, Snowden JM, Nicholson JM, Caughey AB. Risk of stillbirth and infant death stratified by gestational age. *Obstet Gynecol*. 2012;120(1):76-82.
103. Gülmezoglu AM, Crowther CA, Middleton P, Heatley E. Induction of labour for improving birth outcomes for women at or beyond term. *Cochrane Database Syst Rev*. 2012;6:CD004945. doi:10.1002/14651858.CD004945.pub3.
104. Boulvain M, Stan CM, Irion O. Membrane sweeping for induction of labour. *Cochrane Database Syst Rev*. 2005;1:CD000451. doi:10.1002/14651858.CD000451.pub2.
105. Warshauer E, Mercurio M. Update on dermatoses of pregnancy. *Int J Dermatol*. 2013;52(1):6-13.
106. Mehta N, Chen KK, Kroumpouzos G. Skin disease in pregnancy: the approach of the obstetric medicine physician. *Clin Derm*. 2016;34:320-326.
107. Bechtel MA, Plotner A. Dermatoses of pregnancy. *Clin Obstet Gynecol*. 2015;58(1):104-111.
108. Bruce K, Watson S. Management of intrahepatic cholestasis of pregnancy: a case report. *J Midwifery Womens Health*. 2007;52(1):68-72.
109. Brzoza Z, Kasperska A, Oles E, Rogala B. Pruritic urticarial papules and plaques of pregnancy. *J Midwifery Womens Health*. 2007;52:44-48.
110. Harper MA, Meyer RE, Berg CJ. Peripartum cardiomyopathy: population-based birth prevalence and 7-year mortality. *Obstet Gynecol*. 2012;120(5):1013-1019.
111. Dekker RL, Morton CH, Singleton P, Lyndon A. Women's experiences being diagnosed with peripartum cardiomyopathy: a qualitative study. *J Midwifery Womens Health*. 2016;61:467-473.
112. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 75: management of alloimmunization during pregnancy. *Obstet Gynecol*. 2006;108:457.
113. Committee on Practice Bulletins–Obstetrics. Practice Bulletin No. 181: prevention of RH D alloimmunization. *Obstet Gynecol*. 2017;130:e57-e70.
114. De Haas, M, Thurik FF, Koelewijn JM, van der School CE. Haemolytic disease of the fetus and newborn. *VOX Sanguinis*. 2015;109:99-103.
115. Moise KJ. Fetal anemia due to non-Rhesus-D red-cell alloimmunization. *Semin Fetal Neonatal Med*. 2008;13(4):207-214.
116. Sedgh G, Finer LB, Bankole A, Eilers MA, Singh S. Adolescent pregnancy, birth, and abortion rates across countries: levels and recent trends. *J Adolesc Health*. 2015;56(2):223-230.
117. Mathews TJ. Mean age of mothers is on the rise: United States 2000–2014. *NCH Data Brief*. 2016;232:1-8.
118. Leftwich HK, Alves MV. Adolescent pregnancy. *Pediatr Clin North Am*. 2017;64(2):381-388.
119. Kawakita T, Wilson K, Grantz KL, Landy HJ, Huang CC, Gomez-Lobo V. Adverse maternal and neonatal outcomes in adolescent pregnancy. *J Pediatr Adolesc Gynecol*. 2016;29(2):130-136.

120. Maradiegue A. Minor's rights versus parental rights: review of legal issues in adolescent health care. *J Midwifery Womens Health*. 2003;48(3):170-177.
121. Marvin-Dowle K, Burley VJ, Soltani H. Nutrient intakes and nutritional biomarkers in pregnant adolescents: a systematic review of studies in developed countries. *BMC Pregn Childbirth*. 2016;16:268.
122. Felder JN, Epel E, Lewis JB, et al. Depressive symptoms and gestational length among pregnant adolescents: cluster randomized control trial of CenteringPregnancy plus group prenatal care. *J Consult Clin Psychol*. 2017;85(6):574-584.
123. Mills TA, Lavender T. Advanced maternal age. *Obstet Gynaecol Reprod Med*. 2014;24(3):85-90.
124. Stern JE, Luke B, Tobias M, Gopal D, Hornstein MD, Diop H. Adverse pregnancy and birth outcomes associated with underlying diagnosis with and without assisted reproductive technology treatment. *Fertil Steril*. 2015;103(6):1438-1445.
125. Davies MJ, Moore VM, Willson KJ, et al. Reproductive technologies and the risk of birth defects. *N Engl J Med*. 2012;366(19):1803-1813.
126. Goodman D, Danel I. Female genital mutilation/cutting in the United States: updated estimates of women and girls at risk, 2012. *Public Health Rep*. 2016;131(2):340-347.
127. Jacoby SD, Smith A. Increasing certified nurse-midwives' confidence in managing the obstetric care of women with female genital mutilation/cutting. *J Midwifery Womens Health*. 2013;58(4):451-456.
128. Finnegan L. Licit and illicit drug use during pregnancy: maternal, neonatal, and early childhood consequences. Canadian Centre on Substance Abuse. 2013. Available at: http://www.ccsa.ca/Resource%20Library/CCSA-Drug-Use-during-Pregnancy-Report-2013-en.pdf. Accessed October 6, 2017.
129. Wright TE, Terplan M, Ondersma SJ et al. The role of screening, brief intervention, and referral to treatment in the perinatal period. *Am J Obstet Gynecol*. 2016;215(5):539-547.
130. MacDorman MF, Gregory EC. Fetal and perinatal mortality: United States, 2013. *Natl Vital Stat Rep*. 2015;64:1.
131. Stillbirth Collaborative Research Network Writing Group. Causes of death among stillbirths. *JAMA*. 2011;306(22):2459-2468.
132. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 102: management of stillbirth. *Obstet Gynecol*. 2009;113:748-761.
133. National Institutes of Health. NIH Consensus Development Conference on Vaginal Birth After Cesarean: new insights. Final panel statement. Available at: http://consensus.nih.gov/2010/vbacstatement.htm. Accessed October 17, 2011.
134. Guise J-M, Eden K, Emeis C, Denman MA, Marshall N, Fu R, et al. Vaginal birth after cesarean: new insights. Vol. No. 10-E001. Rockville, MD: Agency for Healthcare Research and Quality; 2010.
135. American College of Nurse-Midwives. Clinical Bulletin No. 12: care for women desiring a vaginal birth after cesarean. *J Midwifery Womens Health*. 2011;56:517-525.
136. Cox K. Cousneling women with a previous cesarean birth: toward a shared-decision making partnership. *J Midwifery Womens Health*. 2014;59:237-245.
137. Bovbjerg ML, Cheyney M, Brown J, Cox KJ, Leeman L. Perspectives on risk: assessment of risk profiles and outcomes among women planning community birth in the United States. *Birth*. 2017;44(3):209-221.
138. Tilden EL, Cheyney M, Guise JM, et al. Vaginal birth after cesarean: neonatal outcomes and United States birth setting. *Am J Obstet Gynecol*. 2017;216(4):403.e1-403.e8.
139. Carreau AM, Nadeau M, Marceau S, Marceau P, Weisnagel SJ. Pregnancy after bariatric surgery: balancing risks and benefits. *Can J Diabetes*. 2017;41(4):432-438.
140. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 105: bariatric surgery and pregnancy. *Obstet Gynecol*. 2009;113(6):1405–1413.
141. Parker MH, Berghella V, Niijar J. Bariatric surgery and associated adverse pregnancy outcomes among obese women. *J Matern Fetal Neonatal Med*. 2016; 29(11):1747-1750.
142. Vrebosch L, Bel S, Vansant G, Guelinckx I, Devlieger R. (2012). Maternal and neonatal outcomes after laparoscopic adjustable gastric banding: a systematic review. *Obesity Surgery*. 2012; 22(10):1568–1579.
143. Oyelese Y, Vintzileos AM. The use and limitations of the fetal biophysical profile. *Clin Perinatol*. 2011;38:47-64.
144. Manning FA. Antepartum fetal testing: a critical appraisal. *Curr Opin Obstet Gynecol*. 2009;21:348-352.
145. American College of Obstetricians and Gynecologists. Practice Bulletin No. 145: antepartum fetal surveillance. *Obstet Gynecol*. 2014;124:182-192.
146. Hijazi ZR, East CE. Factors affecting maternal perception of fetal movement. *Obstet Gynecol Surv*. 2009;64:489-494.
147. Mangesi L, Hofmeyr GJ, Smith V, Smyth RMD. Fetal movement counting for assessment of fetal wellbeing. *Cochrane Database Syst Rev*. 2015;10:CD004909. doi:10.1002/14651858.CD004909.pub3.
148. Signore C, Freeman RK, Spong CY. Antenatal testing: a reevaluation. Executive summary of a Eunice Kennedy Shriver National Institute of Child Health and Human Development Workshop. *Obstet Gynecol*. 2009;113(3):687-701.
149. Tan KH, Sabapathy A. Maternal glucose administration for facilitating tests of fetal wellbeing. *Cochrane Database Syst Rev*. 2012;9:CD003397. doi:10.1002/14651858.CD003397.pub2.
150. Paine LL, Johnson TR, Turner MH, Payton RG. Auscultated fetal heart rate accelerations: part II. An alternative to the nonstress test. *J Nurse-Midwifery*. 1989;31(2):73-77.

APPENDIX

22A

Auscultated Acceleration Test

TEKOA L. KING

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The auscultated acceleration test (AAT) detects fetal accelerations using a specific counting method via use of an Allen or Doppler fetoscope.**1** The procedure for conducting an AAT should be performed by two clinicians; one auscultates and reports the fetal heart rate (FHR) count, and the other records the findings.[2-6]

Background

FHR accelerations of 15 bpm or more above the baseline that last for 15 seconds or more from onset to return to the baseline are a reliable indicator of fetal oxygenation. When two accelerations are noted in a 20-minute FHR recording, the chance of stillbirth within the next 7 days is less than 1% (the false-negative rate is 0.3%).[7]

Mendenhall et al. evaluated the efficacy of a single acceleration (n = 1005 AAT tests, 367 women) and found the single acceleration was associated with no perinatal losses. The nonreactive AAT in this series predicted the 4 stillbirths that occurred in the cohort.[8] Other authors have shown that a clinician listening to an FHR recording will detect 77.7% of all FHR accelerations that are 15 bpm or more above baseline and 94% of the nonreactive FHR recordings.[9]

In settings where electronic fetal monitoring is not available or chosen for use, the AAT is a reliable and valid method of detecting fetal accelerations.

Procedure

- Place the woman in a semi-recumbent, comfortable position.
- Auscultate the FHR for a maximum of 6 minutes.
- Count every other 5-second interval.

- Record each 5-second count on the AAT graph (**Figure 22A-1** and **Figure 22A-2**).

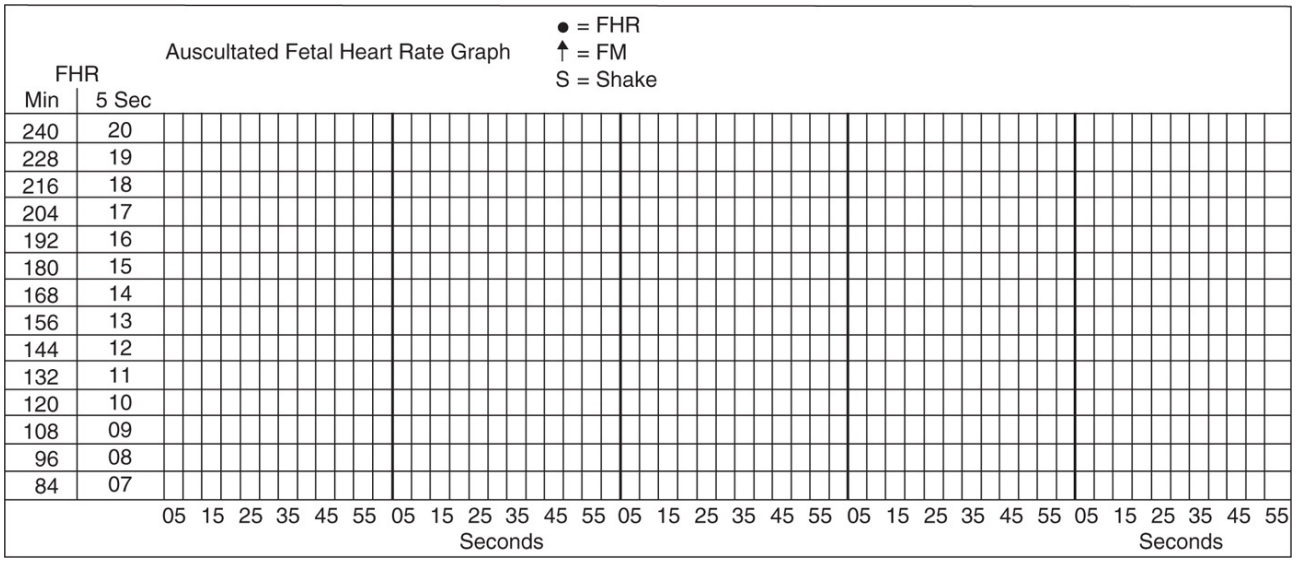


Figure 22A-1 The AAT graph used to document the FHR pattern.

Modified with permission of Lisa L. Paine from Paine LL, Payton RG, Johnson TR. Auscultated fetal heart rate and accelerations: part I. Accuracy and documentation. *J Nurs Midwifery*. 1986;31(2):68-72.[2]

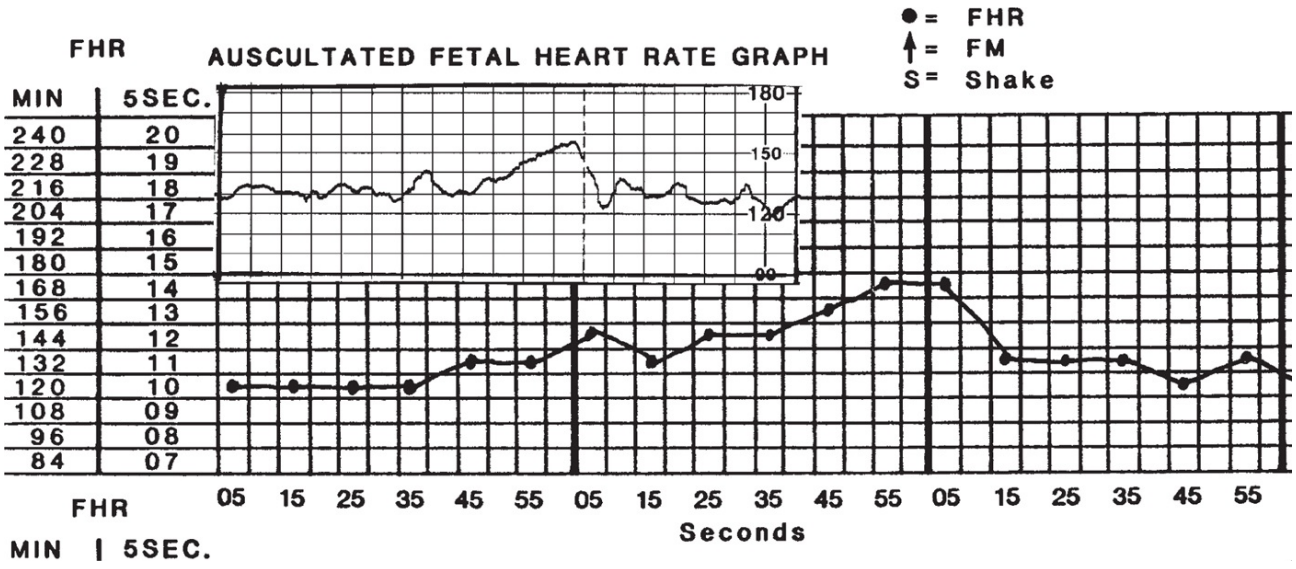


Figure 22A-2 AAT with inset of a nonstress test completed simultaneously.

Reproduced with permission of Lisa L. Paine, who first developed the AAT with colleagues at Malcolm Grow Medical Center, Andrews AFB, Maryland, and later studied its validity and refinement at the Johns Hopkins Hospital in Baltimore, Maryland, with funding from the NIH National Center for Nursing Research Grant No. R-01-NR-01705-01.

- If an acceleration of 2 bpm in a 5-second counting interval is noted in conjunction with fetal movement (FM), the test can be stopped.
- If no acceleration is noted after 3 minutes, the maternal abdomen can be gently shaken in an attempt to waken the fetus or elicit a more active state. The clinician grasps the fetal head and buttocks and slowly moves the fetus slightly from side to side in a gentle

shaking motion for 5 seconds.
- The FHR auscultation procedure is again repeated for a maximum of 2 to 3 minutes.
- Identify baseline FHR and any accelerations by plotting the numbers obtained on the AAT chart (**Figure 22A-1** and **Figure 22A-2**).

Interpretation

Reactive Nonstress Test (NST)

- An acceleration is present when the FHR is up by two grid points (2 bpm in a 5-second period).
- A single FHR acceleration indicates reactivity.[8]

Unsatisfactory

- No acceleration and no fetal movement after fetal stimulation are noted.

Nonreactive

- No accelerations are noted.

References

1. Gegor CL, Paine LL, Johnson TRB. Antepartum fetal assessment: a nurse-midwifery perspective. *J Nurse-Midwifery.* 1991;36(3):153-167.
2. Paine LL, Payton RG, Johnson TRB. Auscultated fetal heart rate accelerations: part I. Accuracy and documentation. *J Nurse-Midwifery.* 1986;31(2):68-72.
3. Paine LL, Johnson TR, Turner MH, Payton RG. Auscultated fetal heart rate accelerations: part II. An alternative to the nonstress test. *J Nurse-Midwifery.* 1989;31(2):73-77.
4. Paine LL, Benedict MI, Strobino DM, Gegor CL, Larson EL. A comparison of the auscultated acceleration test and the nonstress test as predictors of perinatal outcomes. *Nurs Res.* 1992;41(2):87-91.
5. Daniels SM, Boehm N. Auscultated fetal heart rate accelerations: an alternative to the nonstress test. *J Nurse-Midwifery*. 1991;36(2):88-94.
6. Paine LL, Zanardi RL, Johnson TR, Rorie JA, Barger MK. A comparison of two time intervals for the auscultated acceleration test. *J Midwifery Womens Health*. 2001;46(2):98-102.
7. Signore C, Freeman RK, Spong CY. Antenatal testing: a reevaluation. Executive summary of a Eunice Kennedy Shriver National Institute of Child Health and Human Development Workshop. *Obstet Gynecol.* 2009;113(3):687-701.
8. Mendenhall HW, O'Leary J, Phillips K. The nonstress test: the value of a single acceleration in evaluating the fetus at risk. *Am J Obstet Gynecol.* 1980;136:87: 244-246.
9. Baskett TF, Boyce CD, Lohre MA, Manning FA. Simplified antepartum fetal heart assessment. *Br J Obstet Gynaecol.* 1981;88(4):395-397.

CHAPTER

23

Medical Complications in Pregnancy

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Introduction

This chapter reviews medical complications that may occur during pregnancy. Medical disorders may develop for the first time during pregnancy. Preexisting medical disorders may worsen, be unaffected, improve, or have a variable course during pregnancy, particularly because the physiologic changes of pregnancy can alter the course of many illnesses. Medical complications in pregnant women are best cared for by a collaborative, team-based care or by physician referral. Midwives practice in a wide variety of settings with different resources. The model of collaborative care provides a powerful interdisciplinary consensus structure for delivering this kind of practice.[1] Ideally, care models should be flexible, dynamic, and responsive to the unique needs of the individual woman and family. In all circumstances, the optimal care of the woman and her fetus will drive care decisions.

Midwives may be the first healthcare provider to see a woman who has increased risks for adverse perinatal outcomes secondary to a medical disorder, including individuals who present for preconceptional counseling. Identification of risks and consultation, when indicated, is an essential component of midwifery care. Thus, midwives need the knowledge necessary to identify perinatal risks so that they can obtain a thorough history, consult with the appropriate specialist, and initiate evaluations when required.

Midwives also often identify social determinants of care and provide ancillary and supportive care for women with increased risks of poor outcomes. For women with complex medical disorders, prenatal visits can involve many tests and discussions of possible adverse outcomes. Midwifery visits can help a woman who has a high-risk pregnancy in several ways. For example, a midwife visit may focus on the “normal” aspects of her pregnancy and thereby help the woman preserve her sense of well-being. The midwife may also participate in education and support for the extra care required to treat a medical complication.[1]

This chapter does not include a comprehensive review of all possible medical conditions that can adversely impact the course of pregnancy. Instead, a review of commonly encountered problems is provided as exemplars of the approach one takes in caring for a woman with any preexisting medical disorder. For the convenience of the readers, these conditions are generally presented in an alphabetical order rather than incidence of occurrence.

Autoimmune Disorders in Pregnancy

Autoimmune disorders are a broad classification of disorders in which aberrations in the immune response result in the immune system attacking the individual's healthy body tissue. Examples include systemic lupus erythematosus (SLE), multiple sclerosis, rheumatoid arthritis, and irritable bowel syndrome. During pregnancy, the immune system changes in response to accommodation of the fetal immune system. Because of pregnancy, some autoimmune disorders (e.g., rheumatoid arthritis, multiple sclerosis) improve during pregnancy. For others, pregnancy has no effect on the progress of the disorder but the disorder or treatment can have adverse effects on the course of pregnancy (e.g., SLE). Treatment of autoimmune disorders focuses on suppressing abnormal immune function, and a similar group of medications is used to treat many of these disorders. SLE is presented as a common exemplar of the topics of import involved in caring for a woman with different autoimmune disorders during pregnancy.

Systematic Lupus Erythematosus

Systemic lupus erythematosus is an autoimmune connective tissue disorder involving multiple body systems. The pathogenesis of SLE occurs secondary to an immune dysregulation of B cells that results in abnormally increased production of antibodies to nuclear antigens and other self-antigens not usually identified by the immune system as "abnormal." The target antigens for these autoantibodies are membranes, intracellular material, or nuclear material. SLE is more common among women than among men, and appears primarily during the reproductive years. African Americans and Asians have a higher prevalence than Caucasians.[2] The nature of the disease is highly variable, characterized by multiple symptoms, periodic remissions and flare-ups, and differing patterns of progression and prognosis.

Diagnostic criteria for SLE include having four or more of the following signs and symptoms: antinuclear antibodies, lupus antibodies, hemolytic anemia or thrombocytopenia, seizures or psychosis, persistent proteinuria, pleuritic or pericarditis, arthritis in two or more joints, oral pharyngeal ulceration, photosensitivity, discoid lupus, or "butterfly" rash on the face.[3] Some of these symptoms mimic findings associated with normal pregnancy changes, such as malar erythema (cholasma of pregnancy), anemia, and mild thrombocytopenia—which makes detection of an SLE flare-up in a pregnant woman challenging.[3]

Several medications from different drug classes are used to help women manage SLE. Some are safe for the woman and the fetus, some do not affect the fetus, and some are contraindicated. Thus, a thorough review of current medications is one of the initial assessments to be made in a woman with SLE, or other autoimmune disorder.

SLE does not significantly affect a woman's fertility, but pregnancy may increase disease activity and, depending on the systems affected, can lead to serious complications for both the woman and her fetus. One meta-analysis of 37 studies on SLE in pregnancy reported increased maternal risks of lupus flare (25.6%), hypertension (16.3%), nephritis (16.1%), preeclampsia (7.6%), and eclampsia (0.8%). Women with SLE are also at increased risk for thrombophilia, thromboembolism, and thrombocytopenia. With the added SLE activity that can occur in pregnancy, some women with lupus nephritis may experience accelerated progress toward

end-stage renal disease.[2]

Potential pregnancy complications include miscarriage (16.0%), intrauterine growth restriction (12.7%), stillbirth (3.6%), and neonatal death (2.5%).[2] Neonatal lupus is an autoimmune disorder acquired by the fetus secondary to passive transfer across the placenta of maternal autoantibodies; these autoantibodies primarily attack the fetus's skin and heart. Congenital heart block occurs is approximately 2% of fetuses in women with anti-Ro/SSA or anti-La/SSB antibodies. Risks for all complications increase if the woman has lupus nephritis and antiphospholipid, anti-Ro/SSA, or anti-La/SSB antibodies.[1]

Management of Women with Systematic Lupus Erythematosus

The best pregnancy outcomes occur among women who have not had a lupus flare for 6 or more months preceding pregnancy.[3] Women who are having a lupus flare are best managed in a setting that has access to providers of high-risk obstetrical care and subspecialists. Monitoring during pregnancy includes baseline and serial laboratory tests to assess for SLE flare, serial ultrasonography, weekly biophysical profile in the third trimester, and fetal echocardiography for women who have anti-Ro/SSA or anti-LA/SSB antibodies. Women are followed closely for disease exacerbation, indications of thrombotic events, and hypertensive disorders.

Treatment with hydroxychloroquine (Plaquenil) that was started before conception is continued throughout the pregnancy. When antiphospholipid antibodies are present, prophylactic aspirin or low-molecular-weight heparin is indicated to help prevent fetal loss. In the absence of any signs or symptoms of active SLE, affected women usually require no specific treatment during pregnancy. Additional medications are indicated for women who experience an SLE flare during pregnancy.[3]

SLE provides a good example of how the midwife can collaborate in the care of a woman with a high-risk medical condition during pregnancy. The first step is to conduct a thorough history and identify any immediate concerning symptoms, such as hypertension. The midwife should then review the woman's list of current medications to determine whether any are contraindicated or of concern for use in pregnancy. Women with significant risks are referred to physician care for urgent evaluation. For women who have SLE but no immediate risks, physician consultation is obtained to identify tests or laboratory evaluations that need to be scheduled and the appropriate timing for a physician visit. Once the woman is under the care of the appropriate obstetric specialist, the midwife can participate in conducting subsequent prenatal visits as indicated. When a woman with SLE is seen for a prenatal visit by a midwife, accurate assessment of blood pressure and urinalysis are essential, as these tests will help detect the onset of a lupus flare. The fetal heart rate should be counted and the number of beats per minute recorded in the chart at each visit. Breastfeeding is not contraindicated for a woman with SLE and is discussed as usual prenatally.

Diabetes

Approximately 6% to7% of women are affected by diabetes mellitus (DM) during pregnancy. The three types of diabetes are type 1, type 2, and gestational diabetes mellitus (GDM). Type 1 and type 2 diabetes are collectively termed *pregestational diabetes mellitus* (PGDM).[4-6] A fourth syndrome, *prediabetes*, is defined as a high blood glucose level in a person who does not have all the criteria for the diagnosis of diabetes; such an individual is at increased risk for developing diabetes.

Gestational diabetes and type 2 diabetes are diseases of insulin resistance, whereas type 1 diabetes is characterized by beta cells in the pancreas that produce little or no insulin. GDM is further subcategorized as A1GDM (diet changes result in euglycemia) and A2GDM (medication is needed to maintain euglycemia). In the United States, PGDM and GDM have a prevalence of 0.82% and 5.34%, respectively.[7] The incidence of type 2 diabetes before pregnancy and the incidence of GDM are increasing in parallel with the rising rates of obesity in this country.[4] The prevalence of GDM is higher among Hispanic, African American, Native American, Asian, and Pacific Islander women than Caucasian women.

Overall, approximately 29.3% of women in the United States have prediabetes.[8] This condition is associated with obesity as well as dyslipidemia including high triglycerides and/or low high-density lipoprotein (HDL) cholesterol.[5] Research has found that lifestyle intervention and appropriate treatment of prediabetes in pregnancy can significantly lower the associated risks of prediabetes with preeclampsia, macrosomia, shoulder dystocia, and cesarean birth.[6]

Women with diabetes have an increased risk of several pregnancy complications, including spontaneous abortion, preterm birth, preeclampsia, and cesarean section. Risks to the fetus include fetal anomalies, intrauterine fetal demise, neonatal jaundice, hypoglycemia, hyperbilirubinemia, and shoulder dystocia. Adverse effects are more likely with increasing blood glucose levels and longer duration of elevated blood glucose levels.

Historically, gestational diabetes was thought to be a condition unique to pregnancy that increased the risk of having a large infant but resolved after the woman gave birth. The multinational Hyperglycemia and Pregnancy Outcomes (HAPO) trial published in 2008 clarified the issue. This trial placed women (n = 23,316) into three categories based on the degree of hyperglycemia found in fasting and postprandial blood glucose levels. The HAPO trial identified a strong, significant increase in several adverse pregnancy outcomes as maternal glucose levels rose, including macrosomia, shoulder dystocia, cesarean section, preeclampsia, preterm birth, hyperbilirubinemia, polycythemia, neonatal hypoglycemia, and neonatal intensive care admission.[9] Subsequent studies have also shown that women with gestational diabetes are at increased risk for developing type 2 diabetes and their infants are at increased risk for childhood obesity, type 2 diabetes, and metabolic syndrome.[10-12] Treatment of an individual with GDM, however, reduces the risk of adverse outcomes.[13-15]

Although GDM has historically been considered a pregnancy-related complication that is unique to pregnancy, today it is evident that the outcomes, and treatments of women with all types of diabetes are directly related to the severity of glucose dysregulation. Therefore, it is

useful to consider this group of diseases together.

Pathophysiology of Diabetes During Pregnancy

The dynamics of glucose metabolism in pregnancy evolve from early to late pregnancy, as insulin resistance gradually increases and peaks in the late second to third trimester. As the placenta grows, levels of human placental lactogen (hPL) and other diabetogenic hormones increase. These hormones increase cellular resistance to insulin, which results in higher blood glucose levels. In most women, the pancreas can increase the production of insulin to counterbalance the insulin resistance effect of placental hormones. Overt hyperglycemia occurs when the pancreas is unable to produce adequate insulin. The peak effect of hPL occurs around 26 to 28 weeks of pregnancy, which is the recommended window for screening for GDM. Pre-pregnant insulin sensitivity returns in the postpartum period. Optimal care for women with diabetes in pregnancy is responsive to these dynamic changes.[16]

The metabolic milieu of diabetes increases the risk for endothelial dysfunction, which in turn can result in impaired placental growth and fetal growth restriction. In contrast, hyperglycemia and hyperinsulinemia can stimulate hypergrowth in both the placenta and the fetus. Thus, the fetus of a woman with diabetes is at increased risk for fetal growth restriction or macrosomia depending on the degree of hyperglycemia, placental response, and presence of absence of other comorbidities that can adversely affect placental development. As increasing amounts of glucose cross the placenta, the fetus must increase production of insulin to metabolize the increased glucose. This response causes dramatic growth in the fetus, characterized by both hyperplasia and hypertrophy that can result in macrosomia. The result is a change in fetal physiology that can affect the individual throughout life.[6]

Women with GDM have increased risks for the same pregnancy complications as women with PGDM, but their risk is somewhat lower because the degree of hyperglycemia in women with GDM is reduced and the duration of hyperglycemia is shorter compared to women with PGDM. For women with GDM, the critical risk is emergence of type 2 diabetes later in life. Approximately 70% of women with GDM will develop type 2 diabetes within 22 to 28 years after pregnancy.[4] In the United States, approximately 60% of Latina women with GDM develop type 2 diabetes within 5 years after pregnancy.[4]

Diagnosis of Diabetes

The diagnosis of diabetes in nonpregnant adults is reviewed in the *Common Conditions in Primary Care* chapter. However, some women who are given the diagnosis of gestational diabetes actually have undiagnosed PGDM.[6] To clarify the distinction between pregestational type 2 diabetes and gestational diabetes, the American Diabetes Association (ADA) defines GDM as “diabetes diagnosed in the second or third trimester of pregnancy that is not clearly type 1 or 2 diabetes.” Diabetes that is initially detected in the first trimester should be considered pregestational type 2 diabetes.[5] Screening for diabetes during pregnancy is reviewed in the *Prenatal Care* chapter. Diagnosis and prenatal care for women with all types of diabetes is presented in this chapter.

To review, testing for diabetes in pregnancy includes an early assessment for PGDM and a later assessment for GDM:

- *First trimester test for PGDM in women with risk factors.* A hemoglobin A1c level (HbA1c) of 6.5% or higher is diagnostic for diabetes in nonpregnant individuals, and this value is also used for diagnosis of PGDM during pregnancy. However, HbA1c levels are lower in pregnancy due to increased red blood cell turnover, and mild elevations (5.7% to 6.4%) are associated with an increased risk for developing GDM (27.3% versus 8.7%; odds ratio [OR], 3.9; 95% confidence interval [CI], 2.0–7.7).[17] Therefore, the specific HbA1c value used to indicate a need for further testing or closer surveillance during pregnancy is based on institutional guidelines and population-specific parameters. HbA1c is an indirect measure of glucose level, and a fasting glucose or 2-hour postprandial glucose value following a 75-gram glucose load may be recommended for direct measurement.
- *Screening for GDM via a one- or two-step process.* Diagnostic criteria are presented in **Table 23-1**.[5,6] Choice of the screening method is primarily based on local or regional guidelines.

Table 23-1 Diagnostic Criteria for Gestational Diabetes in Pregnancy

Test Strategy	Glucose Load	Fasting (mg/dL)	1-Hour Postprandial (mg/dL)	2-Hour Postprandial (mg/dL)	3-Hour Postprandial (mg/dL)	Diagnostic Criteria
One-step	75 gm	≥ 92	≥ 180	≥ 153	NA	≥ 1 abnormal value
Two-step						
a. Screening	50 gm	NA	≥ 140	NA	NA	NA
b. GTT						
NDDG criteria	100 gm	≥ 105	≥190	≥ 165	≥ 145	≥ 2 abnormal values
Carpenter-Coustan criteria	100 gm	≥ 95	≥ 180	≥ 155	≥ 140	≥ 2 abnormal values

Abbreviations: GTT, glucose tolerance test; NDDG, National Diabetes Data Group.

Note: Some institutions use a cut-off value of 130–135 mg/dL for the screening step. Either the NDDG or the Carpenter-Coustan cut-off values may be used.

Based on American Diabetes Association. Classification and diagnosis of diabetes. *Diab Care.* 2017;40(suppl 1):S11-S24[5]; Salmeen K. Gestational diabetes testing: making sense of the controversy. *J Midwifery Womens Health.* 2016;61(2):203-209.[6]

Management of Women with Pregestational Diabetes Mellitus

Table 23-2 presents an overview of prenatal care for women with diabetes. Women with PGDM are referred to a physician for pregnancy care. Midwives may participate in prenatal care visits as a member of a multidisciplinary team.[18] Depending on the type of diabetes, the

woman's health status, and the healthcare setting, care may include working with diabetes educators and physicians who are specialists in diabetes during pregnancy.

Table 23-2 Overview of Management of Pregestational and Gestational Diabetes

Management	PGDM	GDM	Clinical Considerations
Eye examination	X		First trimester; monitor each trimester, then every 3 months to 1 year
Assessment of renal function	X		First trimester
Four-chamber view of fetal heart including outflow tracts (fetal ultrasound and possible fetal echocardiography)	X		Increased risk for congenital heart defects
Ultrasound for fetal growth, amniotic fluid volume	X		28, 32, and 36 weeks depending on clinical situation to assess for fetal growth restriction, polyhydramnios, and macrosomia
Nutrition therapy	X	X	All women with diabetes manage blood glucose levels via diet; many women with GDM achieve euglycemia with diet changes alone
Glucose monitoring: fasting and postprandial	X	X	Some women with PGDM may benefit from preprandial testing
Glucose monitoring: measure HbA1c	X		Target HbA1c is less than 6%
Medication: Insulin, metformin (Glucophage), or glyburide (Micronase)[a] may be used	X	X	Effectiveness of oral medications is the same as that for insulin; data on long-term effects are lacking
If chronic hypertension coexists with diabetes	X	X	Target blood pressure is 120–160 mm Hg systolic and 80–105 mm Hg diastolic
If EFW ≥ 4500 gm, counseling regarding option of cesarean delivery		X	
Postpartum oral GTT 6–12 weeks after birth		X	
Lifelong screening every 3 years for diabetes and prediabetes		X	

Abbreviations: EFW, estimated fetal weight; GDM, gestational diabetes mellitus; GTT, glucose tolerance test; HbA1c, hemoglobin A1c; PGDM, pregestational diabetes mellitus.

[a] Contraindicated for women with sulfa allergy.

Women with PGDM receive an initial evaluation of HbA1c, thyroid function, echocardiogram, eye examination to assess for retinopathy, and renal function.[5] The goal of care in the first trimester is to maintain euglycemia and avoid hypoglycemia. Care during the second and third trimesters additionally focuses on monitoring fetal growth and preventing stillbirth.[18] Overall, women with PGDM will be seen more frequently during pregnancy and blood glucose values monitored intensively via daily self-monitoring of preprandial and postprandial blood glucose values. Target blood glucose values and target HbA1c values are listed in **Table 23-3**. The lowest risk for congenital malformations exists when the diabetes is well controlled and HbA1c is less than 6.5%.[16]

Table 23-3 Target Blood Glucose Values During Pregnancy

Time of Test	Target Value (mg/dL)
Fasting	≤ 95
Preprandial	≤ 100
1-hour postprandial	≤ 140
2-hour postprandial	≤ 120
HgA1c	≤ 6%

Abbreviation: HbA1c, hemoglobin A1c.

Insulin remains the treatment of choice throughout pregnancy for women with type 1 diabetes. Close monitoring is needed, and adjustments in doses may be necessary, due to the increased dynamic changes in insulin sensitivity throughout pregnancy. Women with this condition are especially vulnerable to hypoglycemia in the first trimester, and ketoacidosis can occur at lower glucose levels than it would outside of pregnancy.[18] For women with type 1 diabetes, insulin requirements are often lower in the first trimester, gradually increase during the second trimester, and decrease again in the late third trimester and in the immediate postpartum period, paralleling the plasma levels of placental hormones that induce insulin resistance.

In women with type 2 diabetes, medication may involve changing from oral agents used before pregnancy to insulin so as to more closely control blood glucose values. Glycemic control in these women often requires higher doses of insulin than are required for women with type 1 diabetes. Combination therapy with an oral agent and insulin is also an option when diet, exercise, and oral agents do not provide sufficient control of glucose levels.[4],[18] However, it is important to remember that insulin does not cross the placenta, whereas oral agents do. The long-term safety of the latter medications has not been established.[5]

Monitoring during pregnancy is directed toward minimizing risks and managing comorbidities. Women with both types of PGDM have an increased risk for developing preeclampsia and, therefore, low-dose aspirin (81 mg) daily is recommended after 12 weeks' gestation. Ultrasounds for fetal growth are generally performed at 28 to 32 weeks. Women with any type of diabetes who require insulin or an antiglycemic oral medication to maintain euglycemia are usually offered antenatal testing twice weekly starting at approximately 32 weeks and asked to perform daily fetal movement counts.

Fetuses of women with diabetes develop respiratory maturity later than do fetuses of women without diabetes, so early delivery poses increased risks for respiratory distress. Nevertheless, the presence of comorbidities, fetal growth restriction, or poorly controlled glucose values may indicate a need for preterm induction of labor. If no complications are present, glucose values are stable, and fetal growth is normal, induction of labor is usually recommended by 39 to 40 weeks' gestation.

Management of Women with Gestational Diabetes Mellitus

Women with GDM can often attain euglycemia via lifestyle modification, improved diet, and exercise. Medication is used when adequate glycemic control is not achieved through these

changes.[18] Recent studies have demonstrated that insulin and oral medications have equivalent efficacy in pregnancy, and that either may be considered for first-line therapy.[4] Women with GDM who are able to maintain euglycemia via diet and exercise alone (Class A1GDM) do not appear to be at risk for stillbirth and are not usually referred for antenatal testing, although induction of labor is usually recommended at 39 to 40 weeks.[4] Diabetes is not a contraindication to breastfeeding. Postpartum care includes follow-up to assess for type 2 diabetes at 6 to 12 weeks postpartum. Lifelong screening for diabetes is recommended every three years.

Gastrointestinal Conditions

Diagnosis of gastrointestinal disorders in pregnancy can be difficult because many symptoms —such as nausea, vomiting, diarrhea, and abdominal discomfort—are prevalent during pregnancy. This section reviews serious gastrointestinal disorders that are common in pregnancy, including those that increase the risks of pregnancy complications and those that are adversely affected by pregnancy.

Acute Abdomen

The definition of acute abdomen is sudden severe abdominal pain. The differential diagnoses for women who present with acute abdomen in pregnancy include the same diagnoses considered in nonpregnant women, but also some additional life-threatening conditions. For example, ectopic pregnancy, placental abruption, acute fatty liver, HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome, or uterine rupture may cause severe abdominal pain during pregnancy. An acute abdomen is a medical emergency that requires urgent medical evaluation.

Appendicitis

Appendectomy is the most common non-obstetric indication for surgery during pregnancy.[19] Signs and symptoms of appendicitis in pregnancy generally mimic those that occur in nonpregnant individuals, including abdominal pain, nausea, abdominal rebound, and tenderness and leukocytosis. Although the appendix is displaced by uterine growth, right lower quadrant pain is the most common presentation regardless of gestational age.[19,20]

Fever and leukocytosis are not considered diagnostic for appendicitis during pregnancy because leukocytosis is a normal physiologic change of pregnancy. However, white blood cell counts may be useful in trending changes over time. Compression ultrasound is the primary diagnostic tool, but because ultrasound visualization of the appendix can be obscured in pregnancy, magnetic resonance imaging (MRI) may be necessary.

Surgery is the definitive therapy for confirmed appendicitis. The rate of appendix perforation is higher in pregnancy, with the highest incidence found in the third trimester—likely due to delays in diagnosis.[20] While a perforated appendix may result in uterine contractions, tocolysis before surgery for prophylaxis is not generally recommended. Possible complications include preterm labor, early birth, or fetal loss as well as increased maternal morbidity.

Inflammatory Bowel Disease, and Irritable Bowel Syndrome

Ulcerative colitis and Crohn's disease are chronic disorders collectively known as inflammatory bowel disease. Pregnancy does not affect the course of Crohn's disease, whereas ulcerative colitis may worsen during gestation. The effect of these disorders on the course of pregnancy depends on the activity and severity of the disease. Women with active disease are at increased risk for low birth weight and preterm birth. It is not clear if women with

inflammatory bowel disease have an increased risk for congenital anomalies.[21]

The primary consideration in caring for women with inflammatory bowel disease during pregnancy is the risk versus benefits of various medications. In general, the risks associated with active disease are higher than the risks associated with adverse effects of the medications used to treat it, with the exception of a few medications (e.g., methotrexate) that are teratogenic and contraindicated during pregnancy. Women with inflammatory bowel disease should be referred to a physician to establish a plan of care; gastrointestinal specialists may be involved in their care as well.

Gallbladder Disorders: Cholecystitis

Cholecystitis (inflamed gallbladder) is the second most common etiology of acute abdomen in pregnancy, after appendicitis. Cholecystitis usually occurs secondary to the development of gallstones that cause biliary obstruction.[22] Biliary sludge accumulates secondary to increased estrogen levels, leading to increased cholesterol synthesis. This process combines with the decreased emptying rate in pregnancy to lead to stone formation and stasis.[20,22]

Pregnant women with gallbladder disorders often present with acute epigastric pain or the classic biliary colic (pain exacerbated by a fatty meal). Other common symptoms include pain in the right upper quadrant, radiation to the right scapula, flank pain, and a positive Murphy's sign.[22] Guarding is not usually present. Anorexia, nausea, vomiting, low-grade fever, tachycardia, and intolerance of fatty foods may also be present. The differential diagnoses include HELLP syndrome, acute fatty liver, and hepatitis.

Diagnosis of cholecystitis is made via ultrasound imaging. Management depends on gestational age and symptom severity. Pain medications, antispasmodics, antibiotics, intravenous fluids, and a low-fat diet constitute conservative management. Risks of conservative treatment include recurrence, increased likelihood of induction, and preterm birth. Surgery may be indicated for women with recurrent or severe symptoms.

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) is a common disorder in the general population. Pregnancy changes such as slower gastrointestinal motility and relaxation of the lower esophageal sphincter may contribute to exacerbation of preexisting disease; alternatively, GERD may initially appear during pregnancy, usually in the second or third trimester. Symptoms are similar to those observed in nonpregnant women.[22] Typical symptoms include heartburn, nausea, and acid reflux. Atypical symptoms that indicate increasing severity and a need for medical consultation include dysphagia, noncardiac chest pain, dyspepsia, abdominal pain, hoarseness, sore throat, and dental erosions.[23]

Initial recommendations for management of GERD include nonpharmacologic measures such as elevating the head of the bed, avoiding eating before lying down, lying on the left side, and avoiding acidic foods. When pharmaceutical methods are needed, antacids, which neutralize the stomach contents decreasing the acidity, are the first-line medication. Antacids containing calcium aluminum, and magnesium are considered safe in pregnancy, whereas those containing

sodium bicarbonate should be avoided. Some authors suggest magnesium-containing antacids should be avoided in the third trimester. Antacids interfere with iron absorption and are therefore not recommended for women who need supplemental iron or should be taken several hours remote from ingestion of iron.

Histamine 2 receptor antagonists reduce gastric acid secretion and are typically the next step if previous measures do not improve symptoms. Cimetidine (Tagamet) and ranitidine (Zantac) are the two H_2-receptor antagonists commonly prescribed for GERD, but ranitidine is the only one of these agents that has proven effectiveness during pregnancy.

Proton pump inhibitors are more effective than H_2-receptor antagonists in inhibiting gastrointestinal acid secretion. Early observational studies suggested proton-pump inhibitors may be associated with teratogenic effects while later larger studies did not find this association. Lansoprazole (Prevacid), rabeprazole (Aciphex), esomeprazole (Nexium), and pantoprazole (Protonix) are all considered safe for use during pregnancy. Omeprazole (Prilosec) is associated with teratogenic effects in animal studies but not in humans.[23]

Pancreatitis

Pancreatitis is a rare but serious condition in pregnancy that occurs in approximately 1 in 1000 to 1 in 3000 women. This disease is typically associated with cholelithiasis, but can also be caused by estrogen-related elevated triglyceride levels.[24] Severe pancreatitis is described as sudden inflammation of the pancreas that causes abdominal pain, nausea, and dehydration. The primary differential diagnosis is hyperemesis gravidarum—the most common misdiagnosis in pregnancy. Pancreatitis is diagnosed by documentation of increases in levels of serum amylase, lipase, and calcium.[24] Preterm birth occurs in approximately 20% of women with severe pancreatitis, pointing to the importance of making a timely referral to an emergency department or specialist. Research has recently linked this disorder to mutations of the cystic fibrosis genes.[24]

Hematologic Disorders in Pregnancy

The hematologic disorders that are the most likely to affect pregnant women include anemias, hemoglobinopathies, and coagulation disorders. A midwife's ability to diagnosis and collaborate in managing these disorders can significantly improve outcomes and help prevent morbidity and mortality for both the pregnant woman and her fetus/infant.

Anemia: An Overview

Anemia is the most common nutritional deficiency worldwide.[25] Anemia in pregnancy is defined as hemoglobin less than 11 g/dL and hematocrit less than 33% in the first and third trimesters.[26] In the second trimester, the criteria for anemia are hemoglobin less than 10.5 g/dL and hematocrit less than 32% (**Table 23-4**).[27-29] The prevalence of anemia in pregnant women globally is approximately 35% to 56%.[28] In low-resource nations, anemia is also frequently a comorbidity of infection (e.g., malaria) or a hemoglobinopathy.

Table 23-4 **Diagnostic Values for the Diagnosis of Anemia in Pregnant Women**

	Hemoglobin (g/dL)	Hematocrit (%)
Trimester		
First trimester	11	33
Second trimester	10.5	32
Third trimester	11	33
Adjustment for Black Race		
First trimester	< 10.2	< 31.0
Second trimester	< 9.7	< 30.0
Third trimester	< 10.2	< 31.0
Adjustment for Living at High Altitude		
First trimester	< 11.2–13.0	< 33.5–39.0
Second trimester	< 10.7–12.5	< 32.0–38.0
Third trimester	< 11.2–13.0	< 33.5–39.0
Adjustment for Smoking		
0.5–0.9 pack/day	+ 0.3	+ 1.0
1.0–1.9 pack/day	+ 0.5	+ 1.5
2.0 or more pack/day	+ 0.7	+ 2.0

Based on American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 95: anemia in pregnancy. *Obstet Gynecol.* 2008;112:201-207[27]; Committee on Nutritional Status During Pregnancy, Food and Nutrition Board, Institute of Medicine. *Nutrition During Pregnancy and Lactation: An Implementation Guide.* Washington, DC: National Academy Press; 1992[28]; Chao C, O'Brien KO. Pregnancy and iron homeostasis: an update. *Nutr Rev.* 2013;71(1):35-51.[29]

Consequences of anemia during pregnancy include increased vulnerability to infection, reduced peripartum blood loss reserves, increased cardiovascular burden, and increased risk for transfusion. Anemia is also associated with an increase in maternal mortality—an association that is strongly influenced by postpartum hemorrhage. Moreover, women who are anemic are at increased risk for preterm birth, fetal growth restriction, low birth weight and prematurity, fetal/infant infection, and infant mortality.[25],[27] In addition, fetal iron stores may be compromised when a pregnant woman is anemic, and suboptimal iron stores in the neonate are associated with cognitive impairments in the child.[29] Most importantly, treatment of women with anemia during pregnancy improves these outcomes.[30],[31]

Differential Diagnosis and Initial Treatment of Anemia

Normocytic anemia is diagnosed when the hemoglobin and hematocrit are low but the mean corpuscular volume (MCV) is in the normal range of 80–100 fL; microcytic anemia refers to small red blood cells wherein MCV is less than 80 fL; and macrocytic anemia refers to large pale red blood cells wherein the MCV is larger than 100 fL. These definitions, and the causes of the various types of anemia, are noted in **Table 23-5**.[26] Anemia is a symptom of many different disorders, and multiple algorithms for evaluation of anemia are available, based on a particular population and the most likely possible diagnoses. However, because most pregnant women with anemia have iron-deficiency anemia, the usual practice is to presumptively treat with iron and then reassess the hemoglobin and hematocrit levels before performing a detailed evaluation of iron status. If iron supplementation is not successful, further evaluation is indicated. One algorithm for evaluating essentially healthy pregnant women who have anemia is presented in **Figure 23-1**.

Table 23-5 **Causes of Anemia by Mean Corpuscular Volume**

Normocytic Anemia: MCV 80–100 fL	Microcytic Anemia: MCV < 80 fL	Macrocytic Anemia: MCV ≥100 fL
Chronic disease	Chronic disease	Folic acid deficiency
Early iron deficiency	Iron deficiency anemia	Drug-induced hemolytic anemia
Hemorrhage	Lead poisoning	Liver disease and/or alcohol abuse disorder
Hemolytic anemia	Thalassemias	Vitamin B_{12} deficiency
Hypothyroidism		

Abbreviations: MCV, mean corpuscular volume; fL, femtoliter (which is 10^{-15} liters).

Note: This table is not comprehensive, rather these are the most common differential diagnoses for anemia that are likely to occur in women seen for primary care services or maternity care.

Based on American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 95: anemia in pregnancy. *Obstet Gynecol.* 2008;112:201-207.[27]

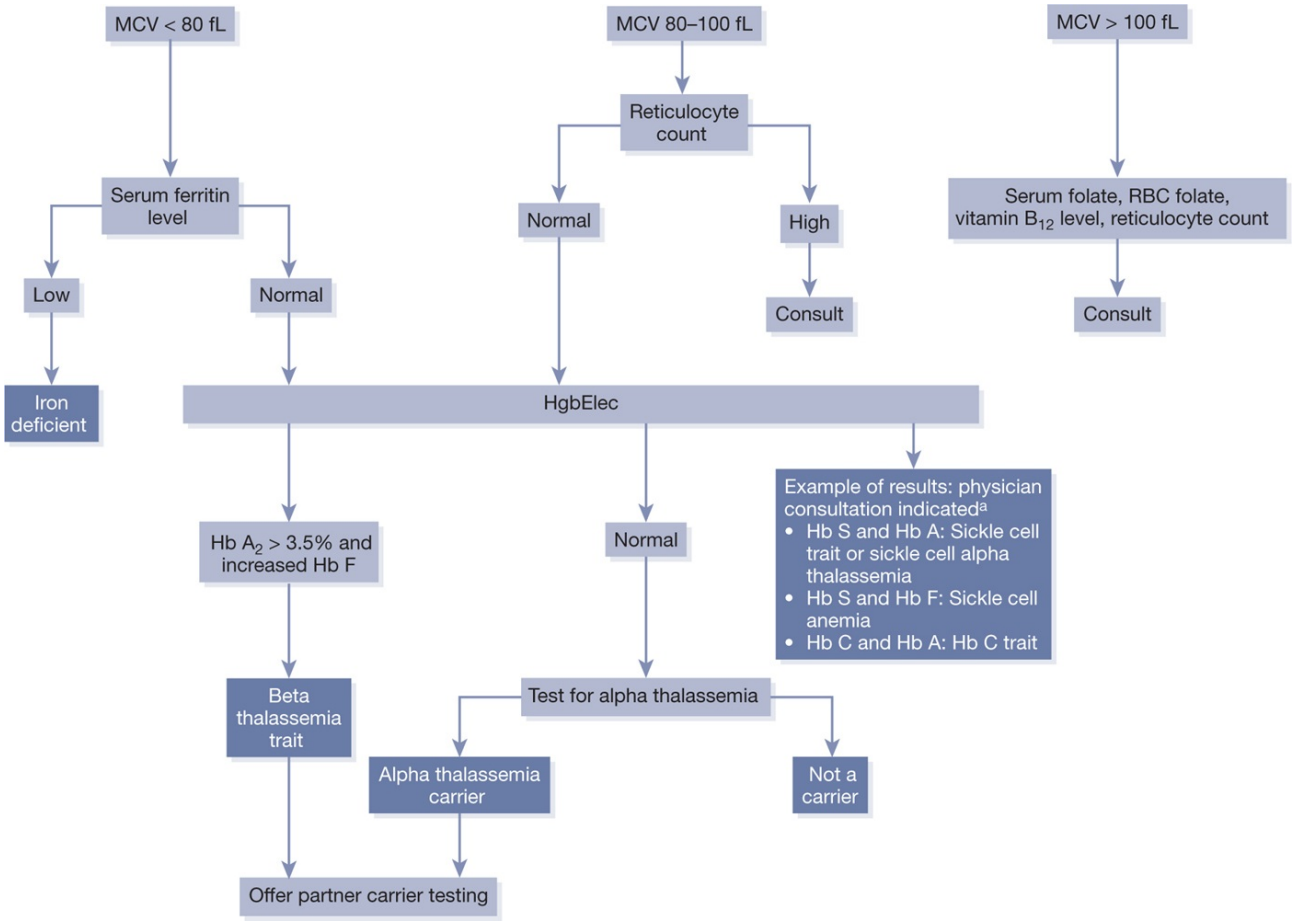


Figure 23-1 Morphologic evaluation of anemia.

Abbreviations: Hb A, hemoglobin A; Hb A_2, hemoglobin A_2; Hb C, hemoglobin C; Hb F, hemoglobin F; Hb S, hemoglobin S; Hb SC, hemoglobin SC; HgbElec, hemoglobin electrophoresis; MCV, mean corpuscular volume; RBC, red blood cell.

Note: The hemoglobin electrophoresis reports the type and percentage of different types of hemoglobin. Possible interpretations include sickle cell trait, sickle cell disease, sickle beta-thalassemia, and Hb SC disease, among others. Physician consultation is recommended for detailed interpretation, diagnosis, and management.

[a] This figure is an example of an algorithm for a population of essentially healthy pregnant women who are not at risk for other comorbid conditions or infections.

Microcytic Anemias

The most common anemia in pregnant women is iron-deficiency anemia. However, the thalassemias and sickle cell disease are also associated with a microcytic anemia.

Iron-Deficiency Anemia

In the United States, approximately 18% of pregnant women are anemic overall, but this rate varies by trimester from 6.9% to 14.3% to 29.5% in the first, second, and third trimesters, respectively.[25] Worldwide, the prevalence of iron-deficiency anemia is much higher. Approximately 40% of women begin pregnancy with low or absent iron stores.[32,33] Increased

risks for iron-deficiency anemia include multifetal gestation, multiparity, short interval between pregnancies, anemia prior to pregnancy, poor nutrition, and poor socioeconomic status.

As a preventive measure, research supports offering iron supplementation to all women in pregnancy. A Cochrane review including 27,402 women in pregnancy found that daily supplementation significantly reduced risks for maternal anemia, iron deficiency at term, and low-birth-weight infants.[31] Interestingly, intermittent supplementation (1 to 3 times per week) has been shown to be as effective as daily supplementation and has fewer side effects. This may be secondary to the regulation of iron absorption in the duodenum.

Iron deficiency is a continuum that ranges from depletion of iron stores to overt anemia. Hemoglobin and hematocrit levels do not begin to decrease until stored iron is depleted, so a pregnant woman may have normal hemoglobin and hematocrit values yet also be iron depleted. For this reason, iron supplementation is recommended for all pregnant women.

Serum ferritin levels are the most sensitive measure for diagnosis of iron-deficiency anemia, as ferritin is the storage form of iron. Serum iron levels and total iron-binding capacity can also be obtained but both have diurnal variations, with higher concentrations noted later in the day. Values for hemoglobin, MCV, serum iron, and total iron-binding capacity (TIBC) in iron-deficient states are listed in **Table 23-6**.[26,34]

Table 23-6 Laboratory Tests Indicative of Iron Deficiency, by Degree of Severity

	Normal Values	Iron Deficient Without Anemia	Iron Deficient with Anemia	Iron Deficient with Severe Anemia
Hemoglobin (Hgb), g/dL	12–13	Normal (> 12)	9–12	6–7
Mean corpuscular volume (MCV), fL	80–100	80–100	< 80	< 80
Red blood cell (RBC) morphology	Normal	Normal	Normal or slight hypochromia	Hypochromic, microcytic
Serum ferritin (ng/dL)	10–150	< 40	< 20	< 10
Serum iron (SI), mcg/dL	40–175	60–150	< 60	< 40
Total iron-binding capacity (TIBC), mcg/dL	216–400	300–390	350–400	> 410
Transferrin saturation (TSAT), %	16–60	30	< 15	< 10
Free erythrocyte protoporphyrin (FEP), mcg/dL	< 3	30–70	> 100	100–200

Based on American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 95: anemia in pregnancy. *Obstet Gynecol.* 2008;112:201-207[27]; Haider BA, Olofin I, Wang M, Spiegelman D, Ezzati M, Fawzi WW; Nutrition Impact Model Study Group (Anaemia). Anaemia, prenatal iron use, and risk of adverse pregnancy outcomes: systematic review and meta-analysis. *BMJ.* 2013;346:f3443.[34]

When iron-deficiency anemia is present, the recommendation is to supplement with 30 to 120 mg per day of elemental iron. The amount of elemental iron varies among supplements as described in the *Nutrition* chapter. The most common side effects of oral iron preparations include bloating, constipation, and black stools. These side effects can be mitigated with smaller doses of elemental iron and spaced dosing to every other day. Given iron absorption can be saturated in the intestine, smaller doses and spaced dosing may be as effective as higher daily doses. A follow-up complete blood count is performed 3 to 4 weeks after

supplementation is initiated. Reticulocytosis should be apparent by 7 to 10 days, and the hemoglobin and hematocrit levels should rise within a few weeks.[26]

Parenteral forms of iron are available and may be of value for women who do not respond to oral supplementation and/or women who have comorbid conditions associated with hemolysis or malabsorption. For example, women who have undergone bariatric surgery have diminished capacity to absorb oral formulations. Historically, parenteral forms of iron were considered a choice of last resort because of the risk of anaphylaxis associated with intravenous iron administration. Newer formulations of low-molecular-weight iron dextran are much less likely to have adverse effects, however, and can be used safely when needed.[35] Intravenous administration of iron will occur in a hospital setting. Several formulations with different doses and protocols are available.

Hemoglobinopathies

When a woman has microcytic anemia and normal iron indices, the next step is to screen for hemoglobinopathies. Normal hemoglobin is made of two pairs of polypeptide chains (two alpha and two beta), each of which has a heme–iron complex attached. More than 100 variant forms of hemoglobin exist.[36] Hemoglobinopathies are genetic synthesis disorders of hemoglobin, of which there are two types: thalassemias and sickle cell disease. Thalassemias are disorders of reduced synthesis of alpha- or beta-globin chains, whereas structural abnormalities of hemoglobin cause sickle cell disease. Both thalassemias and sickle cell disease cause microcytic anemia. Depending on the genetic inheritance, the individual may be homozygous or heterozygous for one of these disorders. The resulting phenotypes vary and are based on the percentage of different hemoglobin variants, and in the case of thalassemias, which globin genes are affected and how many are affected.

Diagnosis and treatments for hemoglobinopathies are determined by hemoglobin electrophoresis, as reviewed in the *Common Conditions in Primary Care* chapter. Implications for pregnancy and care for affected individuals during pregnancy are presented in this chapter.

Alpha-Thalassemia

Alpha-thalassemia is classified on the basis of how many of the four globin chains are affected, as described in **Table 23-7**.[37] Individuals with alpha-thalassemia trait have microcytic anemia and a normal hemoglobin electrophoresis, and DNA testing is done to detect the alpha-globin gene deletions. The primary purpose of detecting alpha-thalassemia carrier status is to identify couples who are at risk for having a fetus with hemoglobin Bart's hydrops fetalis. Prenatal counseling is likely of value for these individuals.[37]

Table 23-7 Classification of Alpha-Thalassemias

Number of Globin Genes	Genotype	Clinical Implications

0	**- -/- -**	Hemoglobin “Bart’s”: incompatible with life. The fetus develops hydrops.
1	α **-/- -**	Hemoglobin H disease. Mild to moderate hemolytic anemia.
2	α **-/**α **- -** **or** α α**/- -**	Homozygous: alpha-thalassemia trait. Mild microcytic anemia that is asymptomatic. This person is a carrier.
3	α **-/**α α	Heterozygous: silent carrier state; not clinically detectable.
4	α α**/**α α	Normal: no thalassemia present.

Beta-Thalassemia

Beta-thalassemia is the result of insufficient or absent production of the beta-globin chains.[38] This disorder is subclassified as minor, intermedia, or major depending on the amount of beta-globin chains produced. Women with beta-thalassemia major (Cooley’s anemia) or beta-thalassemia intermedia have an increased risk of diabetes and cardiovascular disease during pregnancy. Women with beta-thalassemia minor usually have uncomplicated pregnancies; because they are generally asymptomatic, this hemoglobinopathy may be first diagnosed in pregnancy.

Beta-thalassemia trait is diagnosed by hemoglobin electrophoresis, which will reveal more than 3.5% of hemoglobin A_2 and increased levels of hemoglobin F. Once diagnosed, partner testing can be offered. Prenatal diagnosis for beta-thalassemia major is available.

Sickle Cell Disease

Sickle cell disease is characterized by intermittent, painful “sickle cell crisis,” especially during viral infections. Sickle cell disease and trait are highly prevalent in India, the Mediterranean, the Middle East, and sub-Saharan Africa because this hemoglobin variant is protective against malaria. Sickle cell disease is a multiple-organ disorder associated with hemolytic anemia and microvascular obstruction by red blood cell agglutination. Persons who have sickle cell disease can experience intensely painful crises, particularly due to interference with oxygenation of vessels and organs. In addition, they can accumulate iron and become iron overloaded despite having microcytic anemia. Women with sickle cell disease have an increased risk for spontaneous abortion, preterm labor, preeclampsia, stillbirth, fetal growth restriction, prematurity, and low birth weight.[39] These women need specialized care during pregnancy.

Women with sickle cell trait have an increased risk for urinary tract infections during pregnancy secondary to increased levels of free iron in the urine and increased monitoring through periodic urine cultures during pregnancy can reveal asymptomatic bacteriuria. Additionally, iron does not accumulate in these women, so they can become iron deficient in pregnancy and need iron supplementation.

If a pregnant woman has sickle cell disease or sickle cell trait, screening of the father of the fetus is offered, along with genetic counseling, to determine inheritance patterns and the chance that the fetus will have the trait or disease. Sickle cell trait follows a Mendelian autosomal

recessive inheritance pattern. Thus, if the woman has sickle cell trait and the father of the fetus does not, there is a 50% chance in each pregnancy that her fetus will have the trait. If the woman and father of the fetus both have sickle cell trait, there is a 25% chance that the fetus will have sickle cell disease and a 50% chance that the fetus will have sickle cell trait.

Macrocytic Anemia

Macrocytic anemia can be secondary to glucose-6-phospate dehydrogenase (G6PD) deficiency, vitamin B_{12} deficiency, alcoholism, folate deficiency, or medications such as those used to treat human immunodeficiency virus (HIV). Macrocytic anemia is not common in pregnant women.

Glucose-6-Phosphate Dehydrogenase Deficiency

G6PD deficiency is an inherited defect in a red blood cell enzyme that protects the red blood cell from oxidative injury. This disorder can range from severe hemolytic anemia to asymptomatic. Women with a G6PG deficiency rarely manifest symptoms, and it is unusual for G6PD deficiency to adversely affect the course of pregnancy. The primary maternal risk associated with G6PD deficiency is severe hemolysis as the result of use of medications such as nitrofurantoin and sulfa derivatives, infection or surgery, and ingestion of certain foods such as fava beans.

Infants born with G6PD deficiency may present with severe jaundice and kernicterus. If a pregnant woman has G6PD deficiency or a family history of this condition, the pediatric providers for the newborn will need to be aware that the fetus may have this disorder to treat the newborn appropriately.[40] Neonatal screening for G6PD is not universal in the United States.

Thrombocytopenia

Physiologic changes in pregnancy include a reduction in the platelet count, especially in the third trimester. Thrombocytopenia is defined as a platelet count less than 150,000 cells/mL. Pregnancy-related thrombocytopenia includes gestational thrombocytopenia and thrombocytopenia related to severe preeclampsia or HELLP syndrome. Types that evolve from causes unrelated to pregnancy but may first manifest during pregnancy include immune thrombocytopenia, antiphospholipid syndrome, and thrombotic thrombocytopenia. Initial screening involves evaluating the platelet count and the white blood cell count in routine prenatal lab tests.

Gestational thrombocytopenia typically develops in the second trimester and the platelet count is rarely less than 70 cells/mL. This disorder is not associated with fetal thrombocytopenia and generally resolves within days to a few months. In contrast, immune thrombocytopenia can occur at any time during pregnancy and platelet counts can be quite low. Neonatal thrombocytopenia is possible because the maternal autoantibodies cross the placenta.

Due to the serious clinical consequences associated with different forms of thrombophilia, an early response to abnormal platelet counts, diagnosis, and treatment and/or referral is

extremely important.[41,42]

Coagulation Disorders: Venous Thromboembolism and Thrombophilias

Thromboembolic events are one of the leading causes of maternal mortality. Although the absolute incidence is low, a woman's risk of developing a venous thrombolic event (VTE)—either deep vein thrombosis (DVT) or pulmonary embolism (PE)—is four-fold higher during pregnancy than when she is not pregnant.[43] Pregnancy, as a hypercoagulable state, is a significant risk factor for VTE. Additional risk factors include previous VTE, family history of VTE, inherited thrombophilia, diabetes, autoimmune inflammatory disorders, age greater than 35 years, body mass index (BMI) greater than 30 kg/m^2, varicose veins, multifetal gestation, and hospitalization.[44]

Symptoms of DVT include pain, tenderness, swelling, palpable cord, and changes in color and limb circumference. Calf pain may also be present. Homan's sign is no longer used for assessing DVTs because it is neither adequately sensitive nor specific. Nearly 90% of DVTs that develop in pregnant women occur in the left lower extremity, perhaps due to compression of the left iliac artery by the gravid uterus. Women with a PE may present with dyspnea, tachypnea, tachycardia, cough, pleuritic chest pain, fever, anxiety, cyanosis, and hemoptysis. Women with symptoms of a DVT or PE require physician evaluation.

A DVT is diagnosed via compression ultrasound. Antepartum management includes increased surveillance and anticoagulant treatment with low-molecular-weight heparin.[45,46]

Care during pregnancy for women with a history of a VTE depends on several factors. VTEs are classified as provoked or unprovoked. A provoked VTE occurs in persons who have a clinical risk factor such as surgery, bone fracture, or bedrest. A VTE is determined to be unprovoked if no risk factor is identified. If a pregnant woman has a history of a VTE, anticoagulant prophylaxis may be indicated depending on whether the VTE was provoked or unprovoked:

- Women with a history of an unprovoked VTE prior to pregnancy are tested for inherited thrombophilias and it is recommended that they receive anticoagulant therapy prophylactically during pregnancy.
- Women with a history of provoked VTE are generally not at increased risk for reoccurrence. However, if the woman has an underlying thrombophilia, her risk for developing a VTE in pregnancy is significant. Therefore, women with a history of provoked VTE and a first-degree relative with a history of high-risk thrombophilia should be screened for thrombophilia and offered prophylactic anticoagulant therapy if a thrombophilia is diagnosed.[47,48]

Thrombophilias and Risk for Venous Thromboembolism

Inherited thrombophilias are genetic conditions that increase the risk for thromboembolism. Approximately 50% to 60% of women who develop a VTE during pregnancy have an inherited thrombophilia,[44,45] such as factor V Leiden (FVL) deficiency, prothrombin G20210A gene

mutation, protein S deficiency, or protein C deficiency. Most women with thrombophilias are asymptomatic and undiagnosed; thus, one of these disorders may be first identified in a woman who develops a VTE during pregnancy.

Routine screening of pregnant women for inherited thrombophilias is not recommended. However, as noted above, testing for thrombophilias is offered to pregnant women who have either a history of an unprovoked VTE or a first-degree relative who has a known history of a high-risk thrombophilia or a history of VTE that occurred before the age of 50 years.[48]

Thrombophilias and Risk for Adverse Pregnancy Outcomes

The risk for adverse pregnancy outcomes associated with thrombophilias is controversial. Some thrombophilias, such as FVL, are associated with increased risks of preeclampsia, whereas others do not appear to adversely affect the course of pregnancy. Furthermore, some thrombophilias are associated with a higher frequency of VTE than are others. Care of women who have an inherited thrombophilia depends on the type of thrombophilia as well as her personal and family history of VTE; thus, women with a known inherited thrombophilia are referred for physician consultation to establish a plan of care.[48] Although routine screening for thrombophilias is not recommended at this time, current research on pregnancy outcomes associated with specific thrombophilias may identify risks that will change this recommendation.[47],[48]

Hepatic Disorders

Hepatic (liver) disorders that are specific to pregnancy include hyperemesis gravidarum and intrahepatic cholestasis. These conditions are reviewed in the *Pregnancy-Related Conditions* chapter. Preeclampsia, HELLP syndrome, and acute fatty liver of pregnancy are considered both hypertensive and hepatic disorders, are reviewed later in this chapter. Other liver diseases that may adversely affect the course of pregnancy include hepatitis, cirrhosis, autoimmune liver disease, Wilson's disease, and drug-induced hepatotoxicity. Diagnosis and treatment of hepatitis are reviewed in the *Common Conditions in Primary Care* and *Reproductive Tract and Sexually Transmitted Infections* chapters. Physician consultation is indicated for women with signs or symptoms of hepatitis or liver dysfunction.

Acute viral hepatitis during pregnancy increases both maternal and fetal morbidity. Hepatitis A is generally self-limited and management is supportive. Nevertheless, this form of hepatitis may be associated with preterm labor or premature prelabor rupture of membranes if the woman becomes ill during the second or third trimester.

The more common hepatic condition that may occur in women cared for in midwifery practice is positive carrier status for hepatitis B or hepatitis C. The risk of vertical transmission of hepatitis B is as high as 90% when the woman has this infection, and infants who acquire hepatitis B via vertical transmission at the time of birth are likely to become chronic carriers. Thus, screening for hepatitis is recommended for all pregnant women. Testing for the degree of viral load is recommended for women who are identified as carriers of hepatitis B. These women are referred for physician consultation because antiviral medication may be recommended to decrease the risk of vertical transmission at birth.

Hypertension During Pregnancy

Hypertensive disorders of pregnancy are classified into four conditions: (1) gestational hypertension, (2) preeclampsia–eclampsia, (3) chronic hypertension, and (4) chronic hypertension with superimposed preeclampsia–eclampsia.[49] Preeclampsia is further subcategorized as preeclampsia with or without severe features.[49] A severe constellation of hemolysis, elevated liver enzymes, and low platelets—that is, HELLP syndrome—is a variant of preeclampsia that occurs in a small subset of women who develop preeclampsia. The nomenclature for hypertensive disorders of pregnancy has a long history and has changed several times in the past. The 2013 American College of Obstetricians and Gynecologists (ACOG) Taskforce on Hypertension in Pregnancy Report provides an important perspective on the current nomenclature for clinical management.[49] Preeclampsia and eclampsia are not distinct pathologies, but rather disorders that exist on a continuum.[49] Preeclampsia was once thought to be a unique pregnancy-related condition that resolved postpartum. However, just as gestational diabetes is associated with future development of type 2 diabetes, it is now evident that hypertensive disorders of pregnancy are also a continuum linked to long-term adverse cardiovascular outcomes. Thus, it is of value to consider these conditions as a group.

In the United States, 3% of pregnant women develop preeclampsia and 5% to 10% of pregnant women have other hypertensive disorders.[50] Approximately 7.4% of pregnancy-related maternal deaths are attributable to these disorders.[51,52] A recent analysis by the World Health Association found that 14.0% of maternal deaths worldwide were attributable to hypertension, second only to deaths from hemorrhage (27.1%).[53] Hypertension is defined as either a systolic blood pressure of 140 mm Hg or greater *or* a diastolic blood pressure of 90 mm Hg or greater *or* both observed on two occasions at least 4 hours apart.[49] During pregnancy, hypertension is classified as mild/moderate (systolic, 140–159 mm Hg or diastolic, 90–109 mm Hg) or severe (systolic, ≥ 160 mm Hg or diastolic, ≥ 110 mm Hg). Severe hypertension is a medical emergency that is diagnosed when two measurements meeting the criteria are noted within 15 minutes.[50] Blood pressure should always be obtained after the woman is at rest for at least 10 minutes and seated with an appropriately sized cuff positioned at the level of her heart.

Differentiating between the types of hypertensive disorders of pregnancy is not always straightforward and requires a clear understanding of specific diagnostic components. **Table 23-8** provides an overview of the diagnostic criteria for each condition.

Table 23-8 Diagnostic Criteria for Hypertensive Disorders in Pregnancy

Condition	Onset of Hypertension	Diagnostic Criteria
Gestational hypertension	≥ 20 weeks of pregnancy	Hypertension (systolic, ≥ 140 mm Hg and/or diastolic, ≥ 90 mm Hg) in a previously normotensive woman without proteinuria or diagnostic features of preeclampsia
Preeclampsia[a]	≥ 20 weeks of pregnancy	New-onset hypertension plus proteinuria (≥ 300 mg per 24-hour urine collection, or > 1+ dipstick if other quantitative methods are not available,

		or protein/creatinine ratio > 0.3 mg/dL) If no proteinuria, new-onset hypertension with any of the following: Thrombocytopenia (platelet count < 100,000 cells/mcL) Impaired liver function (elevated liver transaminases to twice normal) Renal insufficiency (serum creatinine > 1.1 mg/dL or doubling of serum creatinine in the absence of other renal disease) Pulmonary edema Cerebral or visual disturbances
Preeclampsia with severe features	≥ 20 weeks of pregnancy	Same diagnostic criteria as for preeclampsia but with the addition of at least one of the following: Systolic blood pressure ≥ 160 mm Hg and/or diastolic blood pressure ≥ 110 mm Hg measured twice at least 15 minutes apart Thrombocytopenia (platelet count < 100,000 cells/mcL) Impaired liver function: transaminases 2 times the normal level; severe right upper quadrant or epigastric pain Renal insufficiency: in the absence of other renal disease, serum creatinine > 1.1 mg/dL or twice the baseline level Pulmonary edema New-onset cerebral or visual disturbances
Eclampsia	After or with preeclampsia	Preeclampsia plus new-onset grand mal seizures
Chronic hypertension	Prior to pregnancy or < 20 weeks of pregnancy	Hypertension that predates pregnancy, occurs before 20 weeks of pregnancy, or persists after pregnancy
Chronic hypertension with superimposed preeclampsia/eclampsia	Prior to pregnancy or < 20 weeks of pregnancy	Worsening hypertension with new development of any of the following: proteinuria, elevated liver enzymes, thrombocytopenia, pulmonary edema, cerebral or visual disturbances, renal insufficiency

[a] An atypical form of preeclampsia that occurs before 20 weeks' gestation is associated with hydatidiform mole and antiphospholipid syndrome.

Based on American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy: report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122(5):1122-1131.[49]

Women who have a hypertensive disorder of pregnancy are at increased risk for subsequent cardiovascular disease and mortality as well as for future hypertension and diabetes.[54] The American Heart Association guideline for the prevention of cardiovascular disease in women recognizes preeclampsia, eclampsia, and pregnancy-induced hypertension (gestational hypertension) as independent risk factors for cardiovascular disease, such that assessment of pregnancy history is one of the critical components in determining a woman's cardiovascular risk.[54]

Because of the potential for lifelong adverse effects following hypertensive disorders of pregnancy, educating women and their families during the perinatal period is essential. This health education includes discussing the importance of informing all subsequent healthcare providers of the disorder during pregnancy, addressing the importance of health behaviors to ameliorate modifiable risks, and providing ongoing clinical follow-up and, in some cases,

specialty referral.[54] It is important to recognize that much of the evidence that associated hypertensive disorders of pregnancy with a woman's future health is relatively new. Additional clinical implications of these findings will become evident as research continues.

Gestational Hypertension

The distinguishing characteristics of gestational hypertension are two-fold. First, the woman does not have proteinuria or meet the other diagnostic criteria for preeclampsia/eclampsia. Second, gestational hypertension resolves by 12 weeks after birth. If there is a continuation of the hypertension beyond this period, the diagnosis is changed to chronic hypertension. Approximately 46% of women with gestational hypertension will develop preeclampsia.[55] Often, what initially appears to be gestational hypertension is, in fact, preeclampsia in the process of developing. The likelihood of developing preesclampsia increases in women who develop gestational hypertension before 30 weeks' gestation. If diagnostic criteria arise that are consistent with preeclampsia/eclampsia, the diagnosis is changed to preeclampsia.

Management recommendations include evaluation for signs or symptoms of preeclampsia (e.g., urinary protein excretion; liver enzyme levels; fetal well-being via nonstress test or ultrasound, or both). If preeclampsia is ruled out, women are encouraged to monitor blood pressure at home and are seen once or twice weekly. The fetus is monitored via fetal movement counts and weekly fetal surveillance testing. An ultrasound for fetal growth may be obtained. Women are informed of danger signs and have a plan for whom to call and when to call.[49] The optimal gestational age for birth depends on the severity of the disorder and the status of the fetus. In women with uncomplicated gestational hypertension, induction of labor is generally recommended after 37 to 38 weeks' gestation.[49]

Preeclampsia–Eclampsia

Approximately 2% to 8% of women experience preeclampsia during pregnancy worldwide.[50] In the United States, the current incidence is approximately 3.8%—which represents an increase since 1990, when the incidence was approximately 2%.[51]

Pathophysiology of Preeclampsia

Although the primary feature of preeclampsia is hypertension that usually appears late in pregnancy, preeclampsia is actually a multisystem disorder that begins early in pregnancy. Placental tissue is key to this disorder; that is, placental tissue must be present but a fetus does not need to be. Thus, women with a hydatidiform mole have a risk for developing signs of preeclampsia early in pregnancy. Furthermore, the disease resolves spontaneously only after the placenta is delivered.

Preeclampsia is characterized by abnormal placentation and failed remodeling of the spiral arteries, which usually occurs early in gestation in two distinct phases.[56] In a normally developing placenta, cytotrophoblast cells extend into endothelial lining of the decidual segment of the spiral arteries first; later in a second wave, cytotrophoblast cells invade the

myometrial portion of the arteries. This process causes the spiral arteries to be remodeled so they become large, low-resistance vessels that can accommodate increased blood flow.[57] Failure of this process causes hypoperfusion, hypoxia, and ischemia within the developing placenta.

The second phase of the development of preeclampsia reflects the maternal inflammatory response to the initial abnormal placentation and subsequent placental hypoxia. As a result of maternal systemic endothelial activation, hypoxia and subsequent oxidative stress cause endothelial cell dysfunction in the maternal spiral arteries. The release of cytokines and other substances then results in systemic inflammation, vascular endothelial dysfunction throughout the vascular system, and a prothrombotic condition.[58,59] This process is characterized by hypertension and, in more severe cases, organ damage, including damage to the kidneys, liver, and brain.[59] The kidney and liver both filter blood, which may partly explain why these two organs are especially vulnerable to the abnormal function of the endothelial cells that line all blood vessels. Early-onset preeclampsia (diagnosis before 34 weeks' gestation) is associated with more severe adverse maternal and fetal outcomes as well as increased risk for future cardiovascular morbidity and mortality. The pathophysiology of preeclampsia and clinical findings are described in **Table 23-9**.[60,61]

Table 23-9	Abnormal Findings in Preeclampsia/Eclampsia
Pathophysiology	**Organ System Findings**
Renal	
Release of placental protein leads to endothelial edema and hypertrophy, reducing the glomerular filtration rate and increasing tubular reabsorption	Proteinuria ↑ Serum creatinine ↑ Serum uric acid ↑ Blood urea nitrogen (BUN) ↓ Creatinine clearance Oliguria
Cardiovascular	
Excessive inflammation and endothelial dysfunction cause hyperreactivity of vessels	Hypertension
Fluid shifts from intravascular to extracellular spaces, causing edema	Peripheral edema
Endothelial blood vessel damage leads to fluid leakage into alveoli; most frequently occurs postpartum	Pulmonary edema
Hepatic	
Damage to lining of small blood vessels leads to hemolysis, elevated liver enzymes, and increased fibrin deposits	↑ Aspartate transaminase (AST) ↑ Alanine transaminase (ALT)
The preceding effects can lead to tissue necrosis and infarction; pain is caused by vasospasm	↑ Right upper quadrant or epigastric pain
Central Nervous System	

Endothelial damage allows fluid to leak into brain tissue, leading to cerebral edema Increased vasospasm can result in ischemia	Headaches, visual disturbances Seizures Stroke Hyperreflexia
Hematologic	
Fluid shifts from intravascular to extracellular spaces, decreasing serum levels and causing edema Reduction in plasma volume results in hemoconcentration	↑ Hemoglobin and hematocrit
Inappropriate activation of leukocytes, complement, and clotting factors depletes platelets and increases clotting time, leading to hemolysis	Thrombocytopenia ↓ Fibrinogen Fibrin split products Prolonged prothrombin time (PT) Prolonged partial prothrombin time (PPT)
Placental Function	
Inadequate remodeling of spiral arteries leads to vasoconstriction and reduced placental perfusion	Intrauterine growth restriction ↓ Amniotic fluid index Nonreactive nonstress test Placental abruption

Based on American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy: report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122(5):1122-1131[49]; Snydal S. Major changes in diagnosis and management of preeclampsia. *J Midwifery Womens Health*. 2014;59(6):596-605[60]; Warrington JP, George EM, Palei AC, Spradley FT, Granger JP. Recent advances in the understanding of the pathophysiology of preeclampsia. *Hypertension*. 2013;62(4):666-673.[61]

Preeclampsia is associated with placental abruption, pregnancy loss, stroke, organ failure, and maternal death. For the fetus, adverse effects of this disorder include preterm birth, fetal growth restriction, stillbirth, and neonatal death. Women who develop preeclampsia have a two-fold increase in the risk for cardiovascular disease and mortality from ischemic heart disease, heart failure, or stroke.[62-64] The likelihood of having hypertension after the perinatal period is increased more than three-fold when a woman develops preeclampsia. Factors that affect the increased risk for cardiovascular disease include multiparity, prematurity and severity of the hypertensive disorder.[62-64]

Risk Factors for Preeclampsia

Risk factors for preeclampsia are listed in **Table 23-10**. A history of preeclampsia in a prior pregnancy increases the risk of preeclampsia by seven-fold compared to women without a history of preeclampsia. The U.S. Preventive Services Task Force and other professional associations recommend that women with high risk factors are for preeclampsia take low dose aspirin (81 mg) daily, starting in the second trimester, in subsequent pregnancies.[49,64] Low-

dose aspirin among these women reduces the risk for preeclampsia by 24% (relative risk [RR], 0.76; 95% confidence interval [CI], 0.62–0.95).[64]

Table 23-10 **Risk Assessment for Preeclampsia Based on Medical History**

Risk Level	Risk Factors	Recommendation
High[a]	Autoimmune disease (SLE, APA) Chronic hypertension History of preeclampsia, especially if accompanied by an adverse outcome Multifetal gestation Renal disease Type 1 or 2 diabetes	Recommend low-dose aspirin if the woman has ≥ 1 of these high-risk factors
Moderate[b]	Age ≥ 35 years BMI ≥ 30 kg/m^2 Family history of preeclampsia in mother or sister Nulliparity Personal history risk factors (e.g., low birth weight, small for gestational age, previous adverse pregnancy outcome, > 10 year pregnancy interval) Sociodemographic characteristics (African American race, low socioeconomic status)	Consider low-dose aspirin if the woman has several of these moderate-risk factors

Abbreviations: APA, antiphospholipid antibody; SLE, systemic lupus erythematosus.

[a] Single risk factors that are consistently associated with the greatest risk for preeclampsia. The incidence of preeclampsia in pregnant women with ≥ 1 of these risk factors is approximately ≥ 8%.

[b] A combination of multiple moderate-risk factors may be used to identify women at high risk for preeclampsia. These risk factors are independently associated with preeclampsia, some more consistently than others. These risk factors vary in their association with an increased risk for preeclampsia.

Modified with permission from U.S. Preventive Services Task Force. *Final Recommendation Statement: Low-Dose Aspirin Use for the Prevention of Morbidity and Mortality from Preeclampsia: Preventive Medication*. December 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/low-dose-aspirin-use-for-the-prevention-of-morbidity-and-mortality-from-preeclampsia-preventive-medication. Accessed December 6, 2017.[64]

Diagnosis of Preeclampsia

Preeclampsia may slowly progress over a period of weeks, or it may develop in a fulminant manner over a few hours. Prior to the appearance of diagnostic symptoms of preeclampsia, some clinical findings warrant close observation and follow-up. Fetal growth restriction or new-onset proteinuria in the second half of pregnancy without hypertension may precede development of diagnostic criteria for preeclampsia. While not diagnostic, an elevation of blood pressure late in the pregnancy (above baseline levels from the first trimester) that exceeds 15 mm Hg diastolic or 30 mm Hg systolic is sometimes seen in women who go on to

develop preeclampsia. Recent research has investigated using certain biomarkers and uterine artery Doppler studies to help predict preeclampsia. At this time, no screening method for the disorder is recommended other than taking a complete medical history to assess for known risk factors.[49]

Historically, edema, proteinuria, and hypertension were the classic triad of symptoms that defined preeclampsia. Edema is relatively common in pregnancy, however, so it is no longer considered a diagnostic criterion. Proteinuria is a common manifestation of reduced renal function, but symptoms of other target organ damage may manifest before proteinuria appears, and this condition is sometimes absent in women with preeclampsia. Preeclampsia without proteinuria was previously called "atypical preeclampsia." Today, when hypertension exists but proteinuria is absent, other factors are used to confirm diagnosis, as noted in **Table 23-8**. When a woman with hypertension develops systemic organ involvement or severe hypertension (blood pressure ≥ 160 mm Hg systolic and/or 110 mm Hg diastolic on two occasions), the diagnosis becomes preeclampsia with severe features. Early-onset preeclampsia (diagnosis before 34 weeks' gestation) is associated with more adverse effects for both the woman and the fetus as well as increased risk for future cardiovascular morbidity and mortality.[65]

Although preeclampsia is the most common cause of hypertension and end-organ dysfunction, it is important to remember the differential diagnoses that can mimic preeclampsia:

- Acute fatty liver of pregnancy: In addition to having symptoms of preeclampsia, women with acute fatty liver of pregnancy present with anorexia, nausea and vomiting, and low-grade fever. Liver dysfunction is more severe, and the woman may develop disseminated intravascular coagulation.
- Thrombotic thrombocytopenic purpura or hemolytic uremic syndrome: Women with this syndrome have microangiopathic hemolytic anemia, thrombocytopenia, neurologic and renal abnormalities, and fever.
- HELLP syndrome: HELLP syndrome (discussed later in this section) is considered a severe form of preeclampsia, but women with HELLP syndrome may not present with hypertension or proteinuria. The presentation of HELLP is variable although the most common symptoms are abdominal pain and mid-epigastric or right upper quadrant tenderness. Nausea and vomiting and malaise may be present, causing this condition to be mistaken for hepatitis. Jaundice, visual changes, and ascites can develop.

These disorders are severe illnesses associated with a high incidence of maternal and fetal morbidity. Prompt assessment by a physician or maternal–fetal medicine specialist is indicated for women who have symptoms that suggest a disorder other than preeclampsia is present.

Treatment of Women with Preeclampsia

The role of the midwife in caring for women who develop hypertension during pregnancy focuses on meticulous screening, early identification, referral or consultation, ongoing

assessment, and participation in a collaborative model of care as appropriate. Inpatient care is reviewed in more detail in the *Complications During Labor and Birth* chapter.

The definitive treatment of preeclampsia is delivery of the placenta. Expectant management requires consideration of the health of the woman and the health of her fetus. If birth is not indicated for fetal well-being, then the goal of treatment is to treat the woman's symptoms to allow the fetus to have more time in utero.

Antihypertensive medication is not recommended for women with preeclampsia unless she has severe features.[49] Increased surveillance includes weekly blood pressure measurements, daily fetal movement counting, assessment of maternal symptoms, and monitoring platelet counts and liver enzyme levels.

Management of Women with Severe Hypertension

Severe hypertension (systolic blood pressure ≥ 160 mm Hg and/or diastolic blood pressure ≥ 110 mm Hg) is an acute emergency. Evidence-based guidelines for treating severe hypertension produced by the National Partnership for Maternal Safety,[66] and endorsed by other professional associations, state that women with severe hypertension be evaluated within 15 minutes and treated within 30 minutes. Treatment of severe hypertension is reviewed in the *Complications During Labor and Birth* chapter.

Eclampsia

Eclampsia may present in the antepartum, intrapartum, or early postpartum period. Eclampsia occurs in 2% to 3% of women who have preeclampsia with severe features who are not treated with antiseizure medication and in 0.6% of women with preeclampsia without severe features.[67] Seizures are often preceded by hypertension, severe headache, hyperreflexia, visual disturbances, and right upper quadrant or epigastric pain. Approximately 25% of women who have eclampsia do not have previous symptoms.[49]

During the tonic–clonic seizure associated with eclampsia, a fetal bradycardia typically develops that generally lasts 3 to 5 minutes. The fetal heart rate pattern usually improves after the seizure resolves. Complications of eclampsia include brain hemorrahge, permanent neurologic morbidities, and stroke. In addition, placental abruption may occur if the seizure is prolonged.[67] Treatment of eclampsia is reviewed in the *Complications During Labor and Birth* chapter.

Chronic Hypertension

Chronic hypertension affects more than 5% of all pregnant women, and the incidence is expected to rise as the incidence of obesity increases. Most chronic hypertension is of primary etiology, but in as many as 10% of women chronic hypertension occurs secondary to underlying renal or endocrine disease.[49] Approximately 20% of women who have chronic hypertension will develop preeclampsia, although this risk is higher among those women with more severe hypertension.[68] Factors that increase a woman's risk for superimposed

preeclampsia include having secondary hypertension, diabetes, obesity, and preeclampsia in a previous pregnancy.

Complications associated with chronic hypertension include preterm birth, fetal growth restriction, placenta abruption, and superimposed preeclampsia. Women who have chronic hypertension are referred to a physician for management for prenatal care. Midwives may be involved in prenatal care visits once a plan of care is established.

When a woman with chronic hypertension first presents for prenatal care, an initial history, physical examination, assessment of current medications, and baseline testing of renal function are performed. Specific tests include serum creatinine, electrolytes, uric acid, liver enzymes, platelet count, and a quantitative measure of urine protein.

Assessing for the possibility of secondary hypertension is important, because women with secondary hypertension may require additional management. Chronic renal disease is the most common etiology of secondary chronic hypertension. Resistant hypertension, hypokalemia, elevated serum creatinine, and a family history of renal disease should raise the index of suspicion for secondary hypertension, and providers may consider a referral to a kidney specialist if suspected.[49]

Proteinuria is often present in women with renal complications of hypertension, and it is particularly important to establish a baseline for this condition. New-onset or worsening proteinuria is an indicator of the development of superimposed preeclampsia in a woman with chronic hypertension. Therefore, knowledge of the baseline value of proteinuria present at the onset of care is necessary to detect worsening renal function. Early diabetes screening should also be considered for women with chronic hypertension. An echocardiogram may be recommended for women who have a history of prolonged severe chronic hypertension (> 4 years).

Management of Women with Chronic Hypertension

Several different classes of medication can be used to treat chronic hypertension. All antihypertensive medications cross the placenta. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), mineralocorticoid receptor antagonists (e.g., spironolactone [Aldactone]), and direct renin inhibitors are contraindicated in pregnancy because they are associated with fetal malformations and renal abnormalities.[69,70] Statins are also contraindicated during pregnancy, as they may have teratogenic effects.[49] Because of these contraindications, a review of current medications can be a critical assessment at a preconceptional or an initial prenatal visit. An urgent physician consultation is indicated if a pregnant woman presents for prenatal care and is using one of these classes of medications.

Treatment of chronic hypertension depends on the severity of the hypertension and the presence or absence of comorbidities. Women with mild elevations in blood pressure (systolic, 140–150 mm Hg; diastolic, 90–100 mm Hg) but no other comorbid disorders may be monitored without use of medication, whereas women with mild hypertension who have comorbid diseases such as diabetes may require medication. Some women with mild chronic hypertension can discontinue medication during pregnancy or delay starting medication because the normal physiologic decline in blood pressure during the first half of pregnancy can

result in normal blood pressure values. Women with higher degrees of hypertension are offered antihypertensive medication.[49] Agents recommended for antihypertensive therapy in pregnancy include labetalol (Normodyne), nifedipine (Procardia), and methyldopa (Aldomet). For women with previous, early-onset preeclampsia and preterm birth, daily low-dose aspirin (60–80 mg) can be started late in the first trimester.[49]

Home blood pressure monitoring can be helpful for some women. "White coat hypertension"—that is, elevated blood pressure when present with a healthcare provider—may increase blood pressure readings in the clinical setting above those experienced at home.[71] Home monitoring will also allow for comparing measurements in different environments to ensure accurate diagnosis. Because preeclampsia typically develops after 20 weeks' gestation, increased frequency of blood pressure checks in the second half of the pregnancy is recommended. For women who are accustomed to engaging in exercise and have well-controlled blood pressure, moderate exercise is recommended during pregnancy. Salt-restricted diets and weight loss are not recommended for management of chronic hypertension in pregnancy.

Serial fetal growth ultrasounds help to monitor for fetal growth restriction when women have chronic hypertension. Depending on the severity of maternal disease, weekly or biweekly antenatal fetal testing and amniotic fluid assessment may be recommended starting in the early third trimester. Timing of birth is based on maternal and fetal indications.

Chronic Hypertension with Superimposed Preeclampsia

Initial management of women with chronic hypertension and superimposed preeclampsia follows the same guidelines recommended for women with preeclampsia, including assessment of severity, baseline laboratory values, fetal well-being, and gestational age. When the superimposed preeclampsia has severe features, administration of parenteral magnesium sulfate is indicated during the intrapartum and postpartum periods. Decisions about the timing of birth are driven by consideration of disease severity, the rate at which the disease is progressing, and close assessment of maternal and fetal well-being.

HELLP Syndrome

HELLP syndrome, is characterized by **h**emolysis, **e**levated **l**iver enzymes, and **l**ow **p**latelets. The etiology of this syndrome is unclear. It is usually considered a severe form of preeclampsia, although some experts suggest that HELLP may be a separate autoimmune disorder, especially because as many as 20% of women with this condition do not have hypertension or proteinuria. HELLP occurs in approximately 0.9% of pregnant women.[72] It most commonly arises during the antepartum period, but may emerge postpartum. Risk factors include previous hypertensive disorder of pregnancy, previous HELLP syndrome, and nulliparity.

HELLP syndrome is characterized by abnormal vascular tone, vasospasm, and coagulation defects.[72] Affected women may report cerebrovascular symptoms of severe headache (predominantly frontal), blurred vision, or scotomata and/or right upper quadrant pain or

persistent epigastric pain. Diagnosis is based on the triad of abnormal coagulopathy, liver dysfunction, and thrombocytopenia. The more abnormal the lab findings, the more severe the disease.

The presence of HELLP syndrome increases a woman's risk for eclampsia, disseminated intravascular coagulation, acute renal failure, liver hematoma, placental abruption, fetal growth restriction, preterm birth, neonatal respiratory distress syndrome, and perinatal death. Management focuses on stabilization of blood pressure and assessment of fetal well-being to determine the optimal time for delivery.[73] In general, immediate birth for women who are 34 weeks' gestation or more is recommended. If the woman is less than 34 weeks' gestation, a delay is recommended to allow for administration of corticosteroids to help fetal lung development as long as both the woman and the fetus are stable.[49,72,73]

Infectious Diseases

Several infectious diseases can cause congenital anomalies, fetal harm, or fetal death. TORCH is an older acronym for toxoplasmosis, other, rubella, cytomegalovirus (CMV), and herpes. Although a single test for TORCH antibodies is available. specific tests for individual infections based on risks, exposure, or maternal/fetal signs have become more widely used, as knowledge and testing modalities have expanded.

Midwives are often involved in screening, diagnosis, counseling about prevention, and obtaining physician consultation as needed. The *Reproductive Tract and Sexually Transmitted Infections* chapter reviews prenatal considerations for women who are exposed to or develop syphilis, hepatitis B, herpes, human immunodeficiency virus (HIV), human papillomavirus (HPV), and Zika virus. This section reviews the other clinically significant infections associated with adverse perinatal outcomes.

Cytomegalovirus

Cytomegalovirus, a member of the herpesvirus family, is the most common cause of infection-related congenital disorders. Transmission of CMV occurs by sexual contact and direct contact with body fluids. Toddlers are the most frequent source of infection. CMV is ubiquitous in the environment, and approximately 50% to 80% of reproductive-age women in the United States have experienced an asymptomatic infection and already possess antibodies.[74] However, an initial in utero infection with CMV can cause congenital anomalies, such as fetal growth restriction, microcephaly, hepatosplenomegaly, and thrombocytopenia. Hearing loss, vision impairment, and learning disabilities are the most common sequelae. Approximately 1 in 200 newborns are born with congenital CMV infection but only about one-fifth of them will have health problems related to the infection.[74]

Women with CMV are typically asymptomatic. Routine screening for this virus is not recommended because serologic tests for CMV are not specific. Thus, the first indication of CMV infection is usually signs of fetal compromise identified on ultrasound.[75] Congenital infection can be confirmed via culture or polymerase chain reaction (PCR) analysis of amniotic fluid. Diagnosis of CMV infection in the pregnant woman may be confirmed via PCR analysis of blood, urine, or saliva, although serology is the most commonly used approach. Serum samples collected 3 to 4 weeks apart are tested in parallel for anti-CMV immunoglobulin G (IgG). A four-fold increase or seroconversion from negative to positive will confirm the diagnosis.

There is currently no treatment for CMV infection. Women with suspected CMV infection are referred to a maternal–fetal medicine specialist for care.[75] Women concerned about CMV are counseled to wash hands carefully when around children and when handling body fluids from children.

Listeriosis

Listeria monocytogenes is an intracellular facultative Gram-positive bacterium that grows in

contaminated meat, dairy products, and cheese.[76] The source of the infection is usually soil and decaying vegetable matter that contaminates nonpasteurized products or processed food after pasteurization. Although *Listeria* outbreaks are uncommon in the United States, the rate of infection in pregnant women is 13 times higher than that in the general population.[76]

Listeriosis is generally a mild, self-limited illness. When symptoms are noticed, they generally take the form of gastrointestinal complaints and diarrhea followed by flu-like symptoms. The incubation period for this infection is 24 hours to 10 days after exposure to the pathogen. Fetal risks include miscarriage, preterm labor, and stillbirth, because the bacterium has a predilection for the placenta and fetal central nervous system.[76-78] The perinatal mortality rate following in utero infection is 29%.[77] Prevention is the primary strategy for protecting pregnant women from listeriosis. Foods that have a high risk of being contaminated are listed in the *Prenatal Care* chapter.

Treatment of women with suspected listeriosis is based on presumptive exposure and symptoms. If the woman has a presumptive exposure but no symptoms, no testing or treatment is recommended. If she has a presumptive exposure, management is based on whether she is febrile. Expectant management is offered to women who are not febrile, and blood cultures may be considered. For women who are febrile, blood cultures and treatment with intravenous ampicillin are recommended.[79]

Parvovirus B19

Parvovirus B19 is a single-stranded DNA virus that is responsible for the childhood infection known as “fifth disease”—also called “slap-cheek fever” because the hallmark of this infection is bright red erythema that develops on both cheeks. Seasonal outbreaks occur in early winter. Approximately 50% to 60% of pregnant women are immune to parvovirus B19. The primary source of infection is young children who contract the illness, and the virus is transmitted via respiratory secretions or hand to mouth contact. Approximately 20% to 30% of susceptible women exposed to parvovirus B19 in a nursery school or school classroom will become infected. The incubation period is 4 to 14 days (but can be as long as 20 days) after exposure; during this time, the infected individual is contagious.[80]

Parvovirus causes a mild flu-like illness in women, but may also be asymptomatic. The rate of maternal-to-fetal transmission ranges from 17% to 33%.[80] This virus crosses the placenta and the virus attaches to an antigen on fetal red blood cell precursors and invades them. When the fetus is infected in the first trimester, the risk of death is approximately 10%. When this illness is contracted in the second trimester, the fetus can develop severe anemia, hydrops, high-output cardiac failure, and liver damage. Fetal hydrops secondary to parvovirus accounts for approximately 10% to 25% of all fetuses identified with non-immune hydrops.[80]

Universal screening for parvovirus B19 or targeted screening in low-risk women is not recommended. Women should be counseled to report any exposure to this virus as soon as possible. Management of women who are exposed to parvovirus B19 starts with documenting the date of exposure and obtaining a serologic test for anti-parvovirus antibodies. Immunoglobulin G and M (IgG and IgM) titers should be obtained as soon as possible. If these initial titers are negative, follow-up titers should be drawn to determine whether

seroconversion has occurred. If the woman has positive IgG and negative IgM tests within the first few days after exposure, she can be reassured that she is immune and not at risk for transplacental transmission.[75] Interpretation of laboratory tests for this disorder are outlined in **Table 23-11**.

Table 23-11 **Interpretation of Lab Results for Maternal Parvovirus B19 Serology**

IgG	IgM	Interpretation	Clinical Management
Negative	Negative	Nonimmune and no infection	Repeat tests in 14 days[a]
Negative	Positive	Current active infection	Weekly ultrasound to monitor for fetal hydrops
Positive	Negative	Past infection with immunity and fetal infection very unlikely	Ultrasound to determine whether this was a recent infection in which the IgM has dissipated but the fetus was infected
Positive	Positive	Current active infection	Weekly ultrasound to monitor for fetal hydrops

Abbreviations: IgG, immunoglobulin G; IgM, immunoglobulin M.

[a] There is a window of 7 days immediately after infection during which both IgG and IgM are negative.

If the fetus has not developed hydrops by 8 weeks after exposure, infection is highly unlikely. Thus, the fetus is monitored with serial ultrasounds every 1 to 2 weeks for

approximately 8 to 10 weeks after exposure.[75] Fetal infection can be confirmed via PCR analysis of amniotic fluid.

Rubella

The rubella virus—a single-stranded RNA virus—is the organism responsible for "German measles," or what was once called "three day measles" in contrast to rubeola. Population-level immunization has virtually eliminated maternal rubella infection and congenital rubella syndrome in the United States, but this risk should not be forgotten and outbreaks can and do still occur.

Rubella is transmitted via droplets from infected humans. The incubation period is approximately 12 to 23 days. Viremia is present approximately 7 days before and 7 days after the infection, during which time the individual is contagious. Prodromal symptoms including fever, malaise, and adenopathy, especially in the postauricular nodes, appear first. The characteristic rash is a maculopapular eruption and persists for 1 to 3 days. Approximately 25% to 50% of children with rubella will have a subclinical or asymptomatic course. In adults, the disease is more symptomatic, with 70% developing arthralgia or arthritis in addition to the other symptoms.

Fetal infection rates vary over the course of pregnancy, being high in the first and third trimesters (80% and 100%, respectively) and somewhat lower in the second trimester (25%).[81] Rubella spreads through the circulatory system of the fetus, causing damage to all organs. Deafness, cataracts, and cardiac defects are the classic triad of congenital anomalies referred to as *congenital rubella syndrome*, but the infection is systemic and can affect almost all fetal organs; thus, miscarriage, preterm birth, fetal growth restriction, and stillbirth are possible. The severity of the disease depends on the time of infection, with the highest risks occurring with infection before 18 weeks' gestation.[81]

A pregnant woman who is nonimmune and potentially exposed to rubella is referred for physician consultation. The diagnosis of rubella may be confirmed if the woman is symptomatic and pharyngeal samples are obtained when the virus is present. Interpretation of serology is complicated.

- The presence of IgM is not used for detection of this viral illness. IgM first appears approximately 5 days after the rash develops, but may be falsely positive, as it can appear in response to other infections.
- When IgG is not detected, susceptibility to infection is established and subsequent testing occurs 2 to 3 weeks later to determine whether infection has occurred.
- The presence of IgG does not indicate whether the infection was recent or remote. Specialized reference laboratories can conduct avidity testing, which measures the strength of the antibody response—this strength increases over time. These results can aid in establishing the timing of infection.
- A four-fold increase in IgG is diagnostic.

Fetal infection can be confirmed via culture of amniotic fluid, but ultrasound findings are not

specific. Depending on the gestational age, timing of infection, risk of fetal infection, and maternal desires, pregnancy termination may be offered.

Population-level vaccination in the United States has made rubella infection during pregnancy rare. Occasionally, a woman will receive the vaccine for rubella during pregnancy before she is aware of the pregnancy. Rubella vaccine is a live-attenuated vaccine, so there is a theoretical risk that the vaccine could cause infection. Nevertheless, there are no documented cases of rubella infection or congenital rubella infection following rubella vaccination during pregnancy. Women should be counseled about the theoretical risks, but rubella vaccination is not considered a reason to terminate the pregnancy.[82] In any case, the best prophylaxis for rubella is universal vaccination, if not long before pregnancy, at least during the postpartum period.

Toxoplasmosis

Toxoplasma gondii is an obligate intracellular protozoan parasite that infects humans and usually remains dormant in neural or muscle tissue for the lifetime of the infected individual. Exposure to infection occurs via contact with contaminated cat feces, water, fruits and vegetables, and gardening in affected soil. A population study in the United States reported that 85% of women of childbearing age are susceptible to infection. Vertical transmission in pregnancy is possible and most likely to occur during primary infection. The transmission rate increases from 10% in the first trimester to 25% in the second trimester, and then to 60% in the third trimester.[75]

Maternal infection is usually asymptomatic and most newborns are asymptomatic at birth. Unfortunately, 90% of children infected with *T. gondii* in utero will subsequently develop visual impairment, hearing loss, and developmental delay. Women with a potential exposure to toxoplasmosis are referred for physician consultation.

Interpretation of serology for toxoplasmosis is difficult and standard assays are not available in all laboratories.

- Negative IgM and IgG tests indicate absence of infection or recent infection prior to seroconversion. In this case, a repeat evaluation in 2 to 3 weeks is indicated.
- If IgM is present, the timing of infection cannot be determined because the protozoa can remain in sera in high levels for several months or years. IgM can also be falsely positive. However, if the IgM test is positive and the IgG test is negative initially, and the IgG appears in a test performed 2 weeks later, recent infection can be reliably confirmed.
- The presence of IgG and no IgM indicates that infection has occurred but does not establish the timing of infection. A reference laboratory can perform avidity testing to determine whether the infection was recent or remote.
- The presence of both IgG and IgM does not determine the timing of infection. IgG develops rapidly in the case of toxoplasmosis, and IgM may be detectible for a long time.

Additional specialized tests are available for detection of maternal infection. Ultrasound may show early fetal effects, and amniotic fluid culture can establish the diagnosis of

congenital toxoplasmosis.[75] Routine screening for toxoplasmosis is not recommended in the United States because the prevalence is low and standardized serologic assays are not available. In parts of Europe, serial testing for susceptible women is cost-effective and a standard component of care; in contrast, the low prevalence of toxoplasmosis in the United States does not make this testing a cost-effective prevention strategy.

Treatment of infected women involves administration of spiramycin (Rovamycine), a macrolide antibiotic and antiparasite that is not commercially available in the United States but can be obtained at no cost from the U.S. Food and Drug Administration or special toxoplasmosis testing laboratories. If fetal transmission has not yet occurred, the woman can take spiramycin for the rest of the pregnancy to reduce transmission. If maternal seroconversion develops after 18 weeks' gestation or if fetal infection is confirmed, a complex regimen of specialized antibiotics may reduce the severity of disease in the neonate. Monthly monitoring is indicated throughout pregnancy. Primary prevention counseling is a standard component of routine prenatal care.

Varicella

The varicella zoster virus causes chickenpox and shingles. In the United States, approximately 90% of women of childbearing age are immune to varicella. The incubation period is 10 to 21 days following exposure to an infected individual. The characteristic rash appears as vesicles on the face, trunk, and extremities following a prodromal period of fever, malaise, and myalgia.

The diagnosis of varicella is made clinically, based on the characteristic rash. The viral DNA can be detected via PCR testing of skin at the base of vesicles. Serology interpretation can be difficult, however.

Primary infection in pregnancy is associated with severe maternal disease that can be either uncomplicated or complicated. The complicated form includes pneumonia, which has a mortality rate as high as 40% and is considered a medical emergency.[75] Women with varicella during pregnancy are offered antiviral medication (acyclovir [Zovirax] 800 mg 5 times per day for 7 days), which should be initiated as quickly as possible. Women with complicated varicella infection require hospitalization and specialized care.

The risk of vertical transmission of varicella virus to the fetus is highest in the first trimester of pregnancy, but decreases thereafter; overall, the risk of vertical transmission is approximately 2%. If women develop varicella infection 1 to 4 weeks before giving birth, as many as 50% of newborns will acquire the infection. The mortality rate in infected infants is approximately 30%.[83] Fetal effects of in utero infection include fetal growth restriction, low birth weight, skin lesions, limb hypoplasia, abnormalities such as microcephaly and hydrocephaly, and cognitive impairment.[83]

If a pregnant woman reports exposure to varicella, the first step is to determine if she has a history of vaccination or disease. If she has been vaccinated or has contracted varicella, she is immune and the fetus is not at risk. If her immunity is not known, the second step is to assess the degree of exposure—defined as a household contact, face-to-face contact for more than 5 minutes with an infected individual, or sharing the same hospital room with a contagious

person. If the woman does not have a known history of vaccination or disease, a varicella titer should be obtained. Nonimmune women who are exposed to varicella during pregnancy should receive varicella zoster immune globulin (VZIG), which can be administered up to 10 days after exposure.[75]

Neurologic Conditions

Numerous neurologic conditions may precede or develop during pregnancy. Midwifery care of women includes diagnosing and treating neuropathies such as carpal tunnel syndrome and Bell's palsy, as well as caring for women with migraine headaches during pregnancy.[84] A neurologic examination and interpretation of findings are within the midwifery scope of practice. Women who have complex neurologic illnesses such as multiple sclerosis or seizure disorders require physician care during pregnancy.

Migraine Headache

Headaches are common and can be a symptom of a wide range of disorders, some of which are benign and some of which can be life threatening. As a first consideration, whenever a woman presents with a headache in pregnancy, preeclampsia should be ruled out. Women with headaches that are atypical or concerning warrant prompt physician evaluation.

Migraines are a complex neurovascular brain disorder that affects approximately 18% to 20.2% of women during their childbearing years.[85,86] Many nonpregnant women experience an increase in migraine symptoms in conjunction with their menstrual cycle. The physiologic state of pregnancy with its sustained hormone levels—versus the variability in hormone levels seen during the menstrual cycle—can reduce the frequency of migraines. Many women who have migraine headaches report that the migraines improve or cease during pregnancy. A small proportion experience no change in frequency, and approximately 7% experience new onset of migraines during pregnancy. Improvement is most likely in the second and third trimesters.[87]

Migraine headaches are usually unilateral and may be associated with photophobia or phonophobia, nausea, and vomiting. These headaches are often induced by triggers such as sleep deprivation, stress, or certain foods.[85,86] Migraines do not adversely affect the course of pregnancy, although some studies suggest women who have migraines may have an increased risk for preeclampsia and low-birth-weight infants.[87]

Care of women with migraines during pregnancy starts with avoidance of migraine triggers, adequate fluid and rest, and use of acetaminophen. Formulations that include butalbital, acetaminophen, and caffeine are considered safe for short-term use (4–5 days per month). Triptans have vasoconstrictive effects that could theoretically cause vasoconstriction in the placenta, but sumatriptan (Imitrex) has been studied in pregnant women and not found to be associated with adverse effects.[85,86,88] Ergotamine (Cafergot) is contraindicated during pregnancy due to fetal hypoxia and potential oxytocic type effects. Women who need other medications or additional therapy are best cared for by a physician.

Peripheral Neuropathies

Pathology of the nerve roots may be acquired or inherited. Incidence of peripheral neuropathies during pregnancy is infrequent, especially in women without diabetes. The most common neuropathies that occur in pregnant women include Bell's palsy, carpal tunnel syndrome, and sciatica.

Bell's Palsy

Bell's palsy—a peripheral neuropathy affecting the face unilaterally—is more common among women than men, and is three times more common among pregnant women than among nonpregnant women. This condition is most likely to occur in the third trimester.

Symptoms of Bell's palsy may include pain behind the ear, increased perception of loudness, altered or lost taste of food, drooling, and difficulty with smiling or eye movement. The condition is painful; reports of dry eye (because the eyelid does not completely close), oversensitive hearing, changes in taste, and headache are common. On examination, the face is smooth and the affected side of the mouth droops. One test is to ask the woman to wrinkle her forehead, which she cannot do if Bell's palsy is present.

Management in pregnancy is limited to supportive treatment, and occasionally use of corticosteroids. Physician consultation can aid in making a plan for care, and referral to a neurologist may be of benefit. The eyelid may not close so keeping it moist and protected is important.

Carpal Tunnel Syndrome

Pregnancy is a known risk factor for carpal tunnel syndrome. Increased fluid retention and effects of relaxin have been reported to play a role in the increased frequency of this condition among pregnant women.[89] The presentation of carpal tunnel syndrome includes nocturnal or activity-related hand paresthesias, with nearly two-thirds of women reporting concurrent pain.[89],[90] The condition is more likely to be bilateral in pregnancy when compared to the nonpregnant population.

Treatment consists of wrist splints and local anesthesia injections as indicated. Surgery during pregnancy may be appropriate but generally is reserved for severe cases. Physical therapy can be recommended for additional support with carpal tunnel syndrome.[89]

Meralgia Paresthetica

Meralgia paresthetica is caused by compression of the lateral cutaneous femoral nerve. In pregnancy, it is associated with weight gain, obesity, diabetes, or a large fetus.[90] Affected women may complain of burning, uncomfortable pain, tingling, and numbness over the lateral thigh. Diagnosis is usually possible with careful examination and does not require unnecessary testing.[90] Protective measures include frequent position changes, along with avoidance of prolonged hip flexion or rotation and thigh abduction. Symptom relief may be accomplished by abdominal binders and avoiding tight clothing. Symptoms are typically self-limiting and resolve postpartum. Meralgia paresthesia is not a contraindication for regional anesthesia.[90]

Pelvic Girdle Pain: Sciatica and Symphyseal Separation

Low back pain, symphyseal pain, and sacroiliac joint pain are all common in pregnancy. These conditions are related to musculoskeletal changes of pregnancy and pressure on pregnancy-involved joints, although peripheral nerves can be entrapped and cause pain that radiates down

the back of the buttocks or legs. Pelvic girdle pain can include low back pain, or it may present as isolated pain over one of the pelvic joints. Sciatic pain may occur secondary to hormonal influences during pregnancy or secondary to a herniated disc. In most cases, the various presentations of pelvic girdle pain resolve spontaneously after birth, although low back pain can persist.

Muscle relaxation, rest with legs flexed, avoiding prolonged standing, and abdominal girdles may provide some relief. If the woman has severe pain or difficulty moving, physical therapy may be helpful. Women who have severe pain or other neurologic symptoms should be referred to a physician for consultation.

Epilepsy

Seizure disorders have a range of manifestations and causes, but are collectively called epilepsy.[91] Epilepsy is one of the most common neurologic conditions encountered during prenatal care.[92] In this disorder, abnormal electrical activity in the brain causes altered sensation, lapses in attention, or seizures. Seizure activity may be stimulated by brain trauma, infection, tumors, or specific triggers such as bright lighting.[91] Although epilepsy is most often treated with medications, other treatments include surgical excision of the brain seizure foci, ketogenic diets, and implantable brain neurostimulators.[91]

Ideally, women with epilepsy would seek preconceptional counseling that focuses on medication optimization. Women without a personal or family history of neural tube defects are advised to begin 400 mcg of oral folic acid daily and continue this dose during pregnancy.[93] Daily folic acid is increased to 4 mg orally starting one month before conception for women with a personal or family history of neural tube defect. This dose is also recommended by some clinicians when women use antiepileptic drugs associated with neural tubes defects, including valproic acid (Depakote, Convulex) and carbamazepine (Tegretol).[93]

Infants born to women with epilepsy have a 4% to 14% risk of having a major congenital malformation. Epilepsy-associated malformations include cleft lip and palate, ventricular septal defect, neural tube defects, hypospadias, club foot, and facial abnormalities such as flattened nasal bridge.[93,94] This increased risk of congenital anomalies is likely secondary to the effects of antiepileptic drugs, but the disorder itself may account for some of the increased risk.

One million women of childbearing age are estimated to have epilepsy, with approximately 24,000 infants born to these women annually.[93] Although some antiepileptic drugs are known teratogens, more than 90% of women with epilepsy give birth to healthy newborns. Valproate (Depakote, Convulex) and phenobarbital, which are known to increase the risk of congenital malformations and cognitive impairments, should be avoided when possible during pregnancy.[94,95] Lamotrigine (Lamictal) and levetiracetam (Keppra) have not been associated with congenital malformations.[94] Adjustment of preconception medication to monotherapy (instead of polytherapy) is preferred, because polytherapy is associated with a higher risk of congenital malformations and long-term cognitive deficits in offspring.[92-94] Antiepileptic drugs are cleared more quickly during the second and third trimesters, so serum levels of these

agents are monitored regularly during pregnancy.[93] Midwives are encouraged to participate in the Antiepileptic Drug Pregnancy Registry, which is listed in the Resources section at the end of this chapter.

Estrogen and progesterone affect neuronal excitability, thereby changing seizure thresholds. Approximately 15% to 32% of women with epilepsy will have changes in seizure type, frequency, or severity during pregnancy, although at least half of women experience no change in seizure activity.[93] The best predictor of remaining seizure free during pregnancy is having been seizure free for 9 months prior to pregnancy.[93] Compared to women without epilepsy, women with epilepsy during pregnancy are at higher risk for spontaneous miscarriage, preterm labor, preeclampsia, small-for-gestational-age newborns, fetal growth restriction, induction of labor, cesarean birth with prolonged hospital stay, and excessive postpartum bleeding.[92-95]

Respiratory Conditions

Asthma

Asthma affects as many as 8% of pregnant women in the United States and is often undertreated in adults. The course of asthma during pregnancy is variable, with approximating one-third of women experiencing exacerbations, one-third having fewer episodes, and one-third not experiencing any difference in asthma symptoms or frequency.[96] Overall, acute exacerbations during pregnancy occur in 20% to 36% of women with asthma.[97] The more severe or poorly controlled a woman's asthma is in the year prior to pregnancy, the higher the risk she has of worsening disease and hospital admission during pregnancy.[98]

The effect of asthma on pregnancy is not fully established. However, it appears to be associated with a small increase in various pregnancy complications such as low birth weight, preeclampsia, and preterm birth, but it is not clear if these risks are secondary to asthma, an underlying condition that affects both asthma and pregnancy, or the effects of medications used to treat asthma.

Research consistently demonstrates undertreatment of asthma by providers as well as underutilization of asthma medications by women during pregnancy. In addition, pregnant women with asthma who visit emergency departments for this disease are less likely to have appropriate medications prescribed than are nonpregnant individuals with asthma who receive emergency department care. Effective management of asthma during pregnancy requires vigilance, and multidisciplinary collaborative care is the optimal model for care for achieving this goal.[98]

When initially evaluating a woman who has asthma, it is important to determine the classification of the woman's asthma (see the *Common Conditions in Primary Care* chapter) based on current National Asthma Education and Prevention Program guidelines.[98] Assessment includes obtaining data about the severity, number of episodes, hospitalizations, and medications used in the prior year. Management goals are to (1) prevent chronic symptoms, (2) maintain optimal pulmonary function to support normal levels of activity, (3) prevent exacerbation, (4) maintain fetal oxygenation by preventing maternal hypoxic episodes, and (5) use treatment with no or minimal side effects.[97,98]

Evaluation of asthma during pregnancy is based on clinical evaluation and peak expiratory flow measurements. A stepwise approach is recommended for treating women with asthma, as described in **Table 23-12**.[98] Treatment should be stepped up when control is not achieved and stepped down if the woman has experienced resolution of symptoms for 3 months or more.

Table 23-12 Management of Asthma During Pregnancy

Asthma Classification	Step Therapy for Treating Women with Asthma During Pregnancy
Mild intermittent	No daily medications Albuterol inhaler as needed
Mild persistent	Low-dose inhaled corticosteroid

Moderate persistent	Low-dose inhaled corticosteroid and salmeterol (Advair) or medium-dose inhaled corticosteroid is preferred
Severe persistent	High-dose inhaled corticosteroid and salmeterol (Advair); oral corticosteroid if needed

Note: Alternative therapies are available for each step. If an alternative medication is needed, physician consultation should be obtained.

Based on National Asthma Education and Prevention Program. Working group report on managing asthma during pregnancy: recommendations for pharmacologic treatment. Available at: https://www.nhlbi.nih.gov/files/docs/astpreg_qr.pdf. Accessed November 4, 2017.[98]

While some women may be concerned about using asthma medication out of fear of fetal harm, a fetus needs oxygenation. Therefore, it is safer for pregnant women to be treated for asthma than to have asthma symptoms and complications in pregnancy. There are no documented adverse side effects of the medications for asthma, although only limited data are available on the use of antihistamines in pregnancy.[97,98]

Women with asthma in pregnancy will benefit from health education and preventive health strategies, including information on how to avoid triggers, how to take their medications, cessation of smoking, danger signs of worsening disease, and the importance of fetal movement counts.

Tuberculosis

When pregnancy and tuberculosis coexist, poor outcomes may result.[99] Pregnancy suppresses the pro-inflammatory response, which increases both susceptibility to new infection and reactivation of tuberculosis. Thus, pregnant women are at increased risk for active tuberculosis, and those with latent tuberculosis infection are more likely to progress to active tuberculosis. Outcomes of the disease in pregnancy vary, making the course of the disease difficult to predict. The prevalence of tuberculosis in pregnancy is estimated to be between 0.06% and 0.25% in high-resource countries.[100]

Risk factors for tuberculosis are reviewed in the *Common Conditions in Primary Care* chapter. Individuals with active infection may be asymptomatic. Symptoms include a prolonged, productive cough; fatigue; fever; chills; and night sweats.[101] Compared to pregnant women without tuberculosis, those with the disease have an increased risk of maternal morbidity, anemia, preterm birth, cesarean birth, low-birth-weight infants, birth asphyxia, and perinatal death.[101] Active tuberculosis is also associated with maternal mortality, with the highest rates of death occurring in women coinfected with tuberculosis and HIV. Infants born from women with tuberculosis can contract congenital tuberculosis; in such a case, they may either be asymptomatic or have birth defects.[100]

The Centers for Disease Control and Prevention (CDC) recommends screening pregnant women who are at high risk for tuberculosis, including women with known or suspected contact with a person who has tuberculosis, recent residence in an area where tuberculosis is prevalent (e.g., homeless shelters, countries with a high prevalence of tuberculosis), work in a high-risk setting such as a correctional facility or a hospital, use of injectable drugs, HIV, or immunosuppression.[102,103] Tuberculosis screening during pregnancy may be done with either

the intradermal tuberculin screening test (TST; also referred to as purified protein derivative [PPD]) or the interferon-gamma release assay (IGRA), which uses a serum sample. Both the TST and the IGRA are recommended by the CDC, both measure the body's response to tuberculin, and both are used in pregnancy. For the TST, population factors affect the measurement for determining positivity, whether the woman is pregnant or not (as noted in the *Common Conditions in Primary Care* chapter). When either test is positive, it usually means the woman has been infected with tuberculosis, but a differential diagnosis of latent tuberculosis or active tuberculosis remains to be made.[102,104]

An important confounder in the diagnosis of tuberculosis is the tuberculosis vaccine used in other countries, called bacillus Calmette-Guerin (BCG), which can potentially confer a lifelong positive response to tuberculosis tests. Women who have received BCG can still acquire tuberculosis. Tuberculin testing of women who have received BCG is acceptable if indicated and a positive result requires follow-up to confirm the diagnosis.

When a tuberculosis test is positive, a posterior–anterior chest X ray using shielding can be performed to detect the presence of tuberculosis lesions in the lung. A negative finding rules out active disease, and a diagnosis of latent tuberculosis is then made. Another option is obtaining a sputum for a culture. If the culture is positive for *Mycobacterium tuberculosis*, tuberculosis is confirmed.

Treatment of active tuberculosis in pregnancy is the same as treatment for nonpregnant women.[105,106] Previously, treatment of latent tuberculosis infection was delayed until 3 months postpartum. Currently, it is recommended that treatment be initiated at the time of diagnosis for women with either active or latent tuberculosis, although women with latent tuberculosis infection who do not have recent infection or immunocompromise may delay treatment until after birth.[102,103,106] The CDC recommends 9 months of isoniazid (Nydrazid) with pyridoxine (vitamin B_6 supplement). Either a daily or twice-weekly regimen may be used. Treatment with rifampin (Rifadin, Rimactane), either in combination with isoniazid or alone, is not recommended in pregnancy. Women with tuberculosis are best cared for by physicians with specialist training.

Upper Respiratory Infections

The diagnosis and management of upper respiratory infections (URIs) are described in detail in the *Common Conditions in Primary Care* chapter. In general, symptoms and the course of care for individuals with URIs are the same for pregnant women as for those who are not pregnant. The hormonal shifts and changing body mechanics of pregnancy can affect respiratory function, reducing lung volume and increasing activity in mucosal membranes. Thus, pregnant women may experience more intense symptoms or have a longer delay before health is restored. Although pregnant women may experience dyspnea, tachypnea is never a normal sign of pregnancy. Most importantly, women who are pregnant with a URI are more likely to progress from a cold, bronchitis, or influenza to pneumonia. The differential diagnoses when women have symptoms of URI include the common cold, influenza, bronchitis, sinusitis, and pneumonia.

Common Cold

Pregnant women are as susceptible to the common cold as nonpregnant women. Symptoms include rhinitis, sore throat, cough, and malaise, but adults rarely have a fever. The only difference in progression of this disorder during pregnancy is that a cough may take several days longer to resolve in pregnant women. Treatment focuses on mitigating symptoms. Decongestants, both oral and nasal sprays, are the usual treatment for women who have a URI and are not pregnant. Studies have failed to identify strong teratogenic effects associated with these agents; however, many midwives suggest nonpharmaceutical treatments and use of drugs only when the former fail to provide relief. Dextromethorphan, which is available under many brand names, is an effective antitussive and can be used to treat a woman with a cough.

Sinusitis

Pregnancy-related hormonal effects on the nasal passages increase a woman's risk for developing sinusitis following a common cold. Most cases of sinusitis are viral in origin. Symptoms include nasal congestion, faciomaxillary pain, headache, and facial pain or pressure that is worse when the woman bends forward. During pregnancy, symptoms of sinusitis may be atypical but often appear as a worsening of symptoms of a common cold that was resolving. This disease is generally self-limiting and will resolve in 7 to 10 days. Antibiotic therapy can be initiated if symptoms worsen or do not resolve. The first-line agent is amoxicillin–clavulanate (Augmentin) 875 mg/125 mg administered twice daily assuming the woman is not allergic to penicillin. A higher-dose formulation is available for women with severe infection or risk factors for developing pneumonia.

Bronchitis

Acute bronchitis often follows a common cold and should be considered if a cough persists more than 5 days. Bronchitis is an inflammation of the bronchi generally caused by a viral infection. Women with bronchitis will have clear lungs during auscultation but may have purulent sputum. The color of the sputum does not indicate the presence of bacteria. The differential diagnoses include pneumonia, asthma, and pertussis. Treatment is based on symptoms and antibiotics are not recommended.

Influenza

Annual influenza vaccination is recommended for all adults, including pregnant women. Use of the injectable trivalent inactivated vaccine does not cause harm to women or their fetuses.[107] In addition, maternal vaccination reduces the incidence of infection among one of the highest-risk populations—namely, infants younger than 1 year of age. Influenza can be a serious respiratory disease in pregnant women and is associated with increased risk of morbidity and mortality for both the woman and fetus.[107] The symptoms of influenza are similar to the symptoms of the common cold, but are more severe and include a fever. Hyperthermia is associated with some congenital anomalies in the fetus.[108],[109] The diagnosis is usually made on the basis of clinical

findings but laboratory tests are available.

Treatment should be instituted as quickly as possible when influenza is suspected. Oral oseltamivir (Tamiflu) can be safely used during pregnancy and is preferred because it is systemically absorbed.[109] Acetaminophen to reduce fever is an important component of care. Adjunct medications to treat symptoms such as rhinitis are safe and can be used concomitantly with oseltamivir.

Pneumonia

The symptoms of community-acquired pneumonia among pregnant women are similar to those experienced by nonpregnant women, and include cough, shortness of breath, sputum production, chest pain, fever, and malaise. Tachypnea and tachycardia may be present. On examination, lung consolidation, elevated temperature, tachypnea, and signs of respiratory distress may be evident. Women with suspected pneumonia should have a chest X ray, a complete blood count (CBC), metabolic panel (electrolytes, blood urea nitrogen, creatinine, glucose), and blood gases. Because of the likelihood of rapid disease progression and fetal compromise from decreased oxygenation, hospitalization is often necessary when pneumonia occurs during pregnancy, especially during the third trimester.

In addition to the maternal morbidity directly related to infection, pregnant women with pneumonia have an increased risk of developing preeclampsia. Fetal risks include preterm premature rupture of membranes, fetal growth restriction, prematurity, low birth weight, and small-for-gestational-age infants, as well as morbidity from hypoxemia.

Women who have symptoms of pneumonia during pregnancy are referred for physician evaluation and consideration for hospitalization. Antimicrobial therapy is chosen based on the microorganism although treatment with macrolide and beta-lactam antibiotics is recommended. Fluoroquinolones and clarithromycin (Biaxin) should be avoided.

Thyroid Disorders

Thyroid disorders are common in women of childbearing age. Hyperthyroidism, which is characterized by increased levels of free thyroxine (T_4) and low levels of thyroid-stimulating hormone (TSH), is present in 0.5% of pregnant women. *Graves' disease* accounts for 95% of hyperthyroidism in pregnant women.[111]

In settings where iodine deficiency is rare, the prevalence of hypothyroidism, which is characterized by high TSH levels and low free T_4 levels, is approximately 0.3% among pregnant women.[110] Most hypothyroidism in pregnancy is a result of autoimmune thyroiditis, also called *Hashimoto's disease*.[110] Thyroid storm, a life-threatening condition of severe hyperthyroidism, is rare but often precipitated by an acute event such as parturition, trauma, or surgery. Caused by an excess of thyroid hormone, this condition develops abruptly and can lead to multiple-organ decompensation.

Maternal and fetal complications associated with hyperthyroidism and hypothyroidism are listed in **Table 23-13**.[110-112] In general, overt hyperthyroidism is associated with more severe fetal effects than is overt hypothyroidism. In addition, medications used to treat hyperthyroidism can cause congenital defects, whereas thyroid replacement medications are not associated with adverse fetal effects.

Table 23-13 **Complications of Hyperthyroidism and Hypothyroidism During Pregnancy**

Pregnancy Complications	Hyperthyroidism	Hypothyroidism
Maternal	Gestational diabetes	Abruptio placenta
	Heart failure	Gestational hypertension
	Liver injury secondary to propylthiouracil	Preeclampsia
	Preeclampsia	
	Thyroid storm	
Fetal	Congenital anomalies secondary to thyroid medication	Cretinism
	Fetal hypothyroidism secondary to thyroid medication (fetal goiter)	Lower IQ and psychomotor delay after birth
	Fetal hyperthyroidism secondary to fetal exposure to thyroid-stimulating receptor antibodies	Fetal growth restriction
	Fetal growth restriction	Low birth weight
	Hydrops	Miscarriage
	Low birth weight	Preterm birth
	Neonatal autoimmune hyperthyroidism (neonatal Graves' disease)	Stillbirth
	Preterm birth	
	Stillbirth	
	Tachycardia	
	Thyrotoxicosis	

Based on Stagnaro-Green A. Overt hyperthyroidism and hypothyroidism during pregnancy. *Clin Obstet Gynecol*. 2011;54(3):478-487[110]; Korevaar TIM, Medici M, Visser TJ, Peeters RP. Thyroid disease in pregnancy: new insights

in diagnosis and clinical management. *Nat Rev Endocrinol*. 2017;13(10):610-622[111]; Alexander EK, Pearce EN, Brent GA, et al. 2017 guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2017;27(3):315-389.[112]

Pregnancy-Related Changes in Thyroid Function

Diagnosis of thyroid disorders during pregnancy requires an understanding of the physiologic changes that occur in thyroid function under the influence of pregnancy hormones (see the *Anatomy and Physiology of Pregnancy* chapter). Serum levels of two thyroid hormones, thyroxine (T_4) and triiodothyronine (T_3), increase by 20% to 50%; TSH decreases; and thyroxine-binding globulin (TBG) increases substantially. Several interactions cause these changes. The alpha subunit of human chorionic gonadotropin (hCG) is structurally very similar to TSH and has thyroid-stimulating activity. As T_4 levels rise in response to the hCG stimulation of the thyroid gland, the pituitary releases TSH in response to the normal negative feedback between thyroxine levels and TSH. This creates a transient but physiologic hyperthyroidism. Estrogen induces a two-fold increase in TBG levels during pregnancy. Thyroxine bound to TBG is not active, so to maintain a euthyroid state, total T_4 must also increase.[113] Total T_4 increases in the first trimester and then falls in the second and third trimesters. Under normal conditions, the concentrations of free T_3 and T_4 do not actually change. T_3, the metabolically active hormone, is mostly made via conversion of T_4 in tissue.

Three other aspects of thyroid physiology changes are important considerations in clinical practice:

- The fetal thyroid does not produce T_4 until after 12 weeks' gestation, and does not produce enough to support fetal growth during the first half of gestation. Thus, the fetus depends on transplacental passage of maternal thyroxine until after the fetus is fully formed. T_4 is critical for early brain development, fetal growth, and bone maturation. There are critical windows during gestation for development of tissues that require thyroid hormone. Treatment of pregnant women with overt thyroid disorders is critical, therefore, and is best managed by a combination of specialists familiar with thyroid endocrinology.
- Iodine clearance increases during pregnancy. Iodine is essential for the production of T_4, so its intake needs to increase by approximately 50% in pregnancy. Although rare in the United States, where iodine supplementation in many basic food sources is common practice, iodine deficiency is an important cause of mental impairment worldwide.[113] This aspect of thyroid physiology can be an important consideration for women who have recently resided in an area where iodine deficiency exists. The recommended iodine intake during pregnancy is 220 micrograms per day. In the United States, most women achieve sufficient iodine intake via diet. Because iodine sufficiency is not universal, however, the American Thyroid Association recommends a daily supplement of 150 micrograms of potassium iodide; this amount is found in most (but not all) prenatal vitamins.
- When evaluating thyroid tests, the reference ranges for TSH and T_4 are trimester specific and population based, as there is significant geographic and ethnic diversity in TSH

concentrations during pregnancy. If local reference ranges are not available, the upper limit for TSH should be 4.0 mU/L (range: 0.1–4 mU/L).[112]

An overview of thyroid hormone levels during pregnancy in women with hyperthyroidism, hypothyroidism, and hyperemesis gravidarum is presented in **Table 23-14**.

Table 23-14 Overview of Thyroid Hormone Values During Pregnancy in Women with Thyroid Dysfunction

Thyroid Hormone	Normal Pregnancy	Overt Hyperthyroidism in Pregnancy	Overt Hypothyroidism in Pregnancy	Hyperemesis
Thyroid-stimulating hormone (TSH)	Normal	Suppressed or undetectable	High	
Thyroid-binding globulin (TBG)	Increased	Increased	Normal	Normal or low
Total thyroxine (T_4)	Increased	Increased	Low	Increased
Free thyroxine (FT_4)	Normal	Increased	Low	Increased
Total triiodothyronine (T_3)	Increased	Increased	Low	Normal or increased
Free triiodothyronine (FT_3)	Normal	Increased	Low	Increased

Hyperthyroidism

Table 23-15 presents an overview of management of thyroid disorders during pregnancy. Symptoms of hyperthyroidism are reviewed in the *Common Conditions in Primary Care* chapter. Women who have symptoms of hyperthyroidism or a history of hyperthyroidism should have thyroid function tests performed, but routine screening is not recommended.[114,115] Secondary to stimulation by hCG, approximately 2% to 15% of women will have laboratory values consistent with hyperthyroidism in the first trimester, and this finding is more frequent in women with hyperemesis gravidarum or severe nausea and vomiting. Sometimes called gestational transient hyperthyroidism, this condition resolves after the hCG levels normalize. It is not associated with adverse pregnancy outcomes and does not require treatment.

Table 23-15 Overview of Treatment of Thyroid Disorders in Pregnancy

	Hyperthyroidism	Hypothyroidism
Recommended treatment	PTU in first trimester Methimazole (Tapazole) after first trimester	Levothyroxine (Synthroid)
Monitoring during pregnancy	TSH, FT4 monthly With PTU: liver function tests monthly	TSH monthly through second trimester and once at beginning of third trimester
Postpartum evaluation	TSH, FT4	TSH, FT4

Abbreviations: FT_4, free thyroxin concentration; PTU, propylthiouracil; TSH, thyroid-stimulating hormone.

When hyperthyroidism is first diagnosed during pregnancy, referral for physician management is indicated. The more common scenario that midwives encounter in maternity care settings is one of a woman who has the diagnosis of Graves' disease and is being treated with propylthiouracil (PTU) or methimazole (Tapazole).

PTU has potential adverse effects for the pregnant woman, and methimazole has potential adverse effects for the fetus. PTU can cause a life-threatening hepatotoxicity but is more strongly protein-bound than is methimazole and does not cross the placenta as easily.[114,115] Methimazole is associated with a small increased risk for congenital anomalies, such as choanal atresia, tracheo-esophageal fistula, and omphalocele. Thus, it is recommended that women with Graves' disease use PTU in the first trimester and then switch to methimazole after the first trimester.[112,114,115] Once medication is started, thyroid function is monitored every 2 to 4 weeks by following serial free T_4 levels and adjusted as needed.

Hypothyroidism

Hypothyroidism is more common than hyperthyroidism in pregnant women. Subclinical hypothyroidism, defined as an elevated TSH with normal free T_4 levels, is present in 2% to 5% of women during pregnancy.[115] A few retrospective studies, published in the 1990s, found that subclinical hypothyroidism during pregnancy was associated with impaired neurodevelopment in the child. These findings supported the call for universal screening for hypothyroidism during pregnancy. The data supporting the association between subclinical hypothyroidism and adverse neonatal outcomes were not conclusive, however, and universal screening remained controversial until 2012, when the Controlled Antenatal Thyroid Screening Study (n = 21,846) was completed.[116] This randomized trial screened women for subclinical hypothyroidism and followed women in two groups: One group was treated, while the other group received usual care without treatment. The study found no difference in neurocognitive development at age 3 years in the offspring when the children from both groups of women were compared.[116] Thus, the call for universal screening for hypothyroidism during pregnancy was quelled, and this practice is no longer recommended. Currently, the American College of Obstetricians and Gynecologists recommends screening women who have symptoms, a personal history of thyroid disease, or the finding of a goiter or thyroid nodules on physical examination.[115] The American Thyroid Association has a longer list of recommended criteria for screening as noted in **Table 23-16**.[112,117]

Table 23-16	Factors That Increase the Risk of Thyroid Disease in Pregnancy
Living in an area with known moderate to severe iodine insufficiency	
Family history of thyroid disease	
Past Medical History	
Autoimmune disorder	

Elevated thyroid peroxidase antibodies
Infertility
Irradiation of head or neck or multiple dental X rays
Multiple miscarriages
Pregnancy loss or unexplained preterm birth
Type 1 diabetes mellitus
Thyroid antibody positivity
Thyroid disease or thyroid surgery
Treatment with lithium (Lithobid), amiodarone (Cordarone), or recent administration of a radiologic contrast agent
Physical Examination Findings
Symptoms or clinical signs of thyroid disease
Goiter
Age ≥ 30 years
Body mass index (BMI) ≥ 40 kg/m^2

Based on Alexander EK, Pearce EN, Brent GA, et al. 2017 guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2017;27(3):315-389[112]; De Groot L, Abalovich M, Alexander EK, et al. Management of thyroid dysfunction during pregnancy and postpartum: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2012;97(8):2543-2565.[117]

Diagnosis and Treatment of Hypothyroidism

The initial screening test used to establish thyroid dysfunction is TSH level. In many settings, a TSH and free T_4 level will be obtained at the same time because the addition of the free T_4 level is useful to identify the diagnosis of hypothyroidism. The total T_4 level is not a useful measure in pregnancy because of the increased protein binding that occurs during gestation. If the TSH level is higher than 4.0 mU/L or the population-specific upper limit, treatment with levothyroxine is initiated. Levothyroxine is titrated to maintain TSH in the normal range by trimester. For women diagnosed prior to pregnancy, an increase in the dose by as much as 30% may be needed early in pregnancy. Laboratory monitoring of TSH levels should be performed every 4 weeks until the second half of pregnancy, and then at least once at the beginning of the third trimester.[112]

If the TSH level is higher than 2.5 mU/L but less than 4.0 mU/L and the woman is euthyroid (i.e., free T_4 level in the normal range), a blood test to determine the absence or presence of thyroid autoantibodies (TBOAb) may be recommended. Some studies have found that women with subclinical hypothyroidism (i.e., euthyroid but moderately high TSH levels plus thyroid autoantibodies) have an increased risk for adverse outcomes, but evidence that treatment prevents these outcomes is lacking. Current guidelines from the American Thyroid Association recommend following TSH levels in pregnant women who are euthyroid with positive TBOAb every 4 weeks through mid-pregnancy. Treatment is not recommended in general,[112] but physician consultation is appropriate because guidelines differ by region, population, and individual clinical circumstances.

Recommendations for evaluation of thyroid nodules in pregnancy include serum TSH testing

and a neck ultrasound. Women with abnormal findings may be referred to a specialist.[112]

Urinary Disorders

Women, relative to men, have an increased risk for developing urinary tract infections (UTIs). Such infections, which are common in pregnancy, include asymptomatic bacteriuria, cystitis, recurrent UTI, and pyelonephritis. In addition to pregnancy itself, other risk factors for UTIs include low socioeconomic status, sickle cell disease, previous UTI, and pregestational diabetes. Pregnancy-related changes to the urinary tract predispose women to pyelonephritis once a UTI is present.[118] Diagnostic criteria for urinary disorders are presented in the *Common Conditions in Primary Care* chapter.

Asymptomatic Bacteriuria

The incidence of asymptomatic bacteriuria is similar in pregnant and nonpregnant women, with prevalence rates ranging from 2% to 13%. The pathogen most likely to cause this infection is *Escherichia coli*, which accounts for 63% to 85% of such infections. The combination of having no overt symptoms commonly associated with symptomatic UTI and a clean-catch urine culture that has isolated 100,000 (10^5) colonies or more of no more than two microorganisms is diagnostic for asymptomatic bacteriuria.[119]

Untreated asymptomatic bacteriuria is associated with a 30% to 40% risk for becoming a symptomatic UTI, including pyelonephritis. Treatment of asymptomatic bacteriuria reduces these risks significantly.[120]

When bacteriuria or a symptomatic UTI occurs secondary to group B *Streptococcus* (GBS) infection, the diagnostic criterion for infection is 10,000 (10^4) colonies or more.

Women who have GBS in their urine are presumed to have heavily colonized vaginas. Because GBS is transient, treatment based on a urine culture is not necessarily needed, but these women are subsequently offered prophylactic antibiotics during labor and do not need the routine vaginal/rectal culture at 35 to 37 weeks' gestation.

All pregnant women should be screened for asymptomatic bacteriuria between 12 and 16 weeks' gestation or upon entry to care via a urine culture. Dipstick analysis and microscopy have poor predictive value. If screening for asymptomatic bacteriuria is negative, routine reculture is not indicated or cost-effective in pregnancy. Because there is a 40% false-positive rate with single cultures for asymptomatic bacteriuria, a second culture can be used for confirmation.[119]

The selection of antibiotic to treat asymptomatic bacteriuria is guided by resistance patterns, local costs, availability, and allergy history. Beta-lactams (e.g., ampicillin, penicillin, cephalosporins) are safe when used during pregnancy. Nitrofurantoin (Macrobid, Macrodantin), sulfonamides, and trimethoprim (Bactrim, Septra) may also be used, albeit with some caution.[121] Data regarding an association between these agents and congenital anomalies are conflicting. According to the American College of Obstetricians and Gynecologists, these antimicrobial agents are safe for use as first-line agents during the second trimester, and may be used in the first trimester if other antibiotic choices are not optimal.[121] Sulfonamides and nitrofurantoin are contraindicated for women with G6PD deficiency. Some authors recommend avoiding trimethoprim–sulfamethoxazole in the first and third trimesters because trimethoprim

is a folic acid antagonist and sulfonamides displace bilirubin from plasma-binding sites in the newborn, which theoretically increases the risk for newborn jaundice. Fluoroquinolones, tetracycline, and doxycycline are contraindicated in pregnancy.

In studies, treating asymptomatic bacteriuria for 7 days has proved superior for achieving bacteriologic cure over shorter treatment periods.[122] Commonly used antibiotic regimens for asymptomatic bacteriuria and other UTIs are presented in **Table 23-17**.

Table 23-17 **Commonly Used Antibiotic Regimens for Treatment of Asymptomatic Bacteriuria, Acute Cystitis, and Recurrent Urinary Tract Infections During Pregnancy**

Drug: Generic (Brand)	Dose	Clinical Considerations
Amoxicillin (Amoxil), Ampicillin (Principen)	500 mg orally 3 times/day or 875 mg orally 2 times/day for 3–7 days	Not considered a first-line agent. Beta-lactams may be less effective than other agents due to high rates of resistance. Adverse effects include allergies, candidal overgrowth, and pseudomembranous colitis.
Cephalexin (Keflex)	500 mg 4 times/day for 3–7 days	Not active against enterococci. Risks include allergies and hepatic dysfunction.
Fosfomycin (Monurol)	Single 3-gm oral dose	Lower efficacy than 3 days of other agents, but resistance is rare in the United States. This agent may be an option for women with multiple allergies. Does not reach a therapeutic dose in the kidney, so should not be used if pyelonephritis is suspected.
Nitrofurantoin (Macrobid)	100 mg orally twice daily for 3–7 days	First-choice for uncomplicated UTI. Resistance is unlikely, but bactericidal in urine only; poor penetration into tissues. Does not reach a therapeutic dose in the kidney, so should not be used if pyelonephritis is suspected. Administer with meals to improve absorption. Side effects include gastrointestinal upset, peripheral neuropathy, and pneumonitis.
Trimethoprim–sulfamethoxazole (Bactrim, Septra, Septra DS)	800 mg/160 mg (one double-strength tablet) orally twice daily for 3 days	First-choice agent if resistance in the local area is less than 20%. Avoid during first and third trimesters unless it is the only or best choice. Contraindicated for women with glucose-6-phosphate dehydrogenase deficiency. Allergic reactions are common; serious skin reactions and blood dyscrasias may occur.
Suppressive Therapy		
Nitrofurantoin (Macrobid)	50–100 mg orally once daily at bedtime	May be used daily or postcoital if UTI is sexually related.
Cephalexin (Keflex)	250–500 mg orally once daily at bedtime	May be used daily or postcoital if UTI is sexually related.

Abbreviation: UTI, urinary tract infection.

A test of cure culture is recommended 1-2 weeks after antibiotic treatment is complete, as a significant portion of women with asymptomatic bacteriuria do not clear the infection. If the woman experiences a second recurrence of either asymptomatic bacteriuria or develops a symptomatic UTI, continuous suppressive therapy should be initiated.[119]

Acute Cystitis

Acute cystitis is an infection in the bladder that develops in approximately 15% to 20% of pregnant women. Symptoms in pregnancy include urgency, frequency, dysuria, hematuria, and suprapubic pressure. Fever, chills, flank pain, and costovertebral angle tenderness suggest possible pyelonephritis. In addition to an increased risk for pyelonephritis, studies have found an increase in both preterm birth and low-birth-weight infants in women who develop acute cystitis during pregnancy. The diagnosis of a symptomatic UTI is based on urine culture results but women may be treated presumptively before culture results are available.[119] If the woman has persistent dysuria or dysuria without bacteriuria, testing for sexually transmitted infections is indicated.

Antibiotic regimens are based on culture results and microorganism sensitivities. Either a 3-day or 7-day course is recommended, depending on the severity of symptoms and individual clinical circumstances. A repeat urine culture for test of cure is recommended once the therapy is completed.

Recurrent Urinary Tract Infections

Recurrent UTI is distinguished from relapse or reinfection.

- A woman is considered to have *recurrent UTIs* if she has two or more infections within one year.
- A *relapse* (persistent infection) occurs when subsequent infections are found to be caused by the same organism as the original infection and occur within 2 weeks of the completion of treatment.
- *Reinfection* is defined as a UTI caused by a new organism more than 2 weeks after a negative test of cure.

Women who have recurrent UTIs are offered continuous daily suppressive therapy (**Table 23-17**).

Pyelonephritis

During pregnancy, women with bacteriuria have a 20- to 30-fold increase in their risk of developing pyelonephritis. This infection, which typically manifests in the second or third trimester, is often the consequence of undiagnosed or inadequate treatment of a UTI. Maternal age, nulliparity, sickle cell anemia, diabetes, nephrolithiasis, illicit drug use, and a history of pyelonephritis are all risk factors.

Signs and symptoms of pyelonephritis include low back pain, flank pain, fever, chills,

nausea, vomiting, costovertebral angle tenderness, and pyuria (white blood cells in the urine). Dysuria is less common. Pyelonephritis is a risk for sepsis, septic shock, adult respiratory distress syndrome, anemia, renal insufficiency, preterm birth, and low-birth-weight infant. The differential diagnoses for women with these symptoms are kidney stones, intra-amniotic infection, preterm labor, and placental abruption. Pyuria and bacteriuria will be present in women with pyelonephritis but do not occur in women with kidney stones, intra-amniotic infection, or abruption.

Inpatient management is usually recommended for women with pyelonephritis during pregnancy. Antibiotics are administered parenterally until the woman is afebrile for 24 to 48 hours. These agents are initiated empirically and changed as needed when the urine culture results are available. Once the acute symptoms have resolved, oral medications can be instituted and the woman is discharged from the hospital. Antibiotics are administered for 10 to 14 days and suppressive therapy is begun thereafter.

Outpatient management has been proposed for some women and may be appropriate when medical follow-up is available. Candidates for outpatient management include women less than 24 weeks' gestation who do not have severe symptoms and are healthy (i.e., no comorbid conditions).

Conclusion

Midwifery can be of great benefit for women whose pregnancy is medically complicated. Careful history taking, thorough physical examination, and an understanding of differential diagnoses and danger signs can help assure that women receive the correct diagnosis—the first step in mitigating adverse effects of these disorders, some of which can have lifelong effects. The decision to consult with, collaborate with, or refer to a specialist for ongoing care will be based on the severity of the woman's condition, practice policies, and the resources available. A multidisciplinary, team-based approach can provide the woman and her fetus with optimal care when she experiences complications during her pregnancy, birth, postpartum, and throughout their lives.

Resources

Organization	Description	Webpage
American Thyroid Association (ATA)	Pocket guidelines for thyroid disease during pregnancy.	https://www.thyroid.org
California Maternity Quality Care Collaborative (CMQCC)	Preeclampsia toolkit includes triggers, treatments, resources, and algorithms for caring for women with hypertension during pregnancy.	https://www.cmqcc.org/resources-tool-kits/toolkits/preeclampsia-toolkit
Epilepsy Foundation	A source of information for women and professionals about epilepsy.	http://www.epilepsy.com/
U.S. Food and Drug Administration (FDA)	List of Pregnancy Exposed Registry is a repository of registries for specific or groups of drugs used by women who are pregnant (e.g., women using antiepileptic agents).	https://www.fda.gov/ScienceResearch/SpecialTopics/WomensHealthResearch/ucm13
Society for Maternal-Fetal Medicine	Professional organization focused on medical conditions that occur during pregnancy and include	https://www.smfm.org/publications

	multiple guidelines and resources.	
Apps		
American College of Obstetricians and Gynecologists (ACOG)	Medically indicated late-preterm and early-preterm deliveries. This Committee Opinion and app summarizes ACOG guidelines and evidence for optimal time for induction of labor for women with medically complicated pregnancies.	https://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-o Obstetric-Practice/Medically-Indicated-Late-Preterm-and-Early-Term-Deliveries
Apps for diabetes monitoring	Multiple apps that have information about glucose in foods and track blood sugar values and are available for persons with diabetes.	See the healthcare provider for the recommended app.

References

1. Avery M, Montgomery O, Brandl-Salutz E. Essential components of successful collaborative maternity care models. The ACOG-ACNM project. *Obstet Gynecol Clin N Am.* 2012;39(3):423-434.
2. Smyth A, Oliveira GH, Lahr BD, Bailey KR, Norby SM, Garovic VD. A systematic review and meta-analysis of pregnancy outcomes in patients with systemic lupus erythematosus and lupus nephritis. *Clin J Am Soc Nephrol.* 2010;5(11):2060-2068.
3. Baer AN, Witter FR, Petri M. Lupus and pregnancy. *Obstet Gynecol Surv*. 2011;66(10):639-653.
4. American College of Obstetricians and Gynecologists. Practice Bulletin No. 180: gestational diabetes: *Obstet Gynecol*. 2017;130:e17-e21.
5. American Diabetes Association. Classification and diagnosis of diabetes. *Diab Care*. 2017;40(suppl 1):S11-S24.
6. Salmeen K. Gestational diabetes testing: making sense of the controversy. *J Midwifery Womens Health*. 2016;61(2):203-209.
7. Centers for Disease Control and Prevention. *National Diabetes Statistics Report, 2017*. Atlanta, GA: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 2017. https://www.cdc.gov/diabetes/pdfs/data/statistics/national-diabetes-statistics-report.pdf. Accessed October 25, 2017.
8. Fong A, Serra A, Herrero T, Pan D, Ogunyemi D. Pre-gestational versus gestational diabetes: a population based study on clinical and demographic differences. *J Diab Complications*. 2014;28:29-34.
9. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS; Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med*. 2005;352(24):2477-2486.
10. Kubo A, Ferrara A, Windham GC, et al. Maternal hyperglycemia during pregnancy predicts adiposity of the offspring. *Diab Care*. 2014;37(11):2996-3002.
11. Boney CM, Verma A, Tucker R, Vohr BR. Metabolic syndrome in childhood: association with birth weight, maternal obesity and gestational diabetes mellitus. *Pediatrics*. 2005;115(3):e290-e296.
12. Gillman MW, Rifas-Shiman S, Berkey CS, Field AE, Colditz GA. Maternal gestational diabetes, birth weight and adolescent obesity. *Pediatrics*. 2003;111(3):e221-e226.
13. Landon MB, Spong CY, Thom E, et al. A multicenter randomized trial of treatment for mild gestational diabetes. *N Engl J Med*. 2009;361(14):1339-1348.
14. Langer O, Conway DL, Berkus MD, Xenakis EM, Gonzales O. A comparison of glyburide and insulin in women with gestational diabetes. *N Engl J Med*. 2000;343(16):1134-1138.
15. Rowan JA, Hague WM, Gao W, Battin MR, Moore MP, MiG Trial Investigators. Metformin versus insulin for the treatment of gestational diabetes. *N Engl J Med*. 2008;358(19):2003-2015.
16. American Diabetes Association. Management of diabetes in pregnancy. *Diab Care*. 2017;40(suppl 1):S114-S119.
17. Fong A, Serra AE, Gabby L, Wing DA, Berkowitz KM. Use of hemoglobin A1c as an early predictor of gestational diabetes mellitus. *Am J Obstet Gynecol*. 2014;211(6):641.e1-641.e7.
18. Avery M. Diabetes in pregnancy: the midwifery role in management. *J Midwifery Womens Health*. 2000;45:472-480.
19. Longo S. Gastrointestinal conditions during pregnancy. *Clin Colon Rectal Surg*. 2010;23(02):80-89.
20. Angelini DJ. Obstetric triage revisited: update on non-obstetric surgical conditions in pregnancy. *J Midwifery Womens Health*. 2003;48(2):111-118.
21. Pinder M, Lummis K, Selinger CP. Managing inflammatory bowel disease in pregnancy: current perspectives. *Clin Exper Gastroenterol*. 2016;9:325-335.
22. Body C, Christie JA. Gastrointestinal diseases in pregnancy: nausea, vomiting, hyperemesis gravidarum, gastroesophageal reflux disease, constipation, and diarrhea. *Gastroenterol Clin North Am.* 2016;45(2):267-283.
23. Raga Ali RA, Egan LJ. Gastroesophageal reflux disease in pregnancy. *Best Pract Res Clin Gastroenterol.* 2007;21(5):793-806.
24. Pitchumoni CS, Yegneswaran B. Acute pancreatitis in pregnancy. *World J Gastroenterol*. 2009;15(45):5641-5646.
25. McLean E, Cogswell M, Egli I, Wojdyla D, de Benoist B. Worldwide prevalence of anaemia: WHO Vitamin and Mineral Nutrition Information System, 1993–2005. *Public Health Nutr*. 2009;12:444-454.
26. Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. *MMWR Recommendation Report*. 1998;47(RR-3):1-29.
27. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 95: anemia in pregnancy. *Obstet Gynecol*. 2008;112:201-207.
28. Committee on Nutritional Status During Pregnancy, Food and Nutrition Board, Institute of Medicine. *Nutrition During Pregnancy and Lactation: An Implementation Guide*. Washington, DC: National Academy Press; 1992.
29. Chao C, O'Brien KO. Pregnancy and iron homeostasis: an update. *Nutr Rev*. 2013;71(1):35-51.
30. Allen LH. Anemia and iron deficiency: effects on pregnancy outcome. *Am J Clin Nutr*. 2000;71(suppl):1280S-1284S.
31. Peña-Rosas JP, De-Regil LM, Dowswell T, Viteri FE. Daily oral iron supplementation during pregnancy. *Cochrane*

Database Syst Rev. 2012;12:CD004736. doi:10.1002/14651858.CD004736.pub4.
32. Mei Z, Cogswell ME, Looker AC, et al. Assessment of iron status in US pregnant women from the National Health and Nutrition Examination Survey (NHANES), 1999–2006. *Am J Clin Nutr*. 2011;93(6):1312-1320.
33. Milman N, Bergholt T, Byg KE, Erikson L, Graudal N. Iron status and iron balance during pregnancy: a critical appraisal of iron supplementation. *Acta Obstet Gynecol Scand*. 1999;78(9):749-757.
34. Haider BA, Olofin I, Wang M, Spiegelman D, Ezzati M, Fawzi WW; Nutrition Impact Model Study Group (Anaemia). Anaemia, prenatal iron use, and risk of adverse pregnancy outcomes: systematic review and meta-analysis. *BMJ*. 2013;346:f3443.
35. Khalafallah A, Dennis A, Bates J, et al. A prospective randomized, controlled trial of intravenous versus oral iron for moderate iron deficiency anaemia of pregnancy. *J Intern Med*. 2010;268:286.
36. Thom CS, Dickson CF, Gell DA, Weiss MJ. Hemoglobin variants: biochemical properties and clinical correlates. *Cold Spring Harbor Perspect Med*. 2013;3(3):a011858.
37. Piel FB, Weatherall DJ. The alpha-thalassemias. *N Engl J Med*. 2014;371:1908-1916.
38. Rund D, Rachmilewitz E. Beta-thalassemia. *N Engl J Med*. 2005;353:1135-1146.
39. Howard J, Oteng-Ntim E. The obstetric management of sickle cell disease. *Best Pract Res Clin Obstet Gynecol*. 2012;26:25-36.
40. Horowitz KM, Ingardia CJ, Borgida AF. Anemia in pregnancy. *Clin Lab Med*. 2013;33(2):281-291.
41. Ciobanu AM, Colibab S, Cimpoca B, Peltecu G, Panaitescu AM. Thrombocytopenia in pregnancy. *Maedica (Buchar)*. 2016;11(1):55-60.
42. Boothman L. Platelets in pregnancy: their role and function in disease. *Br J Midwifery*. 2016;24(8):550-555.
43. Marik PE, Plante LA. Venous thromboembolic disease and pregnancy. *N Engl J Med*. 2008;359:2025.
44. Simcox LE, Ormesher L, Tower C, Greer IA. Pulmonary thrombo-embolism in pregnancy: diagnosis and management. *Breathe (Sheff)*. 2015;11(4):282-289.
45. Greer IA. The challenge of thrombophilia in maternal–fetal medicine. *N Engl J Med*. 2000;342:424.
46. Dobbenga-Rhodes Y. Shedding light on inherited thrombophilias: the impact on pregnancy. *J Perinat Neonatal Nurs*. 2016;30(1):36-44.
47. Connors JM. Thrombophilia testing and venous thrombosis. *N Engl J Med*. 2017;377:1177-1187.
48. American College of Obstetricians and Gynecologists, Women's Health Care Physicians. ACOG Practice Bulletin No. 138: inherited thrombophilias in pregnancy. *Obstet Gynecol*. 2013;122(3):706-717.
49. American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy: report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122(5):1122-1131.
50. Duley L. The global impact of pre-eclampsia and eclampsia. *Semin Perinatol*. 2008;33(3):130-137.
51. Shih T, Peneva D, Xu X, et al. The rising burden of preeclampsia in the United States impacts both maternal and child health. *Am J Perinatol*. 2016;33:329-338.
52. Lo J, Mission JF, Caughey AB. Hypertensive disease of pregnancy and maternal mortality. *Curr Opin Obstet Gynecol*. 2013;25(2):124-132.
53. Say L, Chou D, Gemmill A, et al. Global causes of maternal death: a WHO systematic analysis. *Lancet Glob Health*. 2014;2(6):e323-e333.
54. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-based guidelines for the prevention of cardiovascular disease in women—2011 update: a guideline from the American Heart Association. *J Am Coll Cardiol*. 2011;57(12):1404-1423.
55. Barton JR, O'Brien JM, Bergauer NK, Jacques DL, Sibai BM. Mild gestational hypertension remote from term: progression and outcome. *Am J Obstet Gynecol*. 2001;184:979-983.
56. Roberts JM, Hubel CA. The two-stage model of preeclampsia: variations on the theme. *Placenta*. 2009;30(suppl A):S32-S37.
57. Fisher SJ. Why is placentation abnormal in preeclampsia? *Am J Obstet Gynecol*. 2015;213:S115-S122.
58. Burton GJ, Yung HW, Cindrova-Davies T, Charnock-Jones DS. Placental endoplasmic reticulum stress and oxidative stress in the pathophysiology of unexplained intrauterine growth restriction and early onset preeclampsia. *Placenta*. 2009;30(suppl A):S43-S48.
59. Chen CW, Jaffe IZ, Karumanchi SA. Pre-eclampsia and cardiovascular disease. *Cardiovasc Res*. 2014;101(4):579-586.
60. Snydal S. Major changes in diagnosis and management of preeclampsia. *J Midwifery Womens Health*. 2014;59(6):596-605.
61. Warrington JP, George EM, Palei AC, Spradley FT, Granger JP. Recent advances in the understanding of the pathophysiology of preeclampsia. *Hypertension*. 2013;62(4):666-673.
62. Leslie MS, Briggs LA. Preeclampsia and the risk of future vascular disease and mortality: a review. *J Midwifery Womens Health*. 2016;61(3):315-324.
63. Tooher J, Thornoton C, Makris A, Ogle R, Korda A, Hennessy A. All hypertensive disorders of pregnancy increase the risk of future cardiovascular disease. *Hypertension*. 2017;70:798-803.

64. U.S. Preventive Services Task Force. *Final Recommendation Statement: Low-Dose Aspirin Use for the Prevention of Morbidity and Mortality from Preeclampsia: Preventive Medication*. December 2016. Available at: https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/low-dose-aspirin-use-for-the-prevention-of-morbidity-and-mortality-from-preeclampsia-preventive-medication. Accessed December 6, 2017.
65. Tranquilli AL. Early and late-onset preeclampsia. *Pregn Hypertension*. 2014;4(3):241.
66. Bernstein PS, Martin JN Jr, Barton JR, et al. National Partnership for Maternal Safety: consensus bundle on severe hypertension during pregnancy and the postpartum period. *Obstet Gynecol*. 2017;130(2):347-357.
67. Cipolla MJ, Kraig RP. Seizures in women with preeclampsia: mechanisms and management. *Fetal Matern Med Rev*. 2011;22(2):91-108.
68. Sibai BM. Chronic hypertension in pregnancy. *Obstet Gynecol*. 2002;100:369-377.
69. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 125: chronic hypertension in pregnancy. *Obstet Gynecol*. 2012;119:396-407.
70. Bullo M, Tschumi S, Bucher BS, Bianchetti MG, Simonetti GD. Pregnancy outcome following exposure to angiotensin-converting enzyme inhibitors or angiotensin receptor antagonists: a systematic review. *Hypertension*. 2012;60:444-450.
71. Celis H, Fagard RH. White coat hypertension: a clinical review. *Eur J Intern Med*. 2004;15(6):348-357.
72. Aloizos S, Seretis C, Liakos N, et al. HELLP syndrome: understanding and management of a pregnancy-specific disease. *J Obstet Gynaecol*. 2013;33(4):331-337.
73. Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. *Obstet Gynecol*. 2004;103:981.
74. Carlson A, Norwitz ER, Stiller RJ. Cytomegalovirus infection in pregnancy: should all women be screened? *Rev Obstet Gynecol*. 2010;3(4):172-179.
75. American College of Obstetricians and Gynecologists. Practice Bulletin No. 151: cytomegalovirus, parvovirus B19, varicella zoster, and toxoplasmosis in pregnancy. *Obstet Gynecol*. 2015;125:1510-1525.
76. Feldman DM, Keller R, Borgida AF. Toxoplasmosis, parvovirus, and cytomegalovirus in pregnancy. *Clin Lab Med*. 2016;36(2):407-419.
77. American College of Obstetricians and Gynecologists. Committee Opinion No. 614: management of pregnant women with presumptive exposure to listeria monocytogenes. *Obstet Gynecol*. 2014;124(6):1241-1244.
78. Silk BJ, Date KA, Jackson KA, et al. Invasive listeriosis in the Foodborne Diseases Active Surveillance Network (FoodNet), 2004–2009: further targeted prevention needed for higher-risk groups. *Clin Infect Dis*. 2012;54(suppl 5):S396-S404.
79. Jackson KA, Iwamoto M, Swerdlow D. Pregnancy-associated listeriosis. *Epidemiol Infect*. 2010;138:1503-1508.
80. Goff M. Parvovirus B19 in pregnancy. *J Midwifery Womens Health*. 2005;50:536-538.
81. Bouthry E, Picone O, Hamdi G, Grangeot-Keros L, Ayoubi JM, Vauloup-Fellous C. Rubella and pregnancy: diagnosis, management and outcomes. *Prenat Diagn*. 2014;34(13):1246-1253.
82. Centers for Disease Control and Prevention. General recommendations on immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR*. 2011;60(2);1-60.
83. Lamont RF, Sobel JD, Carrington D, et al. Varicella-zoster virus (chickenpox) infection in pregnancy. *BJOG*. 2011;118(10):1155-1162.
84. Pressman EK, Seidman SM, Summers L. Primary care for women: comprehensive assessment of the neurologic system. *J Midwifery Womens Health*. 1995;40(2):163-168.
85. Deneris A, Rosati Allen P, Hart Hayes E, Latendresse G. Migraines in women: current evidence for management of episodic and chronic migraines. *J Midwifery Womens Health*. 2017;62(3):270-285.
86. Moloney MF, Johnson CJ. Migraine headaches: diagnosis and management. J Midwifery Womens Health. 2011;56(3):282-292.
87. Facchinetti F, Allais G, Nappi RE, et al. Migraine is a risk factor for hypertensive disorders in pregnancy: a prospective cohort study. Cephalalgia. 2009;29:286-292.
88. Marchenko A, Etwel F, Olutunfese O, Nickel C, Koren G, Nulman I. Pregnancy outcome following prenatal exposure to triptan medications: a meta-analysis. Headache 2015;55:490.
89. Klein A. Peripheral nerve disease in pregnancy. Clin Obstet Gynecol. 2013;56(2):382-388.
90. Massey EW, Guidon AC. Peripheral neuropathies in pregnancy. Continuum. 2014;20(1):100-114.
91. Liu G, Slater N, Perkins A. Epilepsy: treatment options. Am Fam Physician. 2017;96(2):87-96.
92. Chen D, Hou L, Duan X, Peng H, Peng B. Effect of epilepsy in pregnancy on fetal growth restriction: a systematic review and meta-analysis. Arch Gynecol Obstet. 2017;296(3):421-427.
93. Borgelt L, Hart F, Bainbridge J. Epilepsy during pregnancy: focus on management strategies. Int J Womens Health. 2016;8:505-517.
94. Veroniki AA, Rios P, Cogo E, et al. Comparative safety of antiepileptic drugs for neurological development in children exposed during pregnancy and breast feeding: a systematic review and network meta-analysis. BMJ Open. 2017;7(7):e017248. doi:10.1136/bmjopen-2017-017248.

95. Viale L, Allotey J, Cheong-See F, et al. Epilepsy in pregnancy and reproductive outcomes: a systematic review and meta-analysis. Lancet. 2015;386(10006):1845-1852.
96. Murphy VE, Gibson P, Talbot PI, Clifton VL. Severe asthma exacerbations during pregnancy. Obstet Gynecol. 2005;106:1046.
97. Murphy VE. Managing asthma in pregnancy. Breathe. 2015;11(4):258-267.
98. National Asthma Education and Prevention Program. Working group report on managing asthma during pregnancy: recommendations for pharmacologic treatment. Available at: https://www.nhlbi.nih.gov/files/docs/astpreg_qr.pdf. Accessed November 4, 2017.
99. Bates M, Ahmed Y, Kapata N, Maeurer M, Mwaba P, Zumla A. Perspectives on tuberculosis in pregnancy. *Int J Infect. Dis*. 2015;32:124-127.
100. Ross L, Goff M. Latent tuberculosis infection and BCG vaccination. *J Midwifery Womens Health*. 2005;50(4):344-347.
101. Sobhy S, Babiker Z, Zamora J, Khan KS, Kunst H. Maternal and perinatal mortality and morbidity associated with tuberculosis during pregnancy and the postpartum period: a systematic review and meta-analysis. *BJOG*. 2017;124(5):727-733.
102. Lewinsohn DM, Leonard MK, LoBue PA, et al. Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention clinical practice guidelines: diagnosis of tuberculosis in adults and children. *Clin Infect Dis*. 2017;64(2):111-115.
103. American Thoracic Society. Targeted tuberculin testing and treatment of latent tuberculosis infection. *MMWR Recomm Rep*. 2000;49:1.
104. Mathad KH, Gupta A. Tuberculosis in pregnant and postpartum women: epidemiology, management, and research gaps. *Clin Infect Dis*. 2012;55(11):1532-1549.
105. Horsburgh CR, Barry CE, Lange C. Treatment of tuberculosis. *N Engl J Med*. 2015;373;2149-2160.
106. Getahun H, Matteeli A, Chaisson RE, Raviglione M. Latent *Mycobacterium tuberculosis* infection. *N Engl J Med*. 2015;372:2127-2135.
107. Committee on Obstetric Practice and Immunization Expert Work Group, Centers for Disease Control and Prevention's Advisory Committee on Immunization, United States, American College of Obstetricians and Gynecologists. Committee Opinion No. 608: Influenza vaccination during pregnancy. *Obstet Gynecol*. 2014;124(3):648-651.
108. Luteijn JM, Brown MJ, Dolk H. Influenza and congenital anomalies: a systematic review and meta-analysis. *Hum Reprod*. 2014;29:809-823.
109. Greer LG, Sheffield JS, Rogers VL, Roberts SW, McIntire DD, Wendel GD Jr. Maternal and neonatal outcomes after antepartum treatment of influenza with antiviral medications. *Obstet Gynecol*. 2010;115:711-716.
110. Stagnaro-Green A. Overt hyperthyroidism and hypothyroidism during pregnancy. *Clin Obstet Gynecol*. 2011;54(3):478-487.
111. Korevaar TIM, Medici M, Visser TJ, Peeters RP. Thyroid disease in pregnancy: new insights in diagnosis and clinical management. *Nat Rev Endocrinol*. 2017;13(10):610-622.
112. Alexander EK, Pearce EN, Brent GA, et al. 2017 guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2017;27(3):315-389.
113. Springer D, Jiskra J, Limanova Z, Zima T, Potlukova E. Thyroid screening in pregnancy: from physiology to screening. *Crit Rev Clin Lab Sci*. 2017;54(2):112-116.
114. Cooper DS, Laurberg P. Hyperthyroidism in pregnancy. *Lancet Diab Endocrinol*. 2013;1:238-249.
115. American College of Obstetricians and Gynecologists. Practice Bulletin No. 148: thyroid disease in pregnancy. *Obstet Gynecol*. 2015;125(4):996-1005.
116. Lazarus JH, Bestwick JP, Channon S, et al. Antenatal thyroid screening and childhood cognitive function. *N Engl J Med*. 2012;366:493-501.
117. De Groot L, Abalovich M, Alexander EK, et al. Management of thyroid dysfunction during pregnancy and postpartum: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2012;97(8):2543-2565.
118. Matuszkiewicz-Rowinska J, Malyszko J, Wieliczko M. Urinary tract infections in pregnancy: old and new unresolved diagnostic and therapeutic problems. *Arch Med Sci*. 2015;11(1):67-77.
119. O'Dell KK. Pharmacologic management of asymptomatic bacteriuria and urinary tract infections in women. *J Midwifery Womens Health*. 2011;56(3):248-265.
120. Smaill FM, Vazquez JC. Antibiotics for asymptomatic bacteriuria in pregnancy. *Cochrane Database Syst Rev*. 2015;8:CD000490. doi:10.1002/14651858.CD000490.pub3.
121. American College of Obstetricians and Gynecologists. Committee Opinion No. 717: sulfonamides, nitrofurantoin, and risk of birth defects. *Obstet Gynecol*. 2017;130:e150-e152.
122. Widmer M, Lopez I, Gulmezoglu AM, Mignini L, Roganti A. Duration of treatment for asymptomatic bacteriuria during pregnancy. *Cochrane Database Syst Rev*. 2015;11:CD000491. doi:10.1002/14651858.CD000491.pub3.

PART V

Intrapartum

NANCY K. LOWE

"Not just another day in a woman's life." This conclusion by Penny Simkin,[1,2] childbirth educator and doula extraordinaire, should play itself over and over in the midwife's mind when with or thinking about a woman in labor. They are simple words, yet profound in their description of the importance and permanence of a woman's memory of the day she gives birth. As Simkin's research showed, women remember with exquisite detail and accuracy the words and actions during labor of their providers and partners 15 to 20 years later. What the midwife does and says during labor may affect not only the physiology of the labor process, but also the woman's memory of the important day when she gave birth and welcomed a new child into her life and family. The chapters in this section present the components of midwifery care of women during the labor process.

The midwife is the guide, consultant, and collaborator for a woman and her family during labor and birth. The midwife helps to ensure a safe physical, psychological, and spiritual place for labor and birth, while functioning in the milieu of the specific birth setting, whether in the home, birth center, or hospital. The midwife serves as the buffer between the laboring woman and whatever system of care surrounds her. It is the midwife's responsibility to ensure a protected space where labor and birth can evolve as the unique process that it is for each woman and each infant she carries. The philosophy of midwifery care includes "watchful waiting and non-intervention into normal processes."[3]

There are few other times during the reproductive cycle when inappropriate or ill-timed interventions can have the dire consequences that are possible during labor and birth. The ill-timed amniotomy with a high fetal head can turn an otherwise normal labor into an emergency when a loop of umbilical cord drops into the midwife's hand and the fetal head descends against it. Tachysystole and fetal bradycardia from injudicious exogenous oxytocin administration can trigger a cascade of additional interventions that create anxiety and further disrupt and perhaps even stop the normalcy of labor. While the availability of medical and

surgical interventions during labor can help assure an appropriate safety net for problems that may occur, their injudicious use not only produces more harms than benefits for mother and infant, but also unnecessarily increases the cost of health care.

The midwife is the vigilant protector who ensures safety for a woman and her infant during labor and birth. This vigilance requires an exquisite understanding of the physiology and psychology of human parturition, as well as the scientific basis for midwifery practice during this vulnerable period of transition. The expert midwife is a lifelong student of labor, its many aberrations, and the scientific literature that helps deepen our understanding and expand our expertise.

Although labor is a normal physiologic and developmental process, it is also a period of vulnerability for both mother and infant, during which problems can arise requiring the midwife's expert action through timely identification based on comprehensive assessment, treatment, and collaboration with and potential referral to other members of the healthcare team. Regardless of the problem or the treatment regimen required, the laboring woman and respect for her autonomy and self-determination remain the center of the midwife's care.

An adaptation of a writing by Lao Tzu on leadership and being a midwife (Do you think of your role as a midwife as a role of leadership?) summarizes the midwife's role during the intrapartum period well:

> *Imagine that you are a midwife; you are assisting at someone else's birth. Do good without show or fuss. Facilitate what is happening rather than what you think ought to be happening. If you must take the lead, lead so that the mother is helped, yet still free and in charge. When the baby is born, the mother will rightly say: "We did it ourselves!"*[4(p33)]

References

1. Simkin P. Just another day in a woman's life? Women's long-term perceptions of their first birth experience. Part I. *Birth*. 1991:18(4);203-210.
2. Simkin P. Just another day in a woman's life? Women's long-term perceptions of their first birth experience. Part II. *Birth*. 1992;19(2):64-81.
3. American College of Nurse-Midwives. *Philosophy of the American College of Nurse-Midwives*. Silver Spring, MD: American College of Nurse-Midwives; 2004.
4. Heider J. *The Tao of Leadership: Lao Tzu's Tao Te Ching Adapted for a New Age*. Atlanta, GA: Humanics New Age; 1985.

CHAPTER

24

Anatomy and Physiology During Labor and Birth

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Introduction

Providing care that supports the normal physiologic process of labor and birth requires an understanding of the physiology of this unique period. The anatomy and physiology of the female reproductive system and changes during pregnancy are described in the *Anatomy and Physiology of the Female Reproductive System* and *Anatomy and Physiology of Pregnancy* chapters, respectively. This chapter focuses on the anatomic and physiologic changes that occur during labor and birth in both the woman and the fetus. The uterine changes that proceed from contraction inhibition during pregnancy to uterotropin-associated activation and uterotonin-associated stimulation during latent and active labor are highlighted. *Uterotropins* such as estrogen and progesterone are hormones that cause an effect on the uterus and *uterotonins* such as prostaglandins and oxytocin induce uterine contractions. A review of the anatomic relationships between the fetus and the maternal pelvis during the process of birth follows.

Traditionally, the processes involved in labor and birth have been conceptualized as those that affect the power (uterus), the passenger (fetus), and the passage (pelvis). A fourth "P" is often included in this rubric—the psyche—and will be addressed in this chapter. However, many more complex adaptations also occur during labor and birth. It is important to appreciate that labor and birth involve normal physiologic processes best supported through limited intervention.[1] During labor and birth, interventions have the potential to disrupt complex hormonal regulation, thereby prompting a cascade of additional interventions with potential for further disruption of physiologic processes.[2] Interventions during labor such as administration of synthetic oxytocin, prelabor rupture of membranes, and continuous electronic fetal heart-rate monitoring may have unintended risks and interfere with physiologic processes in low-risk women in spontaneous labor.[2,3] With this knowledge, midwives are prepared both to appreciate and support the normal physiologic processes of labor and birth, and to recognize situations when labor does not progress as anticipated, requiring alternative management strategies.

Premonitory Signs and Symptoms of Labor

At term, the transition leading to labor results from the combined physiologic and hormonal responses of mother and fetus.[2] Premonitory signs and symptoms of impending labor may occur 1 or 2 weeks before active labor starts, as the uterus slowly shifts from a quiescent state to active ongoing contractions.

Maternal Adaptations to Fetal Descent

Lightening—that is, the descent of the fetus into the true pelvis—may occur as early as 4 weeks prior to the onset of labor. The movement of the fetus into a lower position in the true pelvis, referred to as *engagement*, is more common among nulliparous women than multiparous women. The anatomic change in fetal position, which may be measured objectively by a decrease in fundal height, is accompanied by characteristic signs and symptoms in the mother, including partial relief of pressure on the diaphragm, leading to greater ease of breathing and decreased reflux. In turn, pressure on structures adjacent to the pelvis increases, with symptoms such as urinary frequency, pelvic pressure, leg cramps, and dependent edema in the lower extremities becoming more evident. Partial obstruction by the fetal presenting part on the femoral veins reduces venous return to the maternal heart, particularly when the woman is standing. As blood pools in her lower leg veins, increased intravascular pressure promotes fluid movement out of the vessels and into the surrounding interstitial tissue, resulting in edema. When the woman is supine, venous return is enhanced and intravascular pressure is reduced, resulting in improvement of lower extremity edema. Tilting a woman to her left side further improves venous return by reducing pressure on the inferior vena cava, which lies slightly to the right of center. The reduced capacity of the maternal bladder due to anatomic pressure of the engaged fetal presenting part, coupled with increased venous return when recumbent, contributes to increased frequency of urination, which can lead to interrupted sleep in the last weeks of pregnancy.

Cervical Ripening, Effacement, and Dilation

Our understanding of cervical changes during pregnancy, labor, and birth is largely the result of animal studies. Thus, knowledge of human physiology has not yet been fully elucidated with regard to timing and molecular processes. It is known that the cervix undergoes four phases of remodeling over the course of pregnancy, labor, and birth, as described in the *Anatomy and Physiology of Pregnancy* chapter.

Cervical connective tissue changes are central to the significant remodeling that characterizes the third stage of cervical ripening that occurs prior to and during labor. The rearrangement of collagen is the primary change that causes cervical ripening, with realignment of elastin and smooth muscle fibers playing a more minor role in the process.[4,5] The anatomic changes modulated by inflammatory and hormonal influences are accompanied by an increase in water content that leads to softening of the cervix,[6] setting the stage for the full effacement and dilation that precede birth during active labor.

The triggers that promote these cervical changes include increases in inflammation, oxytocin, and prostaglandin activity.[2] The ripening and softening of the cervix, which are essential for effacement and dilation, reflect the enhanced collagen breakdown and elastin remodeling mediated by matrix metalloproteinases (enzymes that break down peptide bonds) that were previously inhibited by progesterone during uterine quiescence.[7] The tight collagen bundles in the nonpregnant cervix respond to hormonal changes by becoming less dense and more loosely packed at midpregnancy. In later pregnancy and during labor, an increase in water content creates a softer consistency, which is also associated with separation of collagen fibrils promoted by the proteoglycan decorin and a reduction in the fibronectin level.[4] Increased levels of enzymes that break down connective tissue, including collagenase and elastase, contribute to the process of cervical remodeling.

Effacement is the result of the lengthening of the muscle fibers at the internal os, which stretch the endocervix upward into the lower uterine segment. This change is evaluated clinically in terms of actual centimeters of length or percentage of the original length, where no effacement is described as 0% and complete effacement is described as 100%. The trend is toward documenting the cervix in actual centimeters of length. However, when a woman is at term and her cervix is being assessed to determine how likely she is to have a vaginal birth, cervical length may be reported as a percentage of the original length noted before the process of effacement begins. Effacement as a percentage remains one of the parameters in determining the Bishop's score, a measure that is discussed in more detail in the *Complications During Labor and Birth* chapter.

Some cervical dilation may occur prior to labor, with effacement and dilation continuing over the course of labor. Nulliparous women may experience some cervical softening and effacement prior to labor, or they may enter labor with the cervix not yet soft or effaced. In nulliparous women, effacement typically precedes dilation. In contrast, parous women often enter labor with some effacement and dilation having already taken place, and these processes continue to occur simultaneously as labor progresses.

Dilation is the widening of the external os, with this structure expanding from an opening of a few millimeters to one that allows the passage of the fetus during birth. The force of contractions, coupled with the hydrostatic action of the amniotic fluid, creates a force that promotes dilation of the low-resistance cervix. In the case of ruptured membranes, the pressure of the presenting fetal part on the cervix also promotes progressive dilation. Dilation is clinically evaluated by measuring the diameter of the cervical opening digitally in centimeters, with 0 centimeters describing a closed external cervical os and 10 centimeters defining complete dilation. The 10-centimeter measure of complete dilation is based on the suboccipitobregmatic diameter of the fetal head, which is approximately 9.5 centimeters at term; it is the widest anterior–posterior diameter of the flexed head during the normal mechanisms of labor when the fetus is in a cephalic presentation. Fetal head diameters are reviewed in detail later in this chapter.

In addition to placing traction on the cervix to promote ripening, uterine contractions may increase in frequency and lead to discomfort before the onset of labor. Historically referred to as "false labor," these early Braxton Hicks–like contractions do not occur in a regular pattern, nor do they cause the progressive cervical dilation and effacement seen during active labor.

They are not “false,” however, because premonitory uterine contractions probably reflect changes within the uterine musculature that occur with advancing gestation and preparation for labor. The hormonal milieu that stimulates cervical ripening, combined with anatomic pressure of the presenting part on the cervix and the traction generated by uterine contractions, also causes the premonitory sign of the passing of the cervical mucus plug. This blood-tinged mucus is expelled from the cervical canal after the cervical changes associated with labor have started. The passage of the mucus plug may occur all at once or, more commonly, over a period of 1 to 2 days. In the absence of trauma or injury to the cervix (e.g., during aggressive vaginal examination), the passing of the mucus plug often indicates impending labor within 48 hours.

Factors That Influence the Onset of Labor

The onset of labor is classically defined as the occurrence of regular painful contractions that promote dilation of the cervix. Contractions that occur at regular intervals with increasing frequency, duration, and intensity are the hallmark of labor.[8,9] The primary basis of our understanding of the process of labor onset is evidence from animal studies, which have been extrapolated to explain the human condition. The onset of labor is likely the result of a complex interplay between biochemical and mechanical influences originating in both the maternal and fetal systems and the complex interactions of hormonal systems regulating one another's activity.[2] In essence, the wide individual variations in the chronology of events associated with labor onset reflect the variability of the human experience.

The influence of genetic (maternal and fetal) and race/ethnicity factors in the timing of labor onset provides some evidence for genetic influences of labor onset.[10-12] Gene–environment interactions that occur as a result of maternal, fetal, and intrauterine factors illustrate the complexity of outcomes, including labor onset.[13,14] Genetic polymorphisms in the maternal genome suggest many factors may influence the duration of pregnancy and timing of onset of labor, including stress[15] and inflammation.[16-18]

Endocrinology of Labor: Parturition Cascade

The onset of labor is likely the result of interactions among the maternal, fetal, and placental units.[19] This process involves a cascade of events that include multiple redundant loops—perhaps the reason why researchers have failed to find a single initiating factor for labor. Two primary mechanisms that are known "players" are a change from progesterone dominance and uterine quiescence to estrogen-stimulated uterotropin activation and placental production of corticotropin-releasing hormone. The overall process of parturition proceeds through four phases (**Figure 24-1**):[9,20,21]

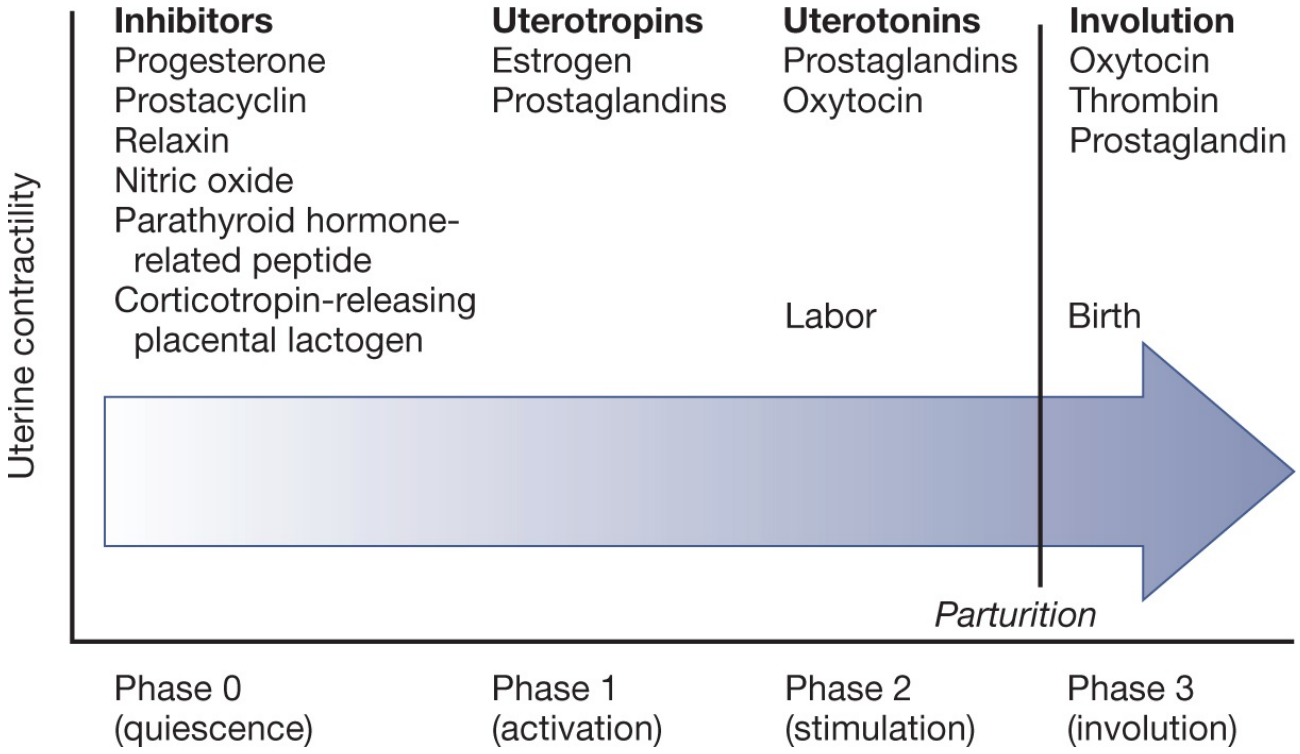


Figure 24-1 Phases of parturition.

Phase 0, Quiescence: Period in late pregnancy of uterine quiescence. Inhibitors of uterine contractions include progesterone, prostacyclin, relaxin, nitric oxide, and other hormones.

Phase 1, Activation: Uterotropins (e.g., estrogen) stimulate upregulation of myometrial receptors for oxytocin and prostaglandins, and activation of gap junctions between myometrial cells.

Phase 2, Stimulation: Uterotonins (e.g., oxytocin and prostaglandins) promote labor progression.

Phase 3, Involution: Uterine involution after birth is mediated by oxytocin and possibly thrombin.

Phase 1: Activation

Increases in myometrial excitability, spontaneous activity, and responsiveness to substances

that stimulate uterine contraction are the primary changes that the uterus undergoes in the transition from quiescence to activation.[22] The activation phase of parturition is characterized by loss of progesterone dominance. The uterus has two forms of progesterone receptors, which have opposing functions. Progesterone primarily binds to the progesterone receptor-B (PR-B) during pregnancy; this binding suppresses inflammation and induces uterine quiescence. During activation, the shortened progesterone receptor-A (PR-A) preferentially binds to progesterone, promoting the dominant pro-inflammatory effects associated with labor.[23] The increased concentration of PR-A relative to PR-B causes a functional progesterone withdrawal without altering the circulating progesterone concentrations.[24-27] Similarly, progesterone stimulates nitric oxide synthesis, which inhibits smooth muscle contraction. When these effects of progesterone, prostacyclin, relaxin, and nitric oxide—which are present during the quiescent period cease, increased uterine activity ensues.

Under the influence of estrogen, uterine myometrial cells express receptors for prostaglandins and oxytocin and develop gap junctions that allow direct communication between muscle fibers. *Gap junctions* are transmembrane proteins that create a pore—that is, a line of communication—between two adjacent myocytes. The action potentials that initiate contraction activity propagate through gap junctions, resulting in synchronous uterine contractions characterized by generation of force along a short distance at low velocity.

Prostaglandins facilitate uterine contractions, increase myometrial sensitivity to oxytocin, and stimulate formation of gap junctions. In labor, PGE_2 favors the prostaglandin EP3 receptor over the prostaglandin EP1 receptor, thereby stimulating uterine contractility through binding to the EP3 receptor.[28]

Role of the Placenta in Labor Initiation

The declining influence of progesterone and nitric oxide also contributes to increased production of corticotropin-releasing hormone (CRH) by the placenta.[23,29] Placenta-derived CRH levels peak at the time of birth, which suggests the placenta has an important role in the timing of labor onset and birth. Placental CRH is transported to the fetus, where it stimulates the fetal hypothalamic–pituitary–adrenal axis, promoting cortisol production by the fetal adrenal gland. Cortisol-induced maturation of fetal lungs triggers increased pro-inflammatory surfactant and phospholipid production, thereby serving as a fetal mechanism for inducing maternal uterine contractions mediated in part by stimulation of prostaglandin synthesis in the intrauterine tissues.[19] CRH also stimulates fetal adrenal production of dehydroepiandrosterone sulfate (DHEA-S), which serves as a substrate for estrogen production, enhancing the fetal influence on the onset and progression of labor. Fetal membranes, which represent the maternal–fetal interface, serve as a vehicle for communication of these hormones between the fetal and maternal systems to stimulate labor.[30,31]

CRH influences the transition from progesterone to estrogen dominance in labor through stimulation of fetal adrenal activity, which increases the fetal production of DHEA-S. The placenta, in turn, converts DHEA-S to estrogens that oppose the function of progesterone. Contractile activity is also modulated by CRH through the promotion of fetal membrane production of the stimulatory prostaglandins $F_2\alpha$ and E_2 and through uterine smooth muscle cell

signaling that increases calcium availability.[32]

Role of Inflammation in Labor Initiation

The activation and stimulation stages of labor are characterized by a heightened state of inflammation. At the onset of labor, leukocytes invade the myometrium, cervix, chorio-decidua, and amnion.[33] Pro inflammatory cytokines released by activated leukocytes promote calcium entry into uterine smooth muscle cells and increased prostaglandin production, enhancing uterine contractions. Neutrophils release enzymes that further degrade extracellular matrix proteins, including fetal fibronectin and collagen, to allow for progressive dilation. Inflammatory cytokines and degrading matrix metalloproteinase enzymes are likewise increased in the fetal membranes, contributing to their weakening and eventual rupture.

Phase 2: Stimulation of Uterine Myometrial Activity

The onset of regular, progressive uterine contractions characterizes the stimulation phase of parturition. The influence of oxytocin becomes especially pronounced during the stimulation phase of labor. Produced in the hypothalamus and secreted via the posterior pituitary, oxytocin is released into the maternal circulation in a pulsatile fashion, reaching peak levels during fetal expulsion.[34] Estrogen dominance also initiates expression of oxytocin genes in the chorio-decidua, providing a local source of oxytocin. Binding of oxytocin to myometrial oxytocin receptors stimulates the smooth muscle contractions that characterize active labor through a calcium-dependent pathway. Prostaglandin production in the maternal decidua is also stimulated by oxytocin-receptor binding; thus, the relationship between prostaglandins and oxytocin is bidirectional.[35,36]

Changes in oxytocin receptor number and sensitivity—rather than the production and release of oxytocin itself—serve as a primary influence on the strength and frequency of uterine contractions during active labor.[29] This relationship has important clinical implications. First, the affinity of oxytocin for its receptor in the uterine fundus increases,[37] maximizing the effect of this hormone even without an increase in the oxytocin plasma concentration. Second, the number of oxytocin receptors present on the myometrial cell membranes peaks in early labor, reflecting an increase of up to 200-fold from the number of such receptors present on myometrial cells in the nonpregnant state. The plasma level of oxytocin does not change from prelabor levels during the first stage of labor, but does increase and peak in the second stage of labor, possibly enhanced by fetal oxytocin synthesis, release, and transport to the maternal side of the placenta.[38] Labor and mechanical stretch also increase expression of oxytocin receptors in the uterine myocytes, further enhancing the potential for oxytocin-receptor binding.[36,39]

Similar to the case for other related G-protein–coupled receptors, desensitization of the oxytocin receptor can be induced by exposure of the receptor to increased concentrations of oxytocin in the maternal circulation. This can occur as the result of increasing plasma levels of oxytocin or prolonged exposure to oxytocin administered for induction or augmentation of labor.[40] Prolonged or repeated stimulation of the oxytocin receptors then contribute to downregulation, which reduces the number of receptors available for oxytocin binding. Uterine

contractions become less forceful and less frequent as a result of attenuation of cell signaling and reduced calcium release, potentially leading to impaired labor progress or uterine atony after birth.

Along with oxytocin, the influence of prostaglandins on the myometrium predominates during the stimulation phase. Produced by the amnion, chorion, and decidua, prostaglandins work in a paracrine fashion to promote uterine contractile activity. Prostaglandin production is also stimulated by inflammatory cytokines. Recent evidence suggests additional effects of these changes may include decreased collagen content and area covered by connective tissue, providing a potential explanation for the higher vaginal delivery rates, tachysystole, and uterine rupture noted with administration of PGE_1 in labor.[41]

Mechanism of Uterine Contraction

Contraction of uterine smooth muscle is a phenomenon unique to labor. The uterine myocyte is an excitable smooth muscle cell type that has contractile ability. Triggers for uterine myocyte contraction include mechanical stretch (myogenic) and hormone-receptor binding. Prostaglandins produced by activated myocytes work in a paracrine fashion, stimulating nearby myocytes to depolarize in a wave of contraction,[19] with action potentials being propagated to adjacent myocytes via gap junctions between muscle groups.[9]

Uterine smooth muscle contractions are intermittent, which allows for reperfusion of the uterine muscle, placenta, and fetus between contractions. Each contraction builds in intensity, reaches its peak, and then decreases in intensity until the muscle returns to a state of relaxation, which persists until the next contraction begins. This pattern is often referred to as the increment, acme, and decrement phases of contraction.

The smooth muscle (myometrium) in the uterus is anatomically different from other types of smooth muscle. Located between the decidua and the perimetrium, the myometrium consists of four muscle layers, each of which produces a distinct response to substances that promote or inhibit contractions. The inner circular muscle layer runs perpendicular to the long axis of the uterus in a spiral fashion, while the two outer layers run parallel to this axis. The middle layer has blood vessels running throughout the interlacing fibers. The uterine myocytes are organized in a bundle, contributing to the generation of tension under local stimulation by uterotonic hormones. The uterine muscle is thickest in the fundus, which demonstrates the greatest contractile strength. The fundal portion of the uterus becomes the active segment (**Figure 24-2**).[42] Uterine muscle becomes thinner toward the lower uterine segment in the isthmus; this thinner lower uterine segment forms the muscular tube through which the fetus passes.

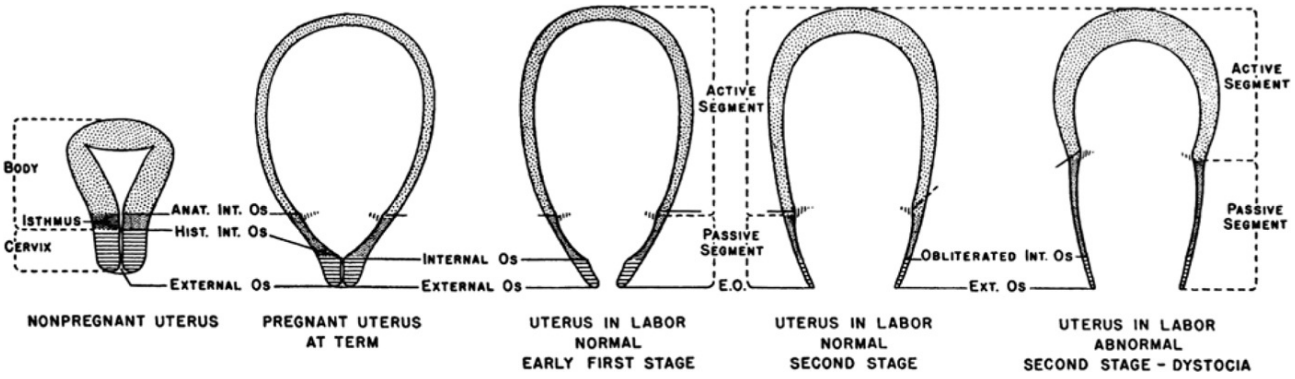


Figure 24-2 Sequence of development of the segments and rings in the uterus in pregnant women at term and in labor. Note the comparison between the uterus of a nonpregnant woman, the uterus at term, and the uterus during labor. The passive lower segment of the uterine body is derived from the isthmus; the physiologic retraction ring develops at the junction of the upper and lower uterine segments. The pathologic retraction ring develops from the physiologic ring.

Abbreviations: Anat. Int. Os = anatomic internal os; E.O. = external os; Hist. Int. Os = histologic internal os.

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The propagation of the contraction wave is often called the triple descending gradient of fundal dominance, wherein contractions (1) start in the fundus, (2) last longer in the fundus, and (3) progress from the fundus toward the isthmus. Fundal dominance is essential for effective cervical dilation but has not been definitively proved to exist. Nonetheless, the muscle bundles in the uterine fundus progressively shorten with each contraction, causing the upper portion of the uterus to thicken and gradually become a smaller cavity. This reduced fundal capacity, in turn, promotes descent of the fetus toward the more passive lower uterine segment. The lower uterine segment muscle bundles become longer in response to contractions in the fundus, creating a distensible structure that is able to accommodate the fetus, a process that promotes fetal descent. The line of demarcation between the active and passive segments is termed the *physiologic retraction ring*. When labor is obstructed, the active segment becomes so much thicker and shorter that one can sometimes see the line between the two uterine segments when observing the abdomen—the so-called *pathologic retraction ring* or *Bandl's ring*.

At the cellular level, contractile units made of myosin and actin that are found in uterine smooth muscle myocytes promote the generation of tension that leads to synchronous smooth muscle contraction. The interaction between the thin actin and thick myosin filaments is central to the generation of force (**Figure 24-3**).[43] Myosin is arranged in two heavy chains and two light chains, with a globular head attached to a tail that protrudes from the heavy chains at regular intervals. The myosin head also contains a site that binds to actin, generating force that is carried along the myosin tails. Entry of calcium into the myocyte in response to specific stimuli prompts intracellular release of additional calcium from a reservoir in the sarcoplasmic reticulum through binding of inositol triphosphate (IP_3) to sarcoplasmic reticulum receptors. Calcium binding to the smooth muscle protein calmodulin causes activation of the myosin light-chain kinase (MLCK) enzyme. In turn, MLCK phosphorylation of

the light chain on the myosin head leads to establishment of a structural link between actin and myosin, catalyzed by myosin adenosine triphosphatase (ATPase) enzyme activity on the myosin head. Energy release from myosin ATPase activity causes the structural cross-bridge to shorten the muscle. At this point, the myosin head rotates and pulls on actin, which generates force and shortening. The action potentials that pass through gap junctions facilitate contraction of adjacent uterine smooth muscle bundles, which results in a coordinated wave of uterine contraction. Decreased intracellular calcium concentrations and the action of myosin light-chain phosphatase enzymatically reverses the actin–myosin linkages through removal of a phosphate from the myosin light chain, prompting smooth muscle relaxation.

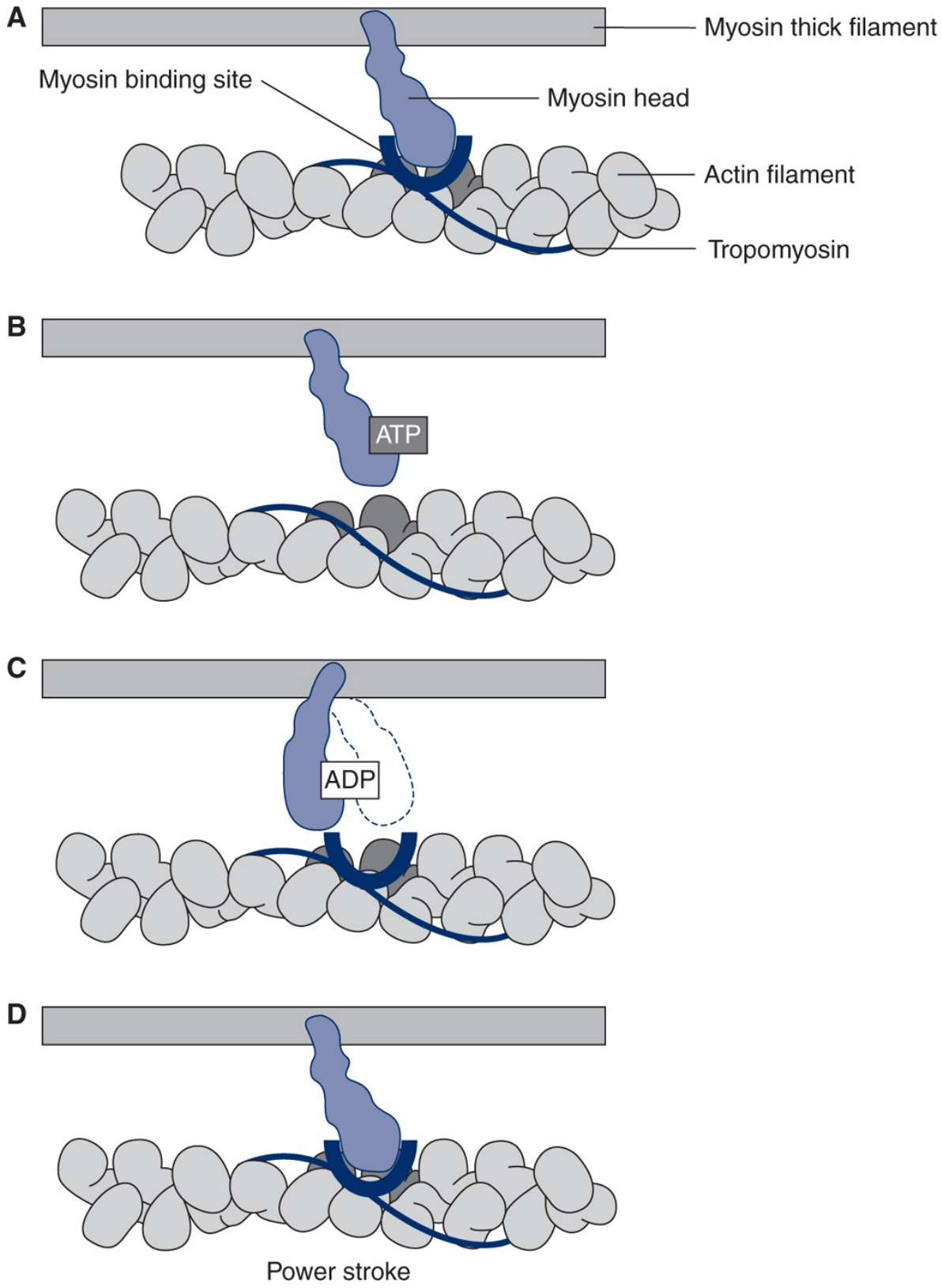


Figure 24-3 Uterine muscle contraction. **A.** Attached: At the start of the cycle, the myosin head is attached to the myosin binding site on the actin filament. **B.** Recharging: A molecule of ATP binds to a large cleft at the back of the myosin head, which causes a slight change in the conformation of the myosin binding site, which reduces the affinity the myosin head has for the myosin binding site. **C.** Cocked: The cleft on the myosin head closes around the ATP, which causes a large shape change so that the head is displaced along the actin filament by a distance of approximately 5 nm. **D.** Force-generating: As ATP is hydrolyzed to ADP, the myosin head once again binds tightly to the next myosin

binding site on the actin filament and in this process the myosin head essentially ratchets along the actin filament, creating the muscle contraction.

Abbreviations: ADP, adenosine diphosphate; ATP, adenosine triphosphate.

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Relaxation involves the removal of contractile stimuli or action of factors that inhibit contractions. Factors that may inhibit this process—and, therefore, that may be used to treat preterm labor contractions—include drugs that stimulate the β-adrenergic signaling pathway (e.g., terbutaline [Brethine]), drugs that inhibit inflammatory pathways (e.g., indomethacin [Indocin]), calcium-channel blockers (e.g., nifedipine [Procardia]), and agents that inhibit the myosin light chain (e.g., magnesium sulfate). In addition, progesterone is used to prevent recurrent preterm labor in women who have experienced a previous preterm birth.

Role of Oxytocin

Oxytocin, a potent uterotonic, is a small nine-amino acid peptide. The word *oxytocin* is derived from the Greek *oxys*, which means "quick," and *tokos*, which means "birth." Many organs have oxytocin receptors, including the uterus, breast, kidney, and central nervous system. Although the primary source of oxytocin is the hypothalamus, oxytocin is also synthesized in the uterus, placenta, corpus luteum, and fetus. Along with exerting uterotonic effects, oxytocin stimulates contraction of myoepithelial cells in the lactating breast (i.e., the let-down reflex), increases sexual arousal in women and men, and may have profound effects on initial maternal–infant bonding. In animal studies, oxytocin has been shown to regulate many maternal behaviors.

For many years, oxytocin was thought to initiate the stimulatory phase of parturition.[37] It is now known that this peptide plays an important role in maintaining labor but most likely does not initiate labor. Oxytocin is manufactured in the hypothalamus and released from the posterior pituitary in a pulsatile fashion. Between 36 and 39 weeks' gestation, pulsations increase in frequency and amplitude in a diurnal rhythm, with maximum plasma levels occurring at night. Trending oxytocin pulses may play a role in the increased frequency of Braxton Hicks contractions in the final weeks of pregnancy.

During spontaneous labor, oxytocin pulses occur approximately 4 times per 30 minutes in the first stage of labor and 7 times per 30 minutes in the second stage. The biologic half-life of this molecule is approximately 3 to 4 minutes. Plasma levels of oxytocin do not correlate with uterine contractility or cervical dilation. Instead, the number and distribution of oxytocin receptors on the surface membranes of myocytes influence the strength and frequency of uterine contractions in labor. The number of oxytocin receptors in the uterus increases 100-fold during the last weeks of pregnancy, reaching a maximum during labor.[34,35] This increase in receptor density explains the increased sensitivity to oxytocin displayed by uterine myometrial cells. Early in pregnancy, the uterus is not sensitive to oxytocin. Myometrial receptivity starts at approximately 20 weeks' gestation and slowly increases over the last half of pregnancy.

The pivotal role of oxytocin receptors also explains why oxytocin can cause both uterine

contractions and uterine atony. The oxytocin receptors can become saturated after a period of time of constant agonist stimulation and downregulate via endocytosis. Thus, when synthetic oxytocin infusions are used for induction or augmentation of labor, the woman may have an increased risk for uterine atony after birth secondary to downregulation of oxytocin receptors.

Clinical Stages of Labor

Labor and birth are divided clinically into four stages. The *first stage of labor* includes three phases. The *latent phase* is the initial period of time wherein contractions become regular, more painful, and more frequent but little cervical dilation occurs. In the *active phase* of labor, the cervix dilates over a period of hours to complete dilation (i.e., 10 centimeters). Historically, it was believed that the active phase of labor includes a *transition phase* that occurs between 8 and 10 centimeters of cervical dilation, when progress is slower. Recent studies of labor progress have not demonstrated that this phase appears consistently in all women.

The *second stage of labor* refers to the time period from complete dilation to birth of the newborn. During the second stage of labor, the woman actively pushes in concert with uterine contractions. When women do not have regional analgesia, the urge to push or bear down is involuntary and thought to occur secondary to pressure of the fetal head on the vaginal floor and rectum.

The *third stage of labor* is defined by the time between birth of the newborn and birth of the placenta. The *fourth stage of labor* is the first hour after birth. The fourth stage of labor is not always referred to in clinical texts but is considered important clinically because it is the time during which the woman is at highest risk for postpartum hemorrhage.

The normal durations and clinical management of each stage of labor are reviewed in more detail in the *First Stage of Labor*, *Second Stage of Labor and Birth*, and *Third Stage of Labor* chapters.

Maternal Fetopelvic Relationships During Labor

The obstetric pelvic types and fetopelvic relationships are reviewed in the *Anatomy and Physiology of Pregnancy* chapter. Confirmation of the fetal presentation and position is made during a vaginal examination once the cervix is dilated enough to feel the fetal presenting part. Fetal suture lines and fontanels, or portions of the fetal face, breech, external genitalia, or fetal extremities (hands or feet), are palpated. Cephalic vertex presentations are by far the most common, so familiarity with the basic landmarks of the fetal skull is essential for monitoring labor progress. The position of a fetus in a vertex presentation is determined vaginally by feeling the anterior or posterior fontanel (the fontanel shape and number/direction of the sutures leading off the fontanel) and identifying which fontanel is in which side and portion of the maternal pelvis. When identifying the parts of the fetal skull, one starts by finding the sagittal suture, which runs directly between the anterior fontanel at one end and the posterior fontanel at the other end. As the fetus passes through the maternal pelvis, additional measurements that describe the relationship of the fetus to the pelvis during labor are noted and documented in the health record, including station, synclitism, asynclitism, caput succedaneum, and molding.

Station

Station is where the lowermost part of the fetal presenting part resides relative to an imaginary line drawn between the ischial spines of the woman's pelvis (**Figure 24-4**). The line between the ischial spines is called 0 station. Station is measured in terms of the number of centimeters above or below this line between the ischial spines. When the presenting part is above the spines, station is designated as –1, –2, –3, –4, or –5. When the lowermost part of the presenting part (the bone, not swelling or soft tissue) is lower than the level of the ischial spines, the presenting part is at +1, +2, +3, +4, or +5 station. When the fetus is at 0 station, the biparietal diameter is most always descended through the inlet and, thus, is engaged. When the presenting part is at +5 station, it will be visible at the vaginal introitus.

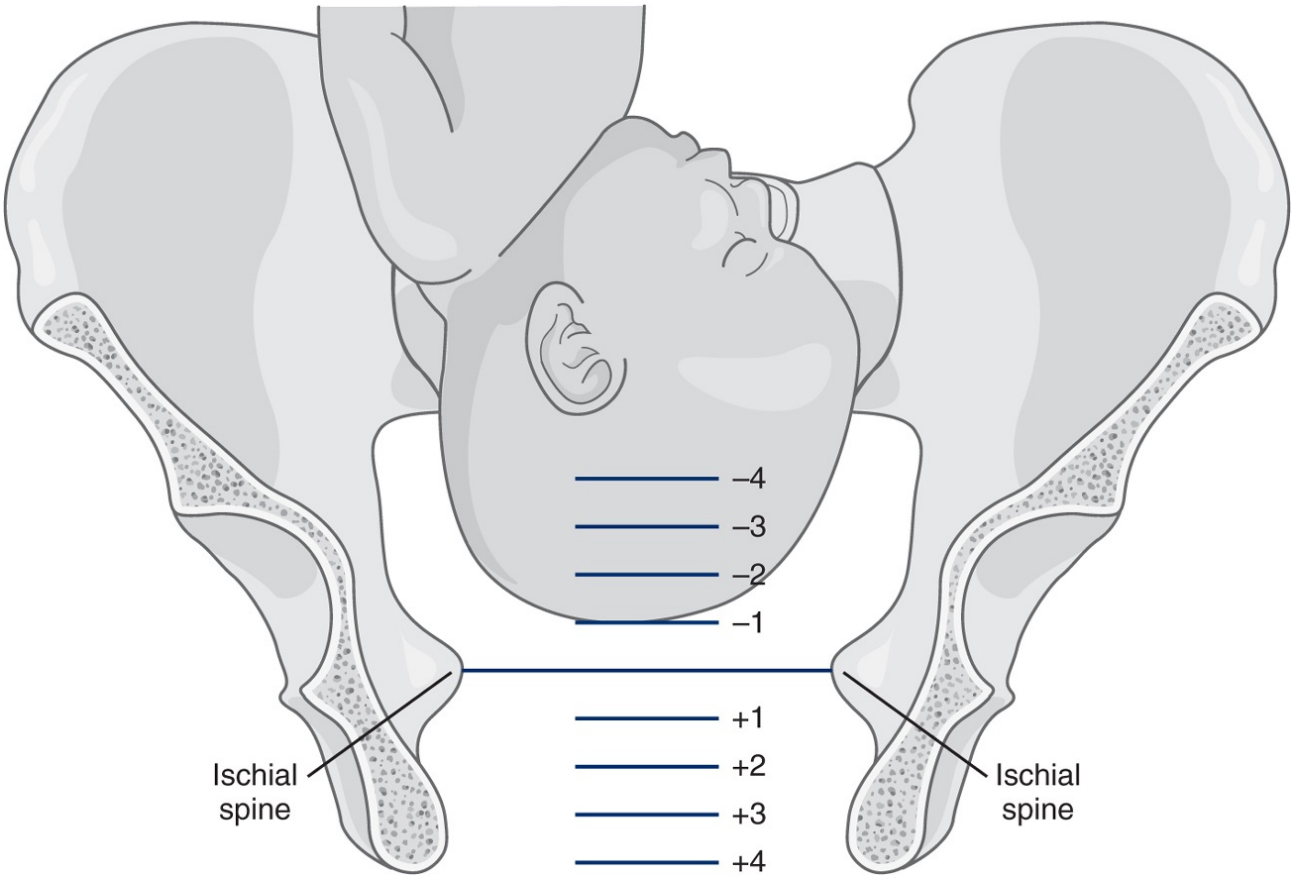


Figure 24-4 Station, or level of descent of the head of the fetus through the pelvis. The location of the forward leading edge (lowest part of the head) is designated in centimeters above or below the plane of the interspinous line.

Synclitism and Asynclitism

Synclitism and *asynclitism* are terms that describe the relationship of the sagittal suture of the fetal head to the symphysis pubis and the sacrum of the maternal pelvis—a relationship determined when the anteroposterior diameter of the fetal head is in alignment with the transverse diameter of the pelvic inlet. This orientation places the sagittal suture line of the fetal skull in the same line as the transverse diameter of the pelvic inlet, and the occiput of the fetal head in the transverse portion of the maternal pelvis. With synclitism, the sagittal suture is located midway between the symphysis pubis and the sacral promontory. With asynclitism, the fetal neck is tilted so that the fetal head leans laterally toward the fetal shoulder somewhat. The alignment of the sagittal suture and transverse diameter of the maternal pelvis are not exact with asynclitism, and the sagittal suture tends to be closer to the symphysis pubis or to the sacral promontory (**Figure 24-5**).

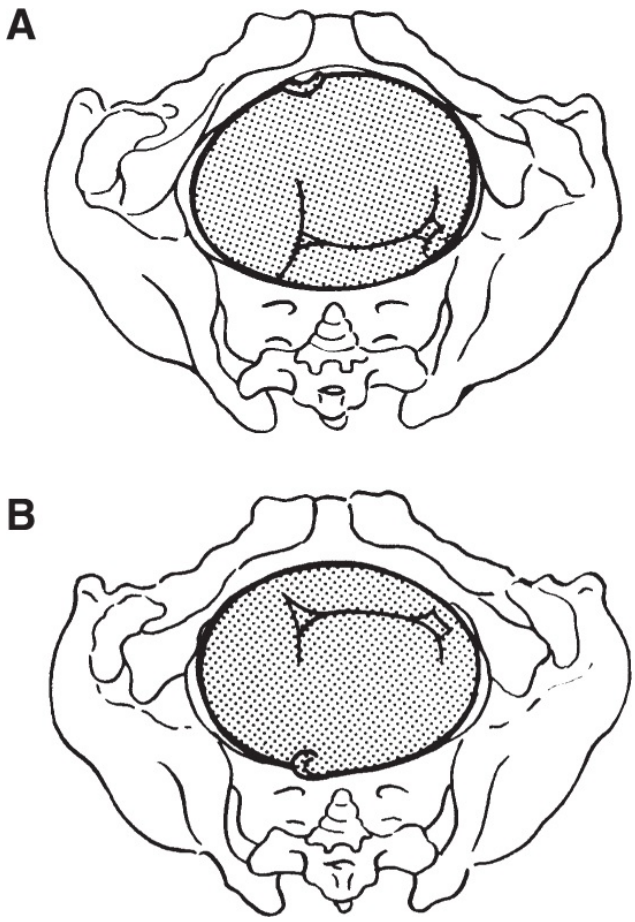


Figure 24-5 Asynclitism. **A.** Anterior. **B.** Posterior.

Determination of anterior asynclitism or posterior asynclitism is based not on the maternal pelvic structure to which the sagittal suture is closer, but rather on which parietal bone is dominant. Therefore, anterior asynclitism occurs when the anterior parietal bone (the one closest to the symphysis pubis) becomes the lowermost or leading part of the presenting part, due to flexion of the head toward the sacral promontory, causing the sagittal suture to lie closer to the sacral promontory. Posterior asynclitism occurs when the posterior parietal bone (the one closest to the sacral promontory) becomes the lowermost part of the presenting part as a result of flexion of the head toward the symphysis pubis, causing the sagittal suture to lie closer to the symphysis pubis. In normal labor, the fetal head usually enters the pelvic inlet with a moderate degree of posterior asynclitism and then changes to anterior asynclitism as it descends farther into the pelvis before the mechanism of internal rotation occurs. This sequential change from posterior to anterior asynclitism facilitates the mechanism of descent; it is an accommodation by the fetus to take advantage of the roomiest portions of the true pelvis.

Molding and Caput Succedaneum

Molding and caput succedaneum are both conditions that result from pressure exerted on the fetal head by the maternal structures of the birth canal during labor and birth. Molding is the change in the shape of the fetal head as a result of the soft skull bones' overriding, or overlapping, one another because they are not yet completely fused, so that movement is

possible at the location of the sutures. Minor degrees of molding occur as the fetal head negotiates the maternal pelvis and are initially considered a normal finding.

The shape of the head depends on the presentation and attitude, which determines the parts of the skull that present first and, therefore, are subjected to pressure. Occiput cephalic presentations are most common, so molding usually occurs as the parietal bones override the occipital bone, which in effect obliterates or minimizes the posterior fontanel. A bony ridge may be palpable where the parietal bones override the occiput. When the parietal bones overlap at the sagittal suture, which is not uncommon, the parietal bone that was anterior in the pelvis overlaps with the "posterior" parietal bone, which was depressed because of pressure from the sacral promontory. Therefore, when a fetus in a right occiput transverse (ROT) position rotates to right occiput anterior (ROA), the left parietal bone overrides the right parietal bone; the reverse is true for a fetus in a left occiput transverse (LOT) position that rotates to left occiput anterior (LOA).

In regard to the overriding of the parietal bones, the location of the left and right parietal bones (either anterior or posterior) should not be confused with anterior and posterior asynclitism. Whether the left or right parietal bone is considered "anterior" is based solely on whether the occiput is to the left or right of the pelvis, which then determines which bone lies closest to the symphysis pubis and which is closest to the sacrum.

Molding involves the entire skull, with overlapping in one area being counterbalanced by movement elsewhere. This creates harmony between the base and the vertex of the skull and prevents destructive tension and possible rupture of the cranial membrane, the dura mater.

Caput succedaneum (commonly referred to as "caput") is the formation of an edematous swelling over the most dependent portion of the presenting fetal head. Pressure around the presenting part by the cervical opening produces congestion and edema of the portion of the fetal head that presents against the cervical opening. If the fetal membranes are ruptured and the fetal head (rather than the membranes) is functioning as the dilating wedge against the cervical opening, a greater amount of caput succedaneum will likely form. If the fetal head is unusually molded or significant caput is present, the head may not be engaged in the pelvis at all, so caput and molding can be clinically quite important. After birth, caput succedaneum can be differentiated from the more serious condition known as cephalohematoma by the fact that caput succedaneum crosses suture lines as a generalized swelling, whereas a cephalohematoma (bleeding beneath the periosteum) may occur over more than one cranial bone but is limited to each individual bone and does not cross any sutures.

The formation of a few millimeters of caput succedaneum is considered normal. A small caput succedaneum may develop during a somewhat prolonged labor resulting from uterine inertia with weak contractions. Formation of extensive caput succedaneum, making the identification of fetal sutures and fontanels impossible, combined with a more severe degree of molding, is usually seen when the pressure has been great and labor prolonged; cephalopelvic disproportion must be suspected in such a case. A sizable caput may also be seen from positional pressure when the fetus was in an occipitoposterior position for a relatively prolonged period. It is important to make sure station is determined based on the level of the fetal skull bone (usually the occiput) and not based on where the caput is in relation to the ischial spine. If caput, which precedes the bone, is used to determine fetal station, the clinician

might mistakenly assume the fetal head is engaged when it is not engaged, or descending when caput is increasing but the head is not descending.

Cardinal Movements of Labor

The *cardinal movements of labor*, also called the mechanisms of labor, describe the movements made by the fetus during labor (**Figure 24-6**) and immediately before birth to negotiate the diameters of maternal pelvis (**Table 24-1**). Understanding the cardinal movements of labor requires knowing the essential average diameters of the fetal head. Although the majority of fetuses enter labor in a cephalic presentation, knowledge of the cardinal movements of labor for each fetal presentation, position, and denominator is essential for the midwife. The cardinal movements are engagement, descent, flexion, internal rotation, extension, restitution, external rotation, and lateral flexion or expulsion.

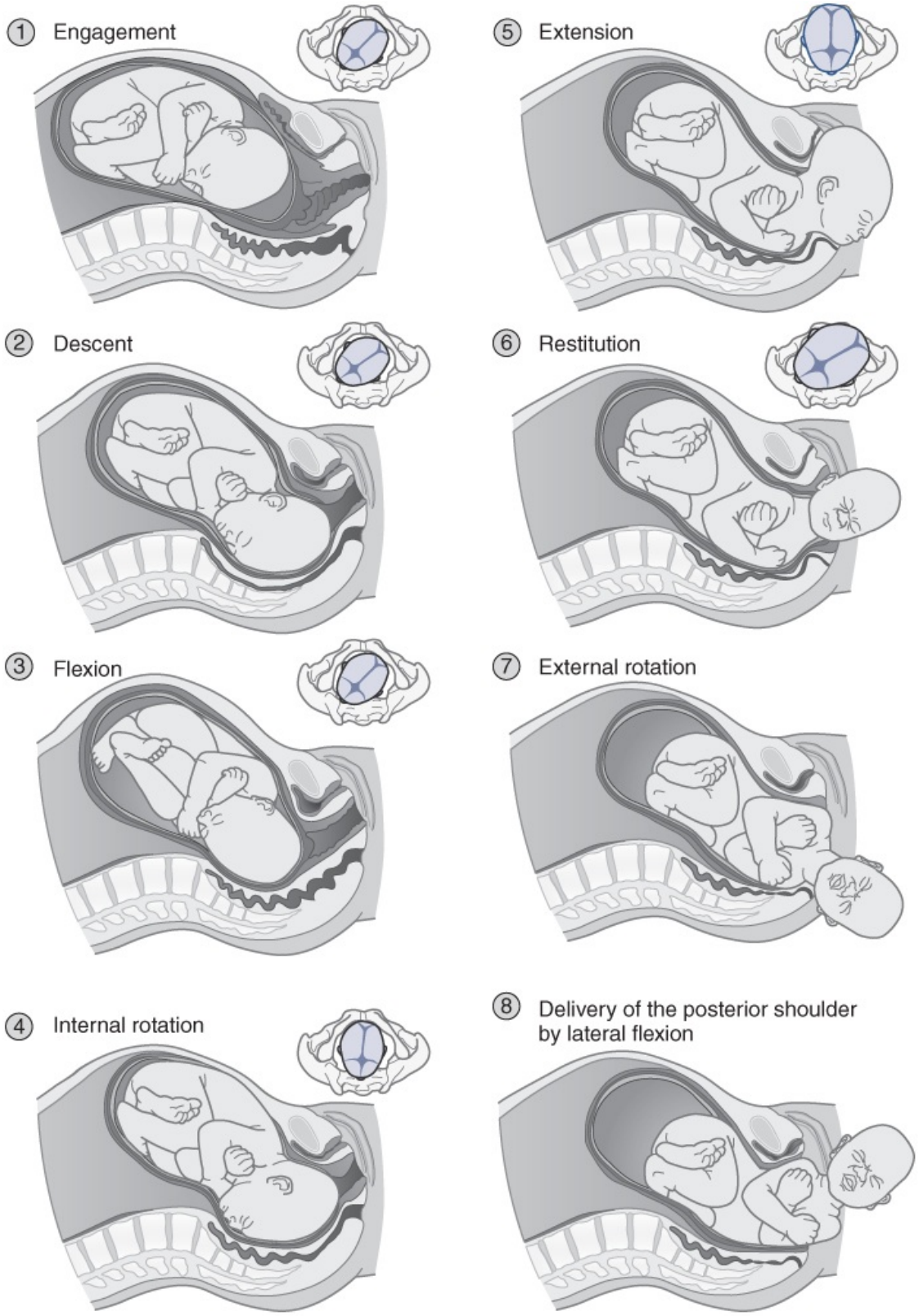


Figure 24-6 Cardinal movements of labor for a fetus in an occiput anterior position.

Table 24-1	Cardinal Movements of Labor for Occiput Anterior and Persistent Posterior Positions

Cardinal Movement	Occiput Anterior	Persistent Posterior

Engagement	ROT or LOT position	Right or left oblique diameter
Descent	Occurs throughout	Occurs throughout
Flexion	Suboccipitobregmatic diameter presenting	Usually less completely flexed
Internal rotation	45° LOA or ROA, 90° LOT or ROT, 135° LOP or ROP to OA	45° LOP or ROP to OP
Extension	Birth of head by extension	Birth of head by first flexion and then extension
Restitution	45° to LOA or ROA	45° to LOP or ROP
External rotation	45° to LOT or ROT	45° to LOT or ROT
Lateral flexion or expulsion	Birth of shoulders by lateral flexion	Birth of shoulders by lateral flexion

Abbreviations: LOA, left occiput anterior; LOP, left occiput posterior; LOT, left occiput transverse; OA, occiput anterior; OP, occiput posterior; ROA, right occiput anterior; ROP, right occiput posterior; ROT, right occiput transverse.

Engagement occurs when the biparietal diameter of the fetal head has passed through the pelvic inlet. *Descent* occurs throughout labor and, therefore, is requisite to and occurs simultaneously with the other cardinal movements. Descent is the result of contractions and maternal pushing efforts during the second stage.

Flexion is essential to further descent once engagement has occurred. During this third cardinal movement, the smaller suboccipitobregmatic diameter becomes the widest fetal head diameter that traverses the maternal pelvis. When the fetal head is flexed so that the fetal chin rests on the thorax, the suboccipitobregmatic is substituted for the larger fetal head diameters that exist when the fetal head is not completely flexed, is in a military attitude, or is in some degree of extension. Flexion occurs when the fetal head meets resistance; this resistance increases with descent and is first met from the cervix, then from the sidewalls of the pelvis, and finally from the pelvic floor. Some degree of flexion, therefore, may occur prior to engagement.

Internal rotation brings the anteroposterior diameter of the fetal head into alignment with the anteroposterior diameter of the maternal pelvis. Most commonly, the occiput rotates to the anterior portion of the maternal pelvis, beneath the symphysis pubis. If internal rotation has not occurred by the time the fetal head has reached the pelvic floor, it takes place shortly thereafter. Internal rotation is essential for vaginal birth to occur, except in unusually small fetuses. The pelvic inlet has a larger transverse diameter than anteroposterior diameter; the midplane and the outlet have larger anteroposterior diameters than transverse diameters. Internal rotation is effected by the V-shape of the pelvic floor musculature and the decreased dimensions of the pelvic cavity. The amount of internal rotation is determined by the distance the occiput must travel from its original position on entering the pelvis to the occiput anterior or occiput posterior position that precedes birth. This distance is expressed in degrees, as it is a portion of the arc of a circle that is being traversed.

When the fetal occiput rotates from an LOP (left occiput posterior), ROP (right occiput posterior), LOT, or ROT position, the shoulders also rotate with the head until the LOA or ROA position has been reached. As the occiput rotates the final 45 degrees into the occiput anterior (OA) position, the shoulders do not continue their rotation with the head, but instead

enter the pelvic inlet in one of the oblique diameters (the left oblique diameter for an LOA and the right oblique diameter for an ROA). The entire cardinal movement, therefore, has the effect of turning the fetus's neck by 45 degrees.

Birth of the head occurs by *extension* (of the fetal head so the chin lifts up from the thorax) in occiput anterior births. This cardinal movement of labor is different when the occiput rotates to an occiput posterior (OP) position, as explained later in this chapter. Extension must occur when the occiput is anterior because of the resistant force of the pelvic floor, which has a curve called the curve of Carus that directs the head upward to the vaginal introitus (**Figure 24-7**). The *curve of Carus* is the lower exiting end of the pelvic curve; the fetus and placenta must follow this curve to be born. The pelvic cavity actually resembles a curved cylinder, such that the direction of either the baby or the placenta coming through it is first downward from the axis of the inlet to just above the tip of the sacrum, and then forward, upward, and outward to the vaginal opening. The suboccipital region impinges under the symphysis pubis and acts as a pivot. At this point, the fetal head is positioned so that further pressure from the contracting uterus and maternal pushing serve to further extend the head as the vulvovaginal orifice opens. Thus, the head is born by extension as the occiput, sagittal suture, anterior fontanel, brow, orbits, nose, mouth, and chin sequentially sweep over the perineum. The suboccipitofrontal diameter is, therefore, the largest anterior–posterior diameter to pass through the vulvovaginal orifice.

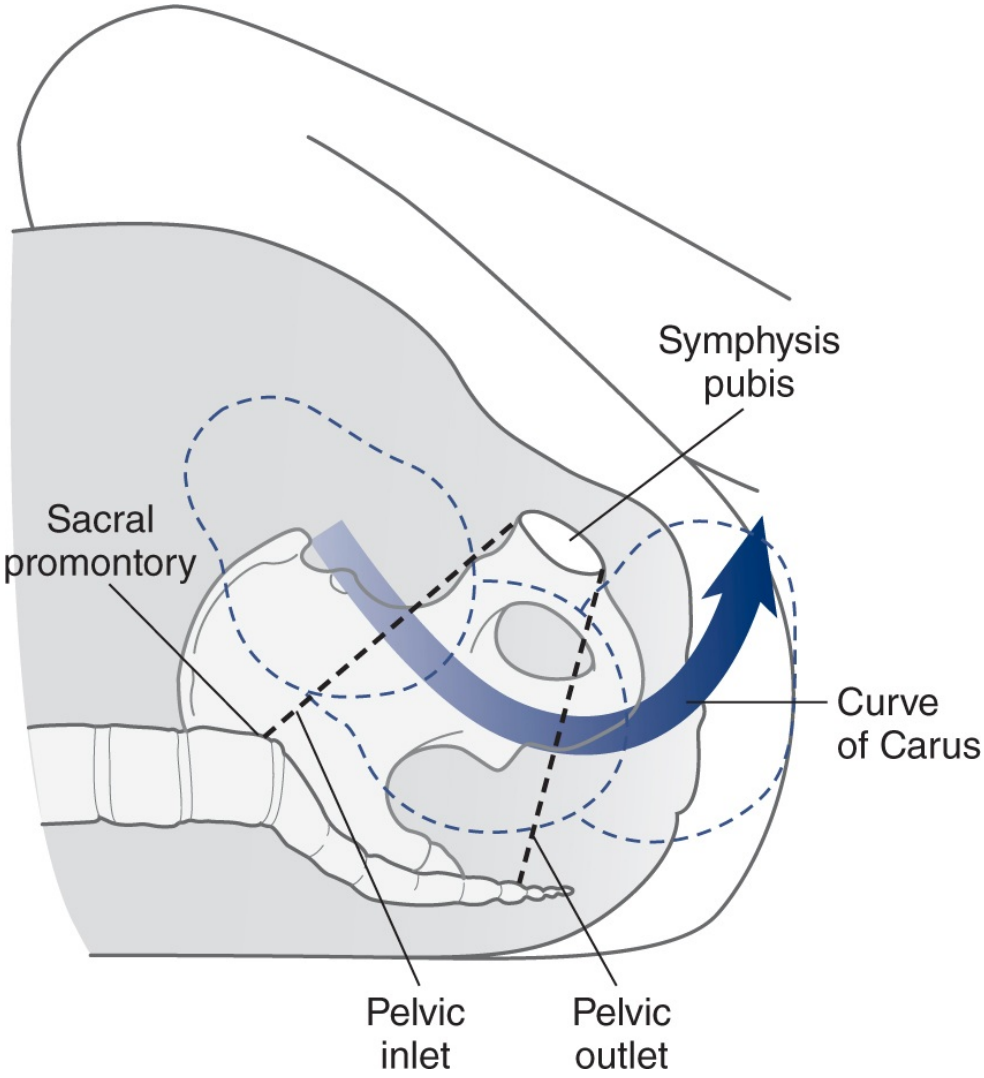


Figure 24-7 Curve of Carus.

Restitution is the rotation of the head 45 degrees to either the left or the right, depending on the direction from which it rotated into the OA or OP position. In effect, in restitution, the neck turns back so that the head is again at a right angle with the shoulders. The sagittal suture is now in one of the oblique diameters of the pelvis, and the bisacromial diameter of the fetus is in the other oblique diameter of the pelvis.

External rotation occurs as the fetus's shoulders rotate 45 degrees, bringing the bisacromial diameter into alignment with the anteroposterior diameter of the pelvic outlet. This causes the head to rotate externally another 45 degrees into the LOT or ROT position, depending on the direction of restitution.

Birth of the shoulders and body occurs by *lateral flexion* to accommodate the curve of Carus. The anterior shoulder comes into view at the vaginal opening, where it impinges under the symphysis pubis; the posterior shoulder then distends the perineum and is born by lateral flexion. After the shoulders are assisted to exit, the remainder of the body follows the curve of Carus and the fetus is readily born.

Cardinal Movements for Occiput Anterior Position

Variations of the eight basic positional movements are determined by the position of the fetus and must be delineated for each movement. When a fetus begins labor in the LOA, LOT, LOP, ROA, ROT, or ROP position and delivers in an occiput anterior position, the cardinal movements are as follows:

1. *Engagement* takes place for the fetus in the LOT and ROT positions, with the fetal sagittal suture in the transverse diameter of the pelvic inlet and the biparietal diameter of the fetus in the anteroposterior diameter of the pelvic inlet. For the LOA, ROA, LOP, and ROP positions, engagement of the fetal head takes place with the sagittal suture in one of the oblique diameters of the pelvis (right oblique diameter for the LOA and ROP positions and left oblique diameter for the ROA and LOP positions). The biparietal diameter, therefore, is in the oblique diameter of the pelvis opposite from the one the sagittal suture is in. The sagittal suture is used as the fetal landmark that determines in which oblique diameter the fetal head enters the pelvis.
2. *Descent* occurs throughout labor.
3. *Flexion* substitutes the suboccipitobregmatic diameter for the diameter that entered the pelvic inlet.
4. *Internal rotation* takes place as follows: 45 degrees (for the LOA and ROA positions), 90 degrees (for the LOT and ROT positions), and 135 degrees (for the LOP and ROP positions—referred to as long arc rotation). The fetal head is now in the OA position in the anteroposterior diameter of the mother's pelvis.
5. Birth of the head occurs by *extension*.
6. *Restitution* is 45 degrees to the LOA or ROA position. The fetal head moves left if the fetus began the cardinal movements of labor with the occiput in the left side of the pelvis; it moves right if the fetus began the cardinal movements of labor with the occiput in the right side of the pelvis.
7. *External rotation* is 45 degrees to the LOT or ROT position. The direction of the rotation of the shoulders is determined by the direction of restitution. External rotation brings the bisacromial diameter of the shoulders into the anteroposterior diameter of the maternal pelvis.
8. Birth of the shoulders and body occurs by *lateral flexion* via the curve of Carus.

Cardinal Movements for Persistent Occiput Posterior Position

A persistent posterior position occurs when the fetus in a left or right occiput posterior position undergoes internal rotation through a short arc of 45 degrees to a direct OP position in the anteroposterior diameter of the maternal pelvis, instead of the more common long arc rotation of 135 degrees to a direct OA position, as described in the previous subsection. Short arc rotation is much less common, occurring approximately 6% to 10% of the time. It is most frequently observed in women with an anthropoid or android type of pelvis.

The cardinal movements of labor for a fetus that begins in the LOP or ROP position and delivers in the OP position are the same as for those fetuses that rotate to an occiput anterior position except as explained here:

1. *Engagement* takes place in the right oblique diameter for the ROP position and in the left oblique diameter for the LOP position.
2. *Descent* occurs throughout.
3. *Flexion* is the same.
4. *Internal rotation* takes place, with the fetal head rotating 45 degrees to an occiput posterior position in the anteroposterior diameter of the mother's pelvis.
5. Birth of the head occurs by the double cardinal movements of first *flexion* and then *extension*. The sinciput impinges beneath the symphysis pubis and becomes the pivotal point for delivery of the head. The head stays flexed as the occiput distends the perineum and is born to the nape of the neck. The remainder of the head is then born by extension, starting with the anterior fontanel and ending with the chin, as the head falls back toward the rectum, with the face looking upward.
6. In *restitution*, the fetal head rotates 45 degrees to the LOP or ROP position, depending on whether internal rotation was from the LOP or ROP position.
7. In *external rotation*, the fetal head rotates 45 degrees to the LOT or ROT position.
8. Birth of the shoulders and body occurs by *lateral flexion* via the curve of Carus.

Persistent posterior position is considered a variation of normal labor. This position may sometimes be maintained for the entire second stage of labor, but not always. A woman with a fetus in a persistent posterior position may experience additional and often severe back pain; alternatively, women who experience severe back pain in labor may have a fetus in an occiput anterior position, so this symptom is not diagnostic for fetal position. Diagnosis of a posterior position is made by abdominal examination and confirmed by vaginal examination or ultrasound. Observation of the contour of the woman's abdomen may give the first clue of a posterior position. A depression in the shape of a saucer is commonly seen at or just below the woman's umbilicus. This depression occurs because the fetus's shoulder is posterior rather than anterior, so there is not a smooth anterior curve but rather what looks like a gap between the cephalic and podalic poles or feet of the fetus. If the head is not engaged, there is a bulge between the symphysis pubis and the saucer-shaped depression.

Maternal Systemic Adaptations in Labor

The adaptations of pregnancy that developed progressively during the quiescent time of gestation provide the essential preparation for the final stages of pregnancy in the intrapartum period. The significant physiologic demands associated with the activation and stimulation phases of labor require maternal adaptations to meet the transient challenges imposed on the maternal and fetal systems.

Cardiovascular Adaptations

The placenta/maternal interface is a low-pressure, high-volume compartment, and blood volume must expand in pregnancy to accommodate this space. The increase in the maternal blood volume reaches its acme of approximately 40% compared to the nonpregnant blood volume in the early third trimester, and is accompanied by a concomitant increase in the cardiac output and left ventricular stroke volume.[44] Oxygen-carrying capacity is supplemented by similar increases in red blood cell (RBC) production, thereby maximizing delivery of oxygen to both the maternal systems and the fetus. Maternal vascular system hypertrophy is promoted by nitric oxide–mediated vasodilation and a progesterone-mediated reduction in vascular tone and resistance, thereby enabling the woman to accommodate the increased blood volume and perfusion requirements of the maternal–placental–fetal unit. The increased plasma volume and decreased viscosity reduce resistance to flow—a phenomenon further supported by decreased vascular resistance; the result is the development of a physiologic arteriovenous shunt that promotes accommodation of increased blood volume and cardiac output.

During labor and birth, additional marked and rapid hematologic adaptations occur secondary to uterine contractions, pain, exertion, loss of the placental compartment at birth, and uterine involution.[45] The woman's systolic and diastolic blood pressures both increase during uterine contractions, returning to baseline between contractions.[44,45] Blood pressure and heart rate may also increase due to pain. Although heart rate often increases, the direction of change is variable, such that both increases and decreases can be observed. The laboring woman's pulse commonly increases during contractions and decreases between contractions. Her cardiac output increases by an additional 10% to 15% in first-stage labor and by as much as 50% in second-stage labor, due primarily to increased stroke volume.[44,46] Cardiac output can also be affected by maternal pain, anxiety, and any anesthetics used.

An inverse relationship exists between myometrial contraction intensity and duration and placental blood flow, with perfusion being reduced during maximal contraction. Restoration of resting tone between contractions provides the opportunity to reestablish perfusion. Approximately 500 to 900 mL of maternal blood flows through the maternal side of the placental site each minute at term.[47,48] Shifts in blood volume that occur during contractions force approximately 300 to 500 mL of blood into the maternal venous system, contributing to increased venous return. Increases in central venous pressure during the second stage of labor can result from pushing efforts, prompting parasympathetic nervous system stimulation during Valsalva maneuvers. Redistribution of blood flow in the maternal system after delivery of the placenta leads to splanchnic circulation vasoconstriction, shunting an additional 500 mL of

blood from the uteroplacental circulation to the maternal circulation. This auto-infusion phenomenon serves to protect the woman when she experiences the usual blood loss of approximately 500 mL after vaginal delivery, by increasing central venous pressure, ventricular preload, and cardiac output. These numbers are not exact, as measurement of blood volume changes during labor is not easily performed.

A woman's cardiac output increases 60% to 80% immediately after birth as the placental site is removed and extra volume is auto-transfused into the maternal circulation. This is followed by a rapid decrease of maternal blood volume within 10 to 60 minutes, but it can take a few months before cardiovascular indices return to their prepregnant levels.[44,49] These remarkable shifts in blood volume, cardiac output, and systemic venous return can be hazardous for women who have preexisting cardiac disorders.

Hematologic Indices During Labor and Birth

Changes in coagulation factors initiated during pregnancy are enhanced during labor, promoting rapid hemostasis after placental separation. Levels of coagulation factors—most notably factor VIII—are markedly increased during active labor. Tissue factor release from the placenta and decidua activates the extrinsic coagulation pathway, promoting clot development. The procoagulant condition reaches its maximal point after placenta separation, with increases in factor V, platelet activation, and fibrin clot formation. Clot development contributes to reduced circulating fibrinogen and platelets, as these components are utilized in the production of fibrin clots. A simultaneous decrease in fibrolytic activity enhances clot development after placental separation, associated with decreased levels of circulating plasminogen.

After birth, clot dissolution is associated with increased production of fibrin degradation products; moreover, d-dimer may promote an anticoagulant effect, potentially interfering with clot development.[50] Uterine contraction and compression of uterine vessels serve to promote accumulation of clotting factors and hemostasis after placental separation, in turn reducing the risk of excessive maternal blood loss.[51] In the event of uterine atony and maternal bleeding, uterine massage and exogenous uterotonics can be administered to promote contraction and intrinsic vasoconstriction. As uterine blood flow contributes to increases in cardiac output ranging from 1% in the nonpregnant woman to 15% at the end of pregnancy, interference with adequate uterine contraction and hemostasis after placental separation can lead to massive maternal hemorrhage.[52]

A slight degree of leukocytosis occurs gradually over the course of pregnancy. During labor the white blood cell count peaks and can be as high as $20 \times 10^3/mm^3$. The etiology of this rise in leukocytes is not known but it is a normal finding and not indicative of infection. Thus, the white blood cell count is not a clinically useful biomarker for infection during labor.

Respiratory Adaptations

Pregnancy adaptations that lead to reduced total lung capacity, residual volume, and functional residual capacity as well as increased tidal volume[53] promote the respiratory system changes that support the woman's increased oxygen requirements during the activation and stimulation

stages of labor. Increased muscular work, metabolic rate, and oxygen consumption can be major challenges to the maternal respiratory system during labor.

Oxygen consumption increases as a woman's muscular activity increases during uterine contractions. Inter-contraction periods of relaxation allow for reperfusion and restoration of oxygen content to the uterine myometrium. Failure to restore oxygenation to uterine muscle cells over time results in anaerobic metabolism, production of lactic acid, and subsequent ischemic pain that is theorized to contribute to the uterine pain felt during labor.

Pain can also lead to an increased respiratory rate and hyperventilation, promoting respiratory alkalosis. During pushing efforts, voluntary muscle activity further contributes to the increased maternal PCO_2 and base deficit, leading to a decline in maternal arterial pH values. A reduction in the woman's respiratory rate in the third stage of labor promotes the return to the usual acid–base balance, which typically normalizes within 24 hours of delivery.

Gastrointestinal Adaptations

The hormonal and anatomic effects on the gastrointestinal system during pregnancy, including delayed motility and reflux, are retained as the quiescent stage transitions into the activation and stimulation stages of labor. The combined effects of decreased gastric motility, relaxation of the gastroesophageal sphincter, and increased intra-abdominal pressure contribute to an increased risk for emesis and aspiration if a laboring woman is sedated or intubated.[53] Transient nausea and vomiting may occur during active labor, particularly during the transition phases.

Nutritional Needs During Labor

Energy demands during the first stage of labor are related to increased cardiac output and work of the respiratory and uterine muscles.[54] The additional work required of voluntary muscles in pushing efforts during the second stage of labor further increases the demand for energy.[55,56] Hypoglycemia due to fasting can lead to use of alternative metabolic pathways that result in accumulation of by-products including lactate and ketones.[57] Strategies that provide energy while limiting risks due to increased volume of gastric contents, including intake of isotonic sports drinks[52,56] and consumption of a light diet,[57] are not associated with any adverse outcomes. Restriction of solid food intake is a common practice that is based on the concern that a woman may aspirate if her stomach is full and she needs to be intubated for an emergency cesarean. However, aspiration is extremely rare and studies have not found either benefit or harm from restriction of food and fluids in laboring women who are at low risk of needing general anesthesia.[58-61]

Pain and Psychobiologic Responses in Labor

Women's experience of pain during labor and methods for mitigating labor pain are reviewed in more detail in the *Support for Women During Labor* chapter and briefly introduced here. Physically, mechanical pressure on anatomic structures, uterine contractions, and cervical

changes are the primary physiologic and anatomic mediators of pain during labor.[62] Pain associated with contractions is conducted from pain receptors (nociceptors) to afferent type A delta and C neurons extending from the uterus to the dorsal spinal cord. The afferent neurons enter the spinal cord via segments of the 10–12 thoracic spinal nerves. Fetal descent induces mechanical pressure on the pelvic floor, vagina, and perineum during the second stage of labor, mediating sensory pain impulses via the pudendal nerve to the 2–4 sacral spinal nerves. Interpretation of pain is achieved when the pain impulse ascends the spinal cord to higher cortical brain centers.[62,63]

The experience of labor pain is, however, a complex dynamic process influenced by multiple factors. For example, anxiety and fear of labor appear to increase the pain of labor.[64] Previous traumatic birth experience, post-traumatic stress disorder, a history of sexual or other abuse, or other unpleasant memories can also worsen the perception of pain during labor.[65–71]

Modulation of pain can be achieved through interruption of the pain pathway (i.e., entry of pain signals into the spinal cord, spinal neurons, and higher brain centers). This knowledge forms the basis for many pain relief strategies.[72-76] For example, decreased transmission of the pain impulse can be promoted through stimulation of inhibitory afferent type A beta pain fibers using interventions such as massage.

Cognitive strategies such as distraction and relaxation techniques can also reduce pain by targeting the woman’s higher cortical brain centers. Attenuation of fear, stress, and anxiety via provision of continuous labor support reduces the perception of pain in labor.[72] Other strategies to promote an optimal psychobiological response to labor include education and anticipatory guidance, support for decision making, and nonpharmacologic methods of pain relief.[72-76]

Fetal Response to Labor

The fetal response to labor is reviewed in detail in the *Fetal Assessment During Labor* chapter. In brief, the primary clinical focus of the fetal experience of labor is maintenance of gas and nutrient exchange across the placenta and through the umbilical cord in the presence of recurrent uterine contractions and fetal descent.

Approximately 40% of fetal cardiac output is located in the fetal–placental circulation. The partial pressure of oxygen at the placental interface is similar to that in the maternal venous system. The fetus maximizes oxygen-carrying capacity via several physiologic parameters, including the presence of fetal hemoglobin, which has a high affinity for oxygen; increased levels of hemoglobin (compared to adult values); a high cardiac output; and a fast heart rate.

Uterine contractions during active labor interrupt delivery of maternal blood to the intervillous space. When researchers used near-infrared spectrometry to measure changes in fetal brain oxyhemoglobin (HbO_2) and deoxyhemoglobin (Hb), they observed a negative change in HbO_2 and a positive change in Hb when the interval between contractions was less than 2 minutes, and a positive change in HbO_2 following longer contraction intervals.[77] Shorter uterine relaxation time, longer contractions, and higher contraction amplitude during labor are more likely to lead to acidosis, as measured by cord blood pH at birth (umbilical artery pH of 7.11 or less), compared to newborns who experience less uterine activity during labor.[78]

When an interruption in the transport of oxygen from the maternal circulation to the fetal circulation occurs that is sufficient to cause a decrease in fetal partial oxygen pressure, the fetus responds with several compensatory mechanisms, including shunting of blood preferentially to vital organs such as the brain, myocardium, and adrenal gland. In addition, the fetus demonstrates a bradycardic response to hypoxia that lowers the metabolic needs of the heart. Failure or exhaustion of compensatory mechanisms that might otherwise ensure fetal oxygen delivery may result in lactic acidemia, cardiac dysfunction, and acidosis.[78]

Conclusion

The physiology of labor is complex, involving hormonal, mechanical, and biochemical events that lead to birth. Endocrine and anatomic changes involving the maternal and fetal systems contribute to the evolution of the phases of labor, including the premonitory signs and symptoms that precede labor and the activation and stimulation phases of uterine activity. Knowledge of the physiology that underlies labor and birth, including a healthy respect for individual normal variations, assures that midwives are in an excellent position to support women during this unique period in their lives.

Resource

Organization	Description	Webpage
Global Library of Women's Medicine (GLOWM)	López-Zeno J, 2008; doi: 10.3843/GLOWM.10126 A free international resource for medical professionals that includes monographs on safe motherhood; this chapter reviews the physiology of labor.	http://www.glowm.com/volume_content/item/2/recordset/64515/value/2

References

1. Mayberry LJ, Avery MD, Budin W, Perry S. Improving maternal and infant outcomes by promoting normal physiologic bir on hospital birthing units. *Nurs Outlook*. 2017;65:240-241.
2. Buckley SJ. *Hormonal Physiology of Childbearing: Evidence and Implications for Women, Babies, and Maternity Care*. Washington, DC: Childbirth Connection Programs, National Partnership for Women & Families; January 2015. Available at: http://www.nationalpartnership.org/research-library/maternal-health/hormonal-physiology-of-childbearing.pdf. Accessed July 31, 2017.
3. American College of Obstetricians and Gynecologists. Committee Opinion No. 687: summary: approaches to limit intervention during labor and birth. *Obstet Gynecol*. 2017;129(2):403-404.
4. Word RA, Li XH, Hnat M, Carrick K. Dynamics of cervical remodeling during pregnancy and parturition: mechanisms an current concepts. *Semin Reprod Med*. 25(1):69-79.
5. Baños A, Wolf M, Grawe C, et al. Frequency domain near-infrared spectroscopy of the uterine cervix during cervical ripening. *Laser Surg Med*. 2007;39(8):641-646.
6. Timmons B, Akins M, Mahendroo M. Cervical remodeling during pregnancy and parturition. *Trends Endocrinol Metab*. 2010;21(6):353-361.
7. Geng J, Huang C, Jiang S. Roles and regulation of the matrix metalloproteinase system in parturition. *Molec Reprod Develop*. 2016;83(4):276-286.
8. Norwitz ER, Robinson JN, Challis JR. The control of labor. *N Engl J Med*. 1999;341:660
9. Liao JB, Buhimschi CS, Norwitz ER. Normal labor: mechanism and duration. *Obstet Gynecol Clin North Am*. 2005;32(2):145-164.
10. York TP, Eaves LI, Neale MC, Straus JF. The contribution of genetic and environmental factors to the duration of pregnancy. *Am J Obstet Gynecol*. 2014;210(5):398-405.
11. York TP, Strauss JF, Neale MC, Eaves LJ. Racial differences in genetic and environmental risk to preterm birth. Lucia A, ed. *PLoS One*. 2010;5(8):e12391.
12. Wu W, Witherspoon DJ, Fraser A, et al. The heritability of gestational age in a two-million member cohort: implications for spontaneous preterm birth. *Hum Genet*. 2015;134(7):803-808.
13. Esplin MS, O'Brien E, Fraser A, et al. Estimating recurrence of spontaneous preterm delivery. *Obstet Gynecol*. 2008;112(3):516-523.
14. Roberts CT. IFPA Award in Placentology Lecture: complicated interactions between genes and the environment in placentation, pregnancy outcome and long term health. *Placenta*. 2010;31(suppl):S47-S53.
15. Latendresse G. The interaction between chronic stress and pregnancy: preterm birth from a biobehavioral perspective. *J Midwifery Womens Health*. 2009;54(1):8-17.
16. Monangi NK, Brockway HM, House M, Zhang G, Muglia LJ. The genetics of preterm birth: progress and promise. *Semin Perinatol*. 2015;39(8):574-583.
17. Pařízek A, Koucký M, Duškovβ M. Progesterone, inflammation and preterm labor. *J Steroid Biochem Molec Biol*. 2014;139:159-165.
18. Jones CW, Gambala C, Esteves KC, et al. Differences in placental telomere length suggest a link between racial disparitie in birth outcomes and cellular aging. *Am J Obstet Gynecol*. 2017;216(3):294.e1-294.e8.
19. Mendelson CR. Minireview: fetal–maternal hormonal signaling in pregnancy and labor. *Molec Endocrinol*. 2009;23(7):947-954.
20. Sfakianaki AK, Norwitz ER. Mechanisms of progesterone action in inhibiting prematurity. *J Matern Fetal Neonatal Med* 2006;19(12):763-772.
21. Challis RJG, Gibb W. Control of parturition. *Prenat Neonat Med*. 1996:1;238.
22. Shynlova O, Tsui P, Jaffer S, Lye SJ. Integration of endocrine and mechanical signals in the regulation of myometrial functions during pregnancy and labour. *Eur J Obstet Gynecol Reprod Biol*. 2009;144(suppl 1):S2-S10.
23. Tan H, Yi L, Rote NS, Hurd WW, Mesiano S. Progesterone receptor-A and -B have opposite effects on proinflammatory gene expression in human myometrial cells: implications for progesterone actions in human pregnancy and parturition. *J Clin Endocrinol Metab*. 2012;97(5):E719-E730.
24. Kamel RM. The onset of human parturition. *Arch Gynecol Obstet*. 2010;281(6):975-982.
25. Zakar T, Hertelendy F. Progesterone withdrawal: key to parturition. *Am J Obstet Gynecol*. 2007;196(4):289-296.
26. Vidaeff AC, Ramin SM. Potential biochemical events associated with initiation of labor. *Curr Med Chem*. 2008;15(6):614-619.
27. Chai SY, Smith R, Zakar T, Mitchell C, Madsen G. Term myometrium is characterized by increased activating epigenetic modifications at the progesterone receptor-A promoter. *Molec Hum Reprod*. 2012;18(8):401-409.
28. Arulkumaran S, Kandola MK, Hoffman B, Hanyaloglu AC, Johnson MR, Bennett PR. The roles of prostaglandin EP 1 an 3 receptors in the control of human myometrial contractility. *J Clin Endocrinol Metab*. 2012;97(2):489-498.
29. Weiss G. Endocrinology of parturition. *J Clin Endocrinol Metab*. 2000;85(12):4421-4425.

30. Golightly E, Jabbour HN, Norman JE. Endocrine immune interactions in human parturition. *Molec Cell Endocrinol*. 2011;335(1):52-59.
31. Smith R. Parturition. *N Engl J Med*. 2007;356:271-283.
32. You X, Gao L, Liu J, et al. CRH activation of different signaling pathways results in differential calcium signaling in humar pregnant myometrium before and during labor. *J Clin Endocrinol Metab*. 2012;97(10):E1851-E1861.
33. Osman I, Young A, Ledingham MA, et al. Leukocyte density and pro-inflammatory cytokine expression in human fetal membranes, decidua, cervix and myometrium before and during labour at term. *Molec Hum Reprod*. 2003;9(1):41-45.
34. Blanks AM, Thornton S. The role of oxytocin in parturition. *BJOG*. 2003;110(suppl 20):46-51.
35. Blanks AM, Shmygol A, Thornton S. Regulation of oxytocin receptors and oxytocin receptor signaling. *Semin Reprod Med*. 2007;25(1):52-59.
36. Terzidou V, Blanks AM, Kim SH, Thornton S, Bennett PR. Labor and inflammation increase the expression of oxytocin receptor in human amnion. *Biol Reprod*. 2011;84(3):546-552.
37. Kim SH, Bennett PR, Terzidou V. Advances in the role of oxytocin receptors in human parturition. *Molec Cell Endocrinol*. 2017;449:56-63.
38. Dawood MY, Raghavan KS, Pociask C, Fuchs F. Oxytocin in human pregnancy and parturition. *Obstet Gynecol*. 1978;51(2):138-143.
39. Terzidou V, Sooranna SR, Kim LU, Thornton S, Bennett PR, Johnson MR. Mechanical stretch up-regulates the human oxytocin receptor in primary human uterine myocytes. *J Clin Endocrinol Metab*. 2005;90(1):237-246.
40. Phaneuf S, Rodriguez Linares B, TambyRaja RL, MacKenzie IZ, Lopez Bernal A. Loss of myometrial oxytocin receptors during oxytocin-induced and oxytocin-augmented labour. *J Reprod Fertil*. 2000;120(1):91-97.
41. Chiossi G, Costantine MM, Bytautiene E, et al. The effects of prostaglandin E_1 and prostaglandin E_2 on in vitro myometria contractility and uterine structure. *Am J Perinatol*. 2012;29(8):615-622.
42. Cunningham F, Leveno KJ, Bloom SL, et al. Physiology of labor. In: Cunningham F, Leveno KJ, Bloom SL, et al., eds. *Williams Obstetrics*. 24th ed. New York, NY: McGraw-Hill; 2014:408-432.
43. Lowe N, Openshaw M, King TL. Labor. In: Brucker MC, King TL, eds. *Pharmacology for Women's Health*. 2nd ed. Burlington, MA: Jones & Bartlett Learning; 2017:1065-1094.
44. Ozuounian JG, Elkayam U. Physiologic changes during normal pregnancy and delivery. *Cardiol Clin*. 2012;30:317-332.
45. Fujitani S, Baldisseri MR. Hemodynamic assessment in a pregnant and peripartum patient. *Crit Care Med*. 2005;33(10 suppl):S354-S361.
46. Soma-Pillay P, Nelson-Piercy C, Tolppanen H, Mebazaa A. Physiological changes in pregnancy. *Cardiovasc J Afr*. 2016;27(2):89-94.
47. Konje JC, Howarth ES, Kaufmann P, Taylor DJ. Longitudinal quantification of uterine artery blood volume flow changes during gestation in pregnancies complicated by intrauterine growth restriction. *BJOG*. 2003;100:301-305.
48. Flo K, Wolsgaard T, Vartun A, Acharya G. A longitudinal study of the relationship between maternal cardiac output measured by impedance cardiography and uterine artery blood flow in the second half of pregnancy. *BJOG*. 2010;117:837-844.
49. Capeless EL, Clapp JF. When do cardiovascular parameters return to their preconception values? *Am J Obstet Gynecol*. 1991;165:883.
50. Tripodi A. d-Dimer testing in laboratory practice. *Clin Chem*. 2011;57(9):1256-1262.
51. James AH, McLintock C, Lockhart E. Postpartum hemorrhage: when uterotonics and sutures fail. *Am J Hematol*. 2012;87(suppl 1):S16-S22.
52. McLintock C, James AH. Obstetric hemorrhage. *J Thromb Haemost*. 2011;9(8):1441-1451.
53. Chang J, Streitman D. Physiologic adaptations to pregnancy. *Neurol Clin*. 2012;30(3):781-789.
54. Kardel KR, Henriksen T, Iversen PO. No effect of energy supply during childbirth on delivery outcomes in nulliparous women: a randomised, double-blind, placebo-controlled trial. *J Obstet Gynaecol*. 2010;30(3):248-252.
55. Banerjee B, Khew KS, Saha N, Ratnam SS. Energy cost and blood sugar level during different stages of labour and duration of labour in Asiatic women. *J Obstet Gynaecol Br Commonw*. 1971;78(10):927-929.
56. Hagerdal M, Morgan CW, Sumner AE, Gutsche BB. Minute ventilation and oxygen consumption during labor with epidura analgesia. *Anesthesiology*. 1983;59(5):425-427.
57. Maganha e Melo CR, Peracoli JC. Measuring the energy spent by parturient women in fasting and in ingesting caloric replacement (honey). *Rev Lat Am Enfermagem*. 2007;15(4):612-617.
58. Sperling JD, Dahlke JD, Sibai BM. Restriction of oral intake during labor: whither are we bound? *Am J Obstet Gynecol*. 2016;214(5):592-596.
59. O'Sullivan G, Liu B, Hart D, Seed P, Shennan A. Effect of food intake during labour on obstetric outcome: randomised controlled trial. *BMJ*. 2009;338:b784.
60. Singata M, Tranmer J, Gyte GML. Restricting oral fluid and food intake during labour. *Cochrane Database Syst Rev*. 2013;8:CD003930. doi:10.1002/14651858.CD003930.pub3.
61. Ciardulli A, Saccone G, Anastasio H, Berghella V. Less restrictive food intake during labor in low-risk singleton

pregnancies: a systematic review and meta-analysis. *Obstet Gynecol*. 2017;129(3):473-478.
62. Lowe NK. The nature of labor pain. *Am J Obstet Gynecol*. 2002;186(5 suppl Nature):S16-S24.
63. McCool WF, Smith T, Aberg C. Pain in women's health: a multi-faceted approach toward understanding. *J Midwifery Womens Health*. 2004;49(6):473-481.
64. Alder J, Breitinger G, Granado C, et al. Antenatal psychobiological predictors of psychological response to childbirth. *J Am Psychiatr Nurs Assoc*. 2011;17(6):417-425.
65. Lally JE, Murtagh MJ, Macphail S, Thomson R. More in hope than expectation: a systematic review of women's expectations and experience of pain relief in labour. *BMC Med*. 2008;6:7. doi:10.1186/1741-7015-6-7.
66. Choi KR, Seng JS. Predisposing and precipitating factors for dissociation during labor in a cohort study of posttraumatic stress disorder and childbearing outcomes. *J Midwifery Womens Health*. 2016;61(1):68-76.
67. Haines HM, Rubertsson C, Pallant JF, Hildingsson I. The influence of women's fear, attitudes and beliefs of childbirth on mode and experience of birth. *BMC Pregn Childbirth*. 2012;12(1):55.
68. Bateman L, Jones C, Jomeen J. A narrative synthesis of women's out-of-body experiences during childbirth. *J Midwifery Womens Health*. 2017;62(4):442-451.
69. Gibson E. Women's expectations and experiences with labour pain in medical and midwifery models of birth in the United States. *Women Birth*. 2014;27(3):185-189.
70. Jokic´-Begic´ N, Zigic´ L, Nakic´ Radoš S. Anxiety and anxiety sensitivity as predictors of fear of childbirth: different patterns for nulliparous and parous women. *J Psychosom Obstet Gynaecol*. 2014;35(1):22-28.
71. LoGiudice JA. A systematic literature review of the childbearing cycle as experienced by survivors of sexual abuse. *Nurs Womens Health*. 2016-2017;20(6):582-594.
72. Hodnett ED, Gates S, Hofmeyr GJ, Sakala C. Continuous support for women during childbirth. *Cochrane Database Syst Rev*. 2013;7:CD003766. doi:10.1002/14651858.CD003766.pub5.
73. Simkin P, Bolding A. Update on nonpharmacologic approaches to relieve labor pain and prevent suffering. *J Midwifery Womens Health*. 2004;49(6):489-504.
74. Trout KK. The neuromatrix theory of pain: implications for selected nonpharmacologic methods of pain relief for labor *[erratum, J Midwifery Womens Health. 2005;50(1)]*. *J Midwifery Womens Health*. 2004;49(6):482-488.
75. Jones L, Othman M, Dowswell T, et al. Pain management for women in labour: an overview of systematic reviews. *Cochrane Database Syst Rev*. 2012;3:CD009234. doi:10.1002/14651858.CD009234.pub2.
76. Smith CA, Levett KM, Collins CT, Crowther CA. Relaxation techniques for pain management in labour. *Cochrane Database Syst Rev*. 2011;12(12):CD009514. doi:10.1002/14651858.CD009514.
77. Peebles DM, Spencer JAD, Edwards AD, et al. Relation between frequency of uterine contractions and human fetal cerebral oxygen saturation studied during labour by near infrared spectroscopy. *Br J Obstet Gynecol*. 1994;101:44-48.
78. Bakker PCAM, Kuver PHJ, Kuik DJ, Va Geijn HP. Elevated uterine activity increases the risk of fetal acidosis at birth. *Am J Obstet Gynecol*. 2007;196(313):e1-313e6.

CHAPTER

25

First Stage of Labor

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Introduction

A normal physiologic labor and birth is one that is powered by the innate human capacity of the woman and fetus.[1] Normal labor and birth do not require medical intervention, yet most women in the United States experience childbirth in hospitals, where they labor in bed with intravenous fluids and their fetuses are monitored with continuous electronic fetal heart rate monitoring. Many individuals who have a hospital birth receive synthetic oxytocin (Pitocin) for induction or augmentation. Despite recognition that unnecessary intervention is pervasive in clinical practice, until recently there has been little guidance for eliminating commonly used interventions that are not needed and may, in fact impede, normal labor.

Normal Physiologic Childbirth

There is now a burgeoning movement to recharacterize normal physiologic labor. In 2012, the American College of Nurse-Midwives (ACNM), Midwives of North America (MANA), and National Association of Certified Professional Midwives (NACPM) published a consensus document titled *Supporting Healthy and Normal Physiologic Childbirth: A Consensus Statement by ACNM, MANA and NACPM*.[1] This document defines normal physiologic childbirth and describes essential components and factors that disrupt normal physiologic childbirth (**Table 25-1**). In 2017, the American College of Obstetricians and Gynecologists Committee on Obstetrical Practice released an opinion, *Approaches to Limit Intervention During Labor and Birth*,[2] that has been endorsed by ACNM. This document contains recommendations for obstetrician-gynecologists to support women during labor through use of practices associated with minimal interventions and high patient satisfaction, which is consistent with midwifery practice. Similar documents and resources from other professional associations can be found in the Resources section at the end of this chapter.

Table 25-1	Normal Physiologic Birth
Characteristics of Normal Physiologic Childbirth	
Involves spontaneous onset and progression of labor	
Includes biological and psychological conditions that promote effective labor	
Results in the vaginal birth of the infant and placenta	
Results in physiological blood loss	
Facilitates optimal newborn transition through skin-to-skin contact and keeping the mother and infant together during the postpartum period	
Supports early initiation of breastfeeding	
Factors That Disrupt Normal Physiologic Childbirth	
Induction or augmentation of labor	
An unsupportive environment (e.g., bright lights, cold room, lack of privacy, multiple providers, lack of supportive companions)	
Time constraints, including those driven by institutional policy and/or staffing	
Nutritional deprivation (e.g., food and drink)	
Opioids, regional analgesia, or general anesthesia	
Episiotomy	
Operative vaginal (vacuum, forceps) or abdominal (cesarean) birth	
Immediate umbilical cord-clamping	
Separation of mother and infant	
Any situation in which the individual feels threatened or unsupported	
Factors That Influence Normal Physiologic Childbirth	
Multiple factors influence the ability of a woman to give birth without intervention	
For the Woman	
Her individual health status and physical fitness	
Autonomy and self-determination in childbirth	

Personal knowledge and confidence about birth, including cultural beliefs, norms, and practices and education about the value of normal physiologic birth
Fully informed, shared decision making
Access to healthcare systems, settings, and providers supportive of and skilled in normal physiologic birth
For the Clinician
Education, knowledge, competence, skill, and confidence in supporting physiologic labor and birth, including helping women cope with pain
Commitment to working with women through education to enhance their confidence in birth and diminish their fear of the process
Commitment to shared decision making
Working within an infrastructure supportive of normal physiologic birth

Reproduced with permission from American College of Nurse-Midwives, Midwives Alliance, and National Association of Certified Professional Midwives. Supporting healthy and normal physiologic childbirth: a consensus statement by ACNM, MANA, and NACPM. 2012. Available at: https://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000272/Physiological%20Birth%2 %20FINAL%20May%2018%202012%20FINAL.pdf.[1]

This chapter presents the evidence for the recommendations in the *Supporting Healthy and Normal Physiologic Childbirth* consensus statement and reviews care for essentially healthy women during spontaneous labor.[1] This document provides a roadmap to optimal care for women during labor and birth and, as the U.S. maternity care system begins to step back from the current overuse of unnecessary interventions, the skills and techniques developed in midwifery practice are being recognized as a critical component of this roadmap. Care for women who require induction of labor and those who have medical or obstetric complications is presented in the *Complications During Labor and Birth* chapter.

Definitions of Labor

Labor is the process by which childbirth occurs, requiring uterine contractions of sufficient frequency, duration, and intensity to cause effacement and dilation of the cervix. The mechanisms underlying the onset and progress of spontaneous labor remain largely unknown and labor is a continuum that does not readily lend itself to measurement. Not only is prospectively defining the onset of labor a significant challenge, but the ability to evaluate its progression is limited because cervical examinations are performed only episodically, if at all. Attempts to divide the continuum of labor into stages and phases add to the complexity. In spite of measurement difficulties, understanding and supporting normal first-stage labor allows time for the events of physiologic labor to unfold without unnecessary interventions, while simultaneously observing the woman for situations in which interventions may be needed. Only in this way can safe, state-of-the-science midwifery care be provided.

The *first stage of labor* begins with true labor contractions, as evidenced by progressive cervical change, and ends when the cervix is completely dilated (approximated at 10 centimeters) and ready for passage of the fetal presenting part. It is known as the stage of cervical dilatation. The *second stage of labor*, known as the expulsive stage, follows. The first stage of labor was classically divided into three sequential, albeit rather nebulously defined phases: latent, active, and deceleration. The *latent phase* begins with the onset of regular uterine labor contractions; the *active phase* begins when the rate of cervical dilation increases. A third *deceleration stage* wherein labor progress slows between 8 and 10 centimeters of cervical dilation was identified by Dr. Emanual Friedman in studies conducted in the 1960's but the reality of a deceleration phase is controversial as reviewed later in this chapter. The second stage of labor begins with complete cervical dilatation and ends with the birth of the infant. The *third stage of labor* is the time between birth of the infant and delivery of the placenta. Abnormalities of labor progress are reviewed in the *Complications During Labor and Birth* chapter.

Each stage and phase of the labor is characterized by distinct physical changes and behavioral responses that are distinct. The physical changes—in particular, cervical dilation and fetal station—are used to evaluate labor progress. The woman's behaviors may be used to help estimate labor progress without resorting to a cervical examination, although individual and cultural responses to labor vary significantly. Thus, the midwife needs to know the woman, her goals, expectations, and concerns as well as her cultural framework to assess labor progress via evaluation of her expression and behaviors. Although interpretations of nonverbal behaviors are a common component of midwifery assessment of persons in active labor, individual's express their experiences in markedly varied ways and healthcare provider's implicit biases frequently result in misinterpretation. Thus, a midwife's interpretation of nonverbal behaviors should always be verified with the individual.

Premonitory Signs and Symptoms of Labor Onset

The onset of spontaneous labor cannot be reliably predicted, although many pregnant women experience premonitory signs or symptoms of impending labor. Common signs and symptoms suggestive of physiologic progress toward labor include descent of the fetus, cervical changes, increase in uncoordinated uterine contractions, rupture of membranes, bloody show or increased mucus discharge from the vagina, maternal perception of increased energy, and gastrointestinal distress. Anticipatory guidance for all women in late pregnancy includes reassurance that these signs and symptoms are normal, promotion of comfort measures, and support for watchful waiting.

Descent of the Fetus

Notable descent of the fetus prior to labor, or "lightening," occurs following the gradual softening of the uterine isthmus (lower uterine segment). The fetal presenting part, most commonly the head, descends to or through the inlet of the maternal pelvis (**Figure 25-1**). Although lightening is not synonymous with engagement of the fetal presenting part (i.e., when the widest diameter of the fetal presenting part is at or below the maternal pelvic inlet), engagement may be the result of lightening. A small reduction in fundal height is common following this prelabor fetal descent. Leopold's maneuvers will reveal that the presenting part is no longer ballottable above the symphysis pubis. Cervical examination typically finds the presenting part as "fixed" and not ballottable. Women often subjectively refer to the physical experience of lightening as "the baby has dropped."

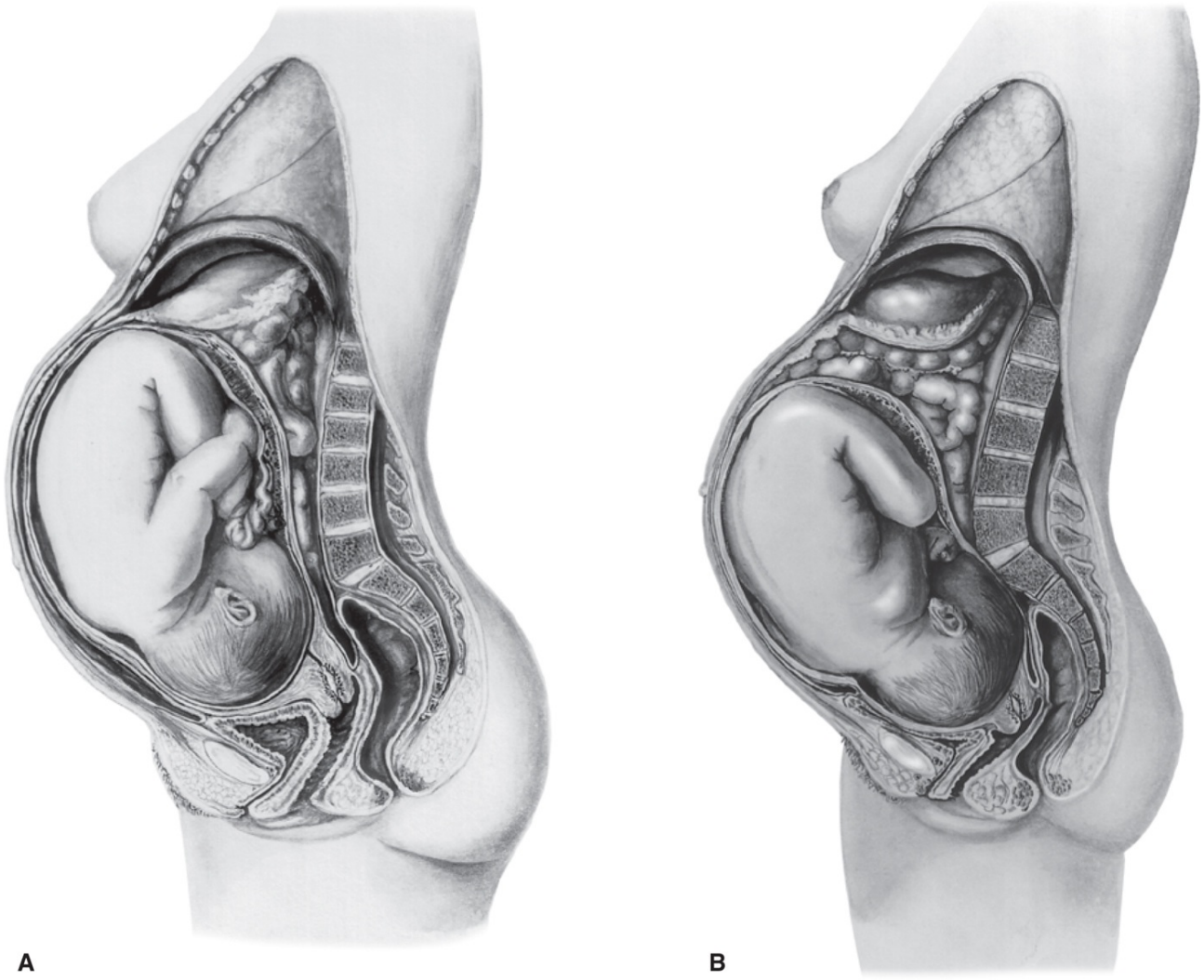


Figure 25-1 **A.** Prelightening. **B.** Postlightening.

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Among many, but not all, nulliparous women, prelabor descent of the fetus may occur two or more weeks in advance of labor and is reassuring for pelvic adequacy at the plane of the inlet. For women who have previously given birth, notable descent may not occur until labor is advanced.

The fetal descent associated with lightening can result in women having more room in the upper abdomen, allowing for better lung expansion and increased stomach capacity. However, lightening often causes new discomforts because of the pressure imposed by the presenting part on other structures in the area of the true pelvis. Urinary frequency can be caused by compression of the bladder between the fetal presenting part and the maternal pelvis. An increase in generalized pelvic pressure contributes to the straddling walk of late pregnancy, and women may relate a constant feeling of pelvic pressure. Increased venous stasis and pelvic congestion can cause more dependent edema, development of hemorrhoids, and vulvar or lower extremity varicosities. Spending some time throughout the day in the knee–chest or left side-lying position may temporarily relieve the discomforts related to generalized pressure and venous stasis.

Finally, sciatic nerve pain, characterized by unilateral shooting pain radiating from the buttocks down the back of the leg, can be aggravated or initiated by the fetal descent. This sciatic pain is self-limited to pregnancy and may be relieved by massage, application of heat/cold, or gentle stretching exercises. Sciatic nerve pain that is debilitating or persists into the postpartum period warrants a referral of the woman for evaluation, as these symptoms may be indicative of vertebral disc herniation.

Cervical Changes

The process of cervical change preparatory to labor is referred to as "ripening." The cervix, which is typically long, closed, and semi-firm throughout pregnancy, becomes soft and malleable during this process. Effacement, dilation, and anterior rotation of the cervix may be noted. While ripening is a reassuring change suggestive of progress toward true labor, it is not a reliable predictor of actual labor onset. Evaluation of ripeness is relative to the individual woman and her parity. For example, during the final weeks of pregnancy, a multiparous woman's cervix may be soft and surprisingly dilated; in contrast, a nulliparous woman's cervix may remain firm and closed until near the onset of labor.

Cervical ripening occurs secondary to the influence of prostaglandin, a hormone that acts locally in a paracrine manner to create changes in collagen matrix and increased extracellular water content. Cervical ripening may occur with or without uterine contractions. Unless such an evaluation is being used to inform clinical decision making, routine assessment of cervical ripeness via digital examination in late pregnancy is unnecessary because it is not predictive of labor onset and may be uncomfortable for the woman.

Prelabor or False Labor

"False labor" is the term historically used to describe the experience of uterine contractions of varying intensity that are not associated with progressive cervical change over time. These contractions are actually an intensification of the usually painless Braxton Hicks contractions, which may first become noticeable in the third trimester; thus, the term "false labor" is not accurate. These prelabor contractions do not intensify over time and may be relieved by walking or position changes, whereas true labor contractions will intensify over time, becoming longer, stronger, and closer together. True labor contractions sometimes intensify with walking and are not relieved by position changes. Most importantly, true labor is associated with progressive cervical change. Use of the term "prelabor contractions" in lieu of "false labor" may help women better understand their discomfort and is a more body-affirming term.

Prelabor contractions may occur for days or intermittently for weeks before the onset of true labor. A woman experiencing persistent or recurrent prelabor contractions may fatigue over time and have difficulty coping, as these contractions can be painful and distressing. Provider assurance that the woman will be able to identify a change in the labor pattern indicating true labor and reinforcement of education regarding true labor characteristics and other signs of impending labor can help the woman cope with prelabor contractions. Comfort measures for prelabor contractions are the same as those used to support women in latent-phase labor.

Bloody Show

A mucus plug, created by cervical secretions from proliferation of the glands of the cervical mucosa early in pregnancy, seals the cervical canal throughout pregnancy, serving as a protective barrier. As the cervix ripens, small capillaries may break, mixing blood with the mucus. Bloody show is the expulsion of this blood-tinged mucus.

The mucus associated with bloody show is thick and tenacious. Both history and examination can help differentiate benign bloody show from frank bleeding. Occasionally the entire mucus plug is expelled en masse. More commonly, the mucus plug is expelled over the course of several days, and noticed by the woman as mild irregular spotting when wiping after urination or defecation.

As a predictive sign of labor, bloody show usually signals the onset of labor within the next few days. However, it is of no value as a sign of labor if a vaginal examination or sexual intercourse has recently occurred, because blood-tinged mucus discharge during this time may be the effect of minor trauma to the ripening cervix or disruption of the mucus plug.

Energy Spurt

Some women perceive an energy spurt in the days preceding labor onset, the cause of which is unknown. Women may benefit by anticipatory guidance that includes a caution against "overdoing it."

Gastrointestinal Distress

In the absence of any other causative factors, a brief occurrence of diarrhea, indigestion, nausea, and vomiting is thought to be indicative of impending labor. These gastrointestinal distress symptoms are likely associated with the increased production of prostaglandins and estrogen and the decrease in progesterone levels that occur with the approach of labor.

Prelabor Rupture of Membranes

Rupture of the fetal membranes with associated loss of amniotic fluid prior to the onset of labor is called prelabor rupture of membranes (PROM). PROM occurs in approximately 8% to 10% of women with term pregnancies, 95% of whom will begin labor spontaneously within 33 to 107 hours after rupture, if given this time.[2-4] Because the duration of ruptured membranes before onset of labor can be a short or long period of time, women who experience PROM at term may either wait (expectant management) for the onset of labor or consider immediate induction of labor. Recommended management varies according to several factors described in more detail in the *Complications During Labor and Birth* chapter.

The ACNM, in a position statement on PROM at term,[3] affirmed that women with PROM should receive counseling and engage in shared decision making based on full knowledge of the risks and benefits of the various management options. For women at term, expectant management for a period of time is a safe alternative to induction of labor under the following

conditions: uncomplicated pregnancy, singleton fetus in vertex presentation with clear amniotic fluid; absence of identified infection, including group B *Streptococcus* (GBS) colonization, hepatitis B, hepatitis C, or human immunodeficiency virus (HIV) infection; absence of fever; no variant fetal heart rate patterns that suggest an increased risk for fetal acidemia; and no other indications for induction of labor. Expectant management requires avoiding all digital cervical examinations, including avoidance of a baseline cervical examination.[3] Leopold's maneuvers or ultrasound may be used to ascertain the fetal presentation in lieu of a digital examination in these cases.

A key consideration in the woman experiencing PROM is whether her vagina is colonized with GBS. Vaginal colonization with GBS is a nonpathologic normal condition for many women, but this bacterium can be transmitted to the fetus during passage through the vagina at birth and infect the newborn, causing sepsis. Thus, a woman's status as a GBS carrier may affect decisions related to expectant management, antibiotic prophylaxis, and recommendations for induction. The Centers for Disease Control and Prevention (CDC) has guidelines for GBS screening and management, including recommendations for antibiotic prophylaxis in the presence of PROM when the woman's GBS status is positive or unknown.[5] The CDC management algorithms are endorsed by all professional associations and used in almost all institutions in the United States. The American College of Obstetricians and Gynecologists recommends that women with term PROM who are GBS positive receive antibiotics for GBS prophylaxis immediately, even if expectant management is the chosen plan for care.[2] Institutional guidelines and recommendations should be supported by written instructions and included in the anticipatory guidance offered to all women approaching term.

The steps for diagnosing PROM are listed in **Appendix 25A**. Accurate diagnosis of rupture of membranes can sometimes be difficult. The woman's perceptions of the event, careful physical examination, and laboratory testing can all contribute to confirming the diagnosis. Laboratory tests that may be used to establish the status of the fetal membranes include the arborization test (fern test), pH test (nitrazine test), ultrasound quantification of amniotic fluid volume, and test for the presence of placental alpha microglobulin-1 (PAMG-1)[6] or fetal fibronectin in vaginal secretions. Differential diagnoses that need to be excluded include urinary incontinence, vaginal or cervical discharge, semen, and (rarely) rupture of the chorion alone.

The Normal First Stage of Labor

The rate of cervical dilation in the first stage of labor, expressed in centimeters per hour (cm/hr), is the backbone of decision making for clinicians providing care to laboring women. Abnormalities in expected durations of the latent, active, and second stage of labor are associated with perinatal morbidities such as intra-amniotic infection, cephalopelvic disproportion, cesarean section, and postpartum hemorrhage. Accuracy in determining cervical dilation depends on the skill of the examiner. Even among experienced professionals, however, the assessment may vary to some degree. There is no objective method to validate the measurements, and cervical dilation can change rapidly. Nevertheless, because cervical dilation rates are highly related to labor duration, measures of labor duration also inform the "normal" boundaries of first-stage labor progression.

Latent Phase

The latent phase encompasses the period of time from the beginning of regular uterine contractions to the point when cervical dilation begins to progress rapidly. Application of this definition is difficult at both ends of the spectrum. First, the onset of the latent phase is difficult to determine objectively, as women typically do not seek labor care until after the onset of labor. Thus, identification of latent-phase onset often depends on maternal memory and discernment of prelabor versus true labor contractions. Second, the end of the latent phase is difficult to determine due to the wide variability in the onset of rapid and progressive cervical dilation. In multiparous women, the rate of cervical dilation commonly accelerates after 6 cm dilation; however, the average labor curve for nulliparous women does not always have a clear inflection point of dilation acceleration that marks the end of the latent phase.[7]

Uterine contractions become established during the latent phase as they increase in frequency, duration, and intensity. It is common for contractions during latent labor to initially be of mild intensity, occurring every 10 to 20 minutes and lasting 15 to 20 seconds. As latent labor advances, contractions typically increase to moderate intensity, occurring approximately every 5 to 7 minutes and lasting 30 to 40 seconds. These parameters alone do not define latent labor, however, as the experience of contractions may vary greatly among women at similar points of the labor process. Only demonstrable effacement and dilation of the cervix truly define labor. Little to no descent of the presenting part occurs during the latent phase.

A woman in latent labor typically experiences a mixture of emotions: She may be excited, happy, and relieved that the end of pregnancy has come and the long period of waiting has ended, but simultaneously feel a sense of anticipation and apprehension about what is yet to come. Many women cope well with this situation. For some women, however, the latent phase of labor may be a time of fear and increased sensitivity to pain and external stimuli. This is the ideal time for the midwife to assess coping skills, establish rapport, and develop an initial management plan.

Active Phase of Labor

The *active phase of labor* encompasses the period of time that starts with an increase in the rate of cervical dilation (the end of the latent phase of labor) and ends with complete cervical dilation (the beginning of the second stage of labor). Progressive descent of the fetal presenting part also typically occurs during the latter part of the active phase and in the second stage of labor. Accurately diagnosing active labor is of utmost importance because a woman's rate of cervical dilation in this phase serves as the basis against which her labor progress is assessed, admission decisions are made, and the need for intervention is determined. It is critical that expectations of cervical dilation during active labor be appropriately defined based on the most current research.

Unfortunately, true active labor can never be diagnosed prospectively, which is limiting to clinicians; rather, it can be determined only retrospectively based on an assessment of adequate cervical dilation over time. Both the time intervals for cervical dilation rates and cervical dilation at the onset of the active phase—factors used for the last several decades by clinicians in practice to guide management—have been recently reevaluated for their validity in assessing progression of labor.

Friedman Studies

Clinical expectations of cervical dilation and fetal descent during labor continue to be influenced by the research of Emanuel Friedman, a physician whose work began in the 1950s.[8-13] Friedman observed women during labor, recorded progress, and plotted these recordings to depict the progress of labor as a sigmoid (S-shaped) curve, varying in duration and slope between women who were nulliparous versus multiparous. Active labor, most often beginning between 2 and 3 cm, was divided into three sequential subphases: an *acceleration phase*, a *phase of maximum slope*, and a *deceleration phase*. Friedman reported that the active phase averaged between 4.4 and 4.9 hours for nulliparous women[8,9,12,13] and had an upper "normal" limit of 11.7 hours (mean + 2 standard deviations [SD]).[9,12,13] For multiparous women, the active phase averaged between 2.2 and 2.4 hours,[10-13] with a statistical upper normal limit of 5.2 hours.[10,13] Regardless of parity, approximately half of the total active phase time for the aggregate groups was spent in the acceleration phase, when little progress in dilation is made. Cervical dilation rates in Friedman's phase of maximum slope for nulliparous and multiparous women at the mean were 3.0 cm/hour and 5.7 cm/hour, respectively; the slowest acceptable rates (i.e., mean – 2 SD) were reported to be 1.2 cm/hour and 1.5 cm/hour, for nulliparous and multiparous women, respectively.[9,10,12,13]

Using a subset of women who had a fetus in vertex presentation, spontaneous labor, and no use of medications, Friedman constructed an idealized labor curve. This idealized labor curve has been used extensively to monitor labor progress and diagnose labor progress abnormalities. Unfortunately, these aggregate active-phase dilation rate estimates are of limited prospective use for individual women, because the cervical dilation at which active labor begins varies widely among women and cervical dilation is affected by multiple intrinsic and extrinsic factors in different populations of women. Differences based on these factors have recently been identified.

There are many differences in practice patterns between the 1950s and labor management

practices today. Today women frequently use epidural analgesia to manage labor pain, whereas in the 1950s women received sedatives and morphine to manage this pain. In Friedman's era, if a woman had not given birth spontaneously after 2 hours in the second stage of labor, this stage was terminated at 2 hours via performance of an episiotomy and forceps delivery. Thus, it is worthwhile to review labor duration studies that have included contemporary labor practices and current populations of women who reside in the United States.

Contemporary Studies of Labor Progress

Many midwives use criteria historically associated with Friedman's work to determine the diagnosis of active labor, which is suggested to begin with cervical dilatation between 3 cm and 5 cm, in the presence of regular uterine contractions.[14] However, true active labor onset for many women with spontaneous onset of labor may not be present at 3 to 5 cm of cervical dilation. For example, Peisner and Rosen found that 75%, 50%, and 24% of low-risk women with regular contractions admitted for spontaneous labor (n = 1699) at 3, 4, and 5 cm, respectively, did not experience cervical dilation at rates indicative of active labor, as traditionally defined by Friedman.[15] Such findings highlight two important realities of contemporary clinical practice: (1) Many women are presumed to be in active-phase labor and managed as such, before they are actually in active labor, and (2) guidelines on expected rates of cervical dilation during traditionally defined active-phase labor progress times (1 cm/hr) are overly stringent. In addition, in situations where multiple professionals perform cervical examinations, variations in inter-rater reliability may raise doubt in accuracy of cervical assessments.

In 1989, Kilpatrick and Laros reported that nulliparous (n = 2302) and multiparous women (n = 3767) who gave birth spontaneously without the use of oxytocin or anesthesia required a mean of 8.1 ± 4.3 hours and 5.7 ± 3.4 hours, respectively, to progress from the onset of regular, painful contractions occurring every 3 to 5 minutes and leading to cervical change, to 10 cm or complete dilation.[16] When these authors compared first- and second-stage labor times for women with and without epidural analgesia, they found that the first stage of labor is longer by approximately 2 hours in women who have epidural analgesia.[16] The clinical implications of these findings with regard to second-stage labor duration are reviewed in the *Second Stage of Labor and Birth* chapter.

Zhang et al., using modern statistical interval measurement techniques (as opposed to the simple mathematical means modeling used by Friedman decades ago) analyzed data from the National Collaboration Perinatal Project (NCPP), a large, multicenter study conducted from 1959 to 1966 including 54,390 births experienced by 48,197 women.[17,18] The NCPP data set detailed the labors of women giving birth during the time when Friedman's labor curves were introduced. The different mathematical model used by Zhang et al. found important differences from Friedman's work, including that the active phase of labor may not start, on average, until cervical dilation reaches 5 cm in multiparous women or even later in nulliparous women.[17] Moreover, the labor curve was hyperbolic in shape, with dilation becoming more rapid as labor advanced and without the deceleration phase reported by Friedman. The median time to

dilate from 4 to 10 cm was 3.7 hours (16.7 hours at the 95th percentile) and 2.2 hours (14.2 hours at the 95th percentile) for nulliparous and multiparous women, respectively.[17]

Collectively, the studies reported by Zhang et al. provide compelling evidence about the duration of first-stage labor and the pattern of labor progression among women with spontaneous labor onset.[7,18,19] In these authors' initial 2002 study that included nulliparous women (*n* = 1162), median dilation rates for each centimeter of progression between 3 and 10 cm were 0.4, 0.6, 1.2, 1.7, 2.2, 2.4, and 2.4 cm/hour, respectively.[19] At the 5th percentile, these dilation rates were 0.1, 0.2, 0.3, 0.5, 0.7, 0.8, and 0.7 cm/hour, respectively, but never exceeded 1 cm/hour. Accordingly, the time needed to dilate from one centimeter to the next was commonly shorter with each passing centimeter after the spontaneous onset of labor for both nulliparous and multiparous women. Moreover, as rates of cervical dilation accelerated with advancing labor, the time necessary to dilate from one centimeter to the next was typically less variable.[19]

In perhaps their most influential studies, this research team used data from the Consortium on Safe Labor (CSL), a multicenter, retrospective, observational study that contains the labor and delivery records of 228,668 women who gave birth in different areas of the United States; 87% of these births occurred in 2005–2007.[7] Women included in this data set may have received care including interventions such as amniotomy, oxytocin augmentation, and epidural analgesia. Most importantly, the researchers measured the time interval between one cervical dilation and the next (i.e., how long it took to progress from 4 cm to 5 cm, and then the time interval between 5 cm and 6 cm). This approach contrasts with that used in previous studies, which usually calculated the mean time interval for a particular phase of labor (i.e., the active phase). Durations of the first stage of labor for women of different parity are shown in **Table 25-2**.

Table 25-2 **Duration of First-Stage Labor with Spontaneous Onset in Hours, by Parity, Beginning with Cervical Dilations Commonly Associated with Active Labor Onset**

Initial to Complete Dilatation	Parity 0 (n = 43,576)[a] Median (95%)	Parity 1[b] (n = 27,471)[a] Median (95%)	Parity 2+[b] (n = 27,312)[a] Median (95%)
4 cm to complete dilatation, hours	6.5 (24.0)	—	—
5 cm to complete dilatation, hours	3.6 (15.1)	3.0 (15.0)	2.8 (15.5)
6 cm to complete dilatation, hours	2.2 (10.0)	1.6 (8.9)	1.3 (8.9)

[a] Data from the Consortium on Safe Labor.

[b] Parous women tended to be admitted at more advanced dilations than nulliparous women; thus, measures of labor duration started at 5 cm for these women.

Data from Laughon SK, Branch DW, Beaver J, Zhang J. Changes in labor patterns over 50 years. *Am J Obstet Gynecol.* 2012;206:419.e1-419.e9.[18]

Critical findings from the studies by the Zhang group include the following: (1) The average labor curve for nulliparous women did not show a clear inflection point of dilation acceleration; (2) labor appeared to accelerate after 6 cm in multiparous women; and (3) no deceleration phase was detected.[7,17–19] The slope of cervical dilation was not linear; instead, cervical dilation rates progressively accelerated with each passing centimeter, thereby forming a hyperbolic curve for the aggregate, as shown in **Table 25-3** and **Figure 25-2**.[7,18,19]

Table 25-3 Duration of Labor from One Centimeter Dilation to the Next Centimeter Dilation, by Parity, in Women with Spontaneous Onset of Labor

Cervical Dilation (cm)	Parity 0 (n = 25,624)[a] (hr) Median (95%)	Parity 1 (n = 16,755)[a] (hr) Median (95%)	Parity 2 (n = 16,219)[a] (hr) Median (95%)
3–4	1.8 (8.1)	—	—
4–5	1.3 (6.4)	1.4 (7.3)	1.4 (7.0)
5–6	0.8 (3.2)	0.8 (3.4)	0.8 (3.4)
6–7	0.6 (2.2)	0.5 (1.9)	0.5 (1.8)
7–8	0.5 (1.6)	0.4 (1.3)	0.4 (1.2)
8–9	0.5 (1.4)	0.3 (1.0)	0.3 (0.9)
9–10	0.5 (1.8)	0.3 (0.9)	0.3 (0.8)

[a] Data from the Consortium on Safe Labor.

Modified with permission from Zhang J, Landy HJ, Branch DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcomes. *Obstet Gynecol.* 2010;116(6):1281-1287.[7]
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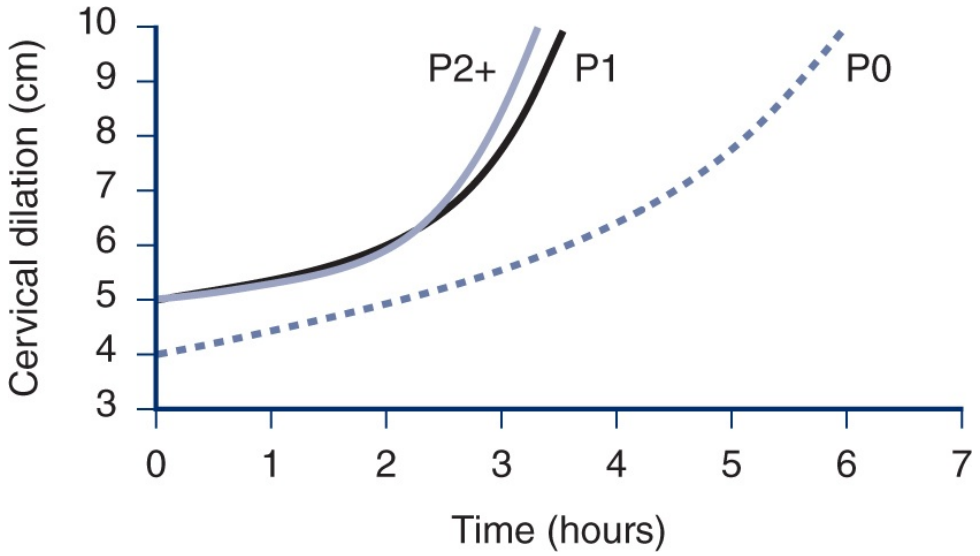


Figure 25-2 Average labor curves by parity in singleton term pregnancies with spontaneous onset of labor, vaginal delivery, and normal neonatal outcomes (95th percentile curves are not shown).

Abbreviations: P0, nulliparous women; P1, women of parity 1; P2+, women of parity 2 or higher.

Reproduced with permission from Zhang J, Landy HJ, Branch DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcomes. *Obstet Gynecol.* 2010;116(6):1281-1287.[7] Copyright 2010 by Lippincott Williams & Wilkins. *Obstetrics and Gynecology* by American College of Obstetricians and Gynecologists; American Academy of Obstetrics and Gynecology. Reproduced with permission of Lippincott Williams & Wilkins.

When the Zhang et al. group compared labor progress times found in the NCPP (1959–1966) data to labor progress times recorded in the CSL (2002–2008) database (using the same statistical analysis), which contains records of contemporary women giving birth, it became evident that the first stage of labor in the CSL cohort was longer by a median of 2.6 hours in nulliparous women and 2.0 hours in multiparous women, after adjusting for differences in maternal and pregnancy characteristics (e.g., older age, higher body mass index [BMI], and higher birth weight in the CSL when compared to the NCPP cohort).[18] Differences in labor duration may be partially attributed to significant changes in practice patterns over time, increasing prevalence of obesity, and average older age of laboring women. In addition, Friedman's statistical analysis was simpler than the advanced analytic technique applied by Zhang et al.

Spontaneous Active-Phase Labor

Labor and birth is a normal physiologic process. Thus, the sparse number of studies reporting on the duration and progress of women in *spontaneous* active-phase labor who do not experience interventions that could affect labor progress indicates the existence of an important knowledge gap—one that is difficult to study especially today due to the frequency of various interventions in contemporary practice. Albers[20,21] and Jones and Larson[22] specifically aimed to identify the duration of spontaneous active labor (i.e., no oxytocin, no epidurals, and no operative deliveries) among low-risk women who gave birth vaginally. In their studies, active labor was defined as the time necessary for the cervix to dilate from 4 to 10 cm (**Table 25-4**).

Viewed linearly, active labor dilation rates in these studies averaged 0.8 to 1.0 cm/hour for nulliparous women and 1.1 to 1.4 cm/hour for multiparous women; at the mean – 2 SD, rates were 0.3 to 0.5 cm/hour and 0.4 to 0.5 cm/hour, respectively.

Table 25-4 Duration and Cervical Dilation in Women During Spontaneous Active-Phase Labor

Trial	*n*	Dilation Used as Evidence of Active-Phase Onset (cm)[a]	Active-Phase Duration (hr)		Rate of Dilation (cm/hr)[b]	
			Mean (SD)	Mean + 2 SD	*Mean*	*Limit*
Nulliparous Women						
Albers et al., 1996[20]	949	4	7.7 (5.9)	19.4	0.8	0.3
Albers, 1999[21]	2511	4	7.7 (4.9)	17.5	0.8	0.3
Jones et al., 2003[22]	240	4	6.2 (3.6)	13.4	1.0	0.5
Multiparous Women						
Albers et al., 1996[20]	949	4	5.7 (4.0)	13.7	1.1	
Albers, 1999[21]	2511	4	5.6 (4.1)	13.8	1.1	
Jones et al., 2003[22]	240	4	4.4 (3.4)	11.6	1.4	

[a] Absolute value taken as evidence of active-phase onset (cm).

[b] Calculated based on the assumption that the cervical dilation phase ends at 10 cm, which approximates complete cervical dilation.

Based on Albers LL, Schiff M, Gorwoda JG. The length of active labor in normal pregnancies. *Obstet Gynecol*. 1996;87(3):355-359[20]; Albers LL. The duration of labor in healthy women. *J Perinatol*. 1999;19(2):114-119[21]; Jones M, Larson E. Length of normal labor in women of Hispanic origin. *J Midwifery Womens Health*. 2003;48(1):2-9.[22]

In their systematic review, which was limited to nulliparous women (*n* = 7009), Neal et al. concluded that the weighted mean rate of cervical dilation, based on linear calculations, was 1.2 cm/hour, when beginning with criteria commonly associated with the onset of active labor (i.e., dilation of 3 to 5 cm and regular contractions).[23] The weighted cervical dilation rate at the mean – 2 SD was 0.6 cm/hour. Studies included in this review reflected the diverse care patterns in contemporary practice—for example, frequent use of epidural analgesia, amniotomy, and oxytocin augmentation. These findings confirmed those of Perl and Hunter, who suggested that nulliparous labors progressing at 0.5 cm/hour or faster, in the absence of other problems or symptoms, are within normal limits.[24] In their study, 89.7% of term, nulliparous women with a spontaneous labor onset (*n* = 453 of 505) progressed at a rate of 0.5 cm/hour or faster. Only 8.6% of this cohort required a cesarean section (*n* = 37 of 453) and 0.8% required oxytocin augmentation of labor progress (*n* = 4 of 453).

Current research is exploring normal labor progress in subpopulations who may have intrinsic differences in their duration of labor. Factors such as race, ethnicity, obesity, age, induction of labor, and previous history such as cesarean have been assessed in a few studies to date.[25-30] Women who have a high BMI also progress in labor differently than do women with lower BMIs,[28,30] and induced labor progresses more slowly than spontaneous labor.[29] For midwives, it is critical that dilation rate expectations be based on the best contemporary research evidence and evolve when new evidence comes to light. Only by understanding typical labor progression can clinicians decide if and when interventions aimed at speeding

labor progress should be presented as appropriate options to laboring women.[31]

Partograph Studies

Partographs are tools that allow labor progress to be graphically recorded and visually assessed. They aid in the early detection of abnormal labor progress and are credited by some for decreasing rates of prolonged labor, oxytocin use, cesarean sections, and intrapartum morbidity/mortality as compared to usual care, although authors of a 2013 Cochrane systematic review did not find that use of a partograph was associated with improved outcomes.[32] Common partograph designs display time (in hours) on the *x*-axis and cervical dilation (in centimeters) on the *y*-axis. Correct use of the partograph is initiated during active labor.

Many partographs incorporate a graphically straight "alert" line, first introduced by Philpott and Castle,[33,34] that represents a dilation rate of 1 cm/hour based on findings from Friedman in the mid-1950s (**Figure 25-3**). Alert line incorporation was meant to represent the cervical dilation rate of the slowest 10% of nulliparous women in active labor, so that timely transfer from lower- to higher-resource settings could be accomplished.[33] An "action" line is conventionally placed a number of hours to the right of the "alert" line, most commonly 4 hours. A 4-hour action line is crossed only when cervical dilation averages less than 0.64 cm/hour for partographs initiated at 3 cm, less than 0.60 cm/hour for partographs initiated at 4 cm, and less than 0.56 cm/hour for partographs initiated at 5 cm. Only when the "action" line is reached are more aggressive management interventions, such as oxytocin augmentation, typically initiated in an attempt to accelerate labor progress.

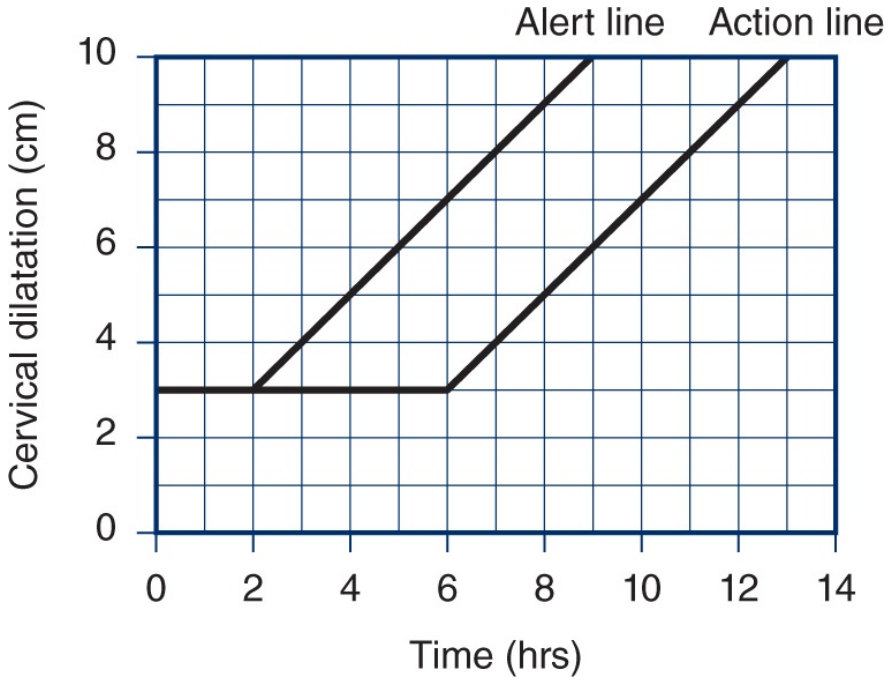


Figure 25-3 Central component of common partographs.

Reproduced with permission from Philpott RH, Castle WM. Cervicographs in the management of labour in primigravidae. I. The alert line for detecting abnormal labour. *J Obstet Gynaecol Br Commonw.* 1972;79(7):592-598.[33] Copyright © 1972 by John Wiley & Sons Ltd. Reproduced with permission of

Although the “alert” line is purported to identify only the slowest 10% of nulliparous labors,[33] studies spanning the past four decades have consistently reported that approximately 18% to 56% of nulliparous women cross the “alert” line following partograph initiation.[33-43] Among exclusively multiparous or mixed-parity cohorts, 10% to 42% of women cross the “alert” line.[35,36,42-44] With these findings, it became evident that a linear active labor cervical dilation rate expectation of 1 cm/hour is overly stringent for a large percentage of women diagnosed as being in active labor. Indeed, *it is the “action” line rather than the “alert” line that better segregates the slowest 10% of women*, although even the 4-hour “action” line is crossed by 10% to 45% of women.[36-38,40-43]

Contemporary findings regarding the hyperbolic progression of labor indicate that most existing partographs are not physiologically based for contemporary parturients. Even so, the potential for physiologically based partographs to provide an evidence-based framework for homogeneous labor assessment is great.[45,46] Large-scale, hypothesis-testing studies of physiologic partograph implementation are warranted because such instruments may go a long way toward improving birth outcomes in both low- and high-resource settings.

Current Controversies

Despite a wealth of research, definitive time frames for normal labor progress have not been fully established. The use of different statistical analyses and different populations has resulted in some controversy, primarily with regard to the cervical dilation at which most women are in active versus latent labor.[47,48] Findings from contemporary studies indicate that a cervical dilation rate approximating 0.5 cm/hour is achievable for approximately 90% of nulliparous laboring women with spontaneous labor onset. However, this *linear* rate must be evaluated judiciously in light of the physiologic acceleration of dilation that occurs during typical labor. Assessments of active labor progression are best based on appropriate hyperbolic labor curves; linear conceptualizations of cervical dilation, although common in contemporary practice, are fundamentally flawed and should be abandoned. The inter-individual variability in rate of progression during normal first-stage labor is sufficiently broad that management must be individualized to provide every woman with complete and accurate information so she can make informed health decisions.

Clinical Correlates of Duration of Labor Studies

Labor Dystocia

The expectation of cervical dilation during active labor is intimately linked to diagnosis of labor *dystocia*, which is broadly characterized as slow, abnormal progression of active labor. Furthermore, dystocia is the most common indication for primary cesarean birth in the United States.[49,50] To lower the rate of cesarean births, the diagnosis of dystocia and the management of women with this diagnosis need to be reevaluated.

In practice, diagnoses of dystocia are most often based on ambiguously defined delays in cervical dilation, beyond which labor augmentation is deemed justified. Criteria used to diagnose dystocia vary widely among clinicians, and commonly used definitions of dystocia neither differentiate normal from abnormal labor progress nor discriminate labors more prone to adverse outcomes. When clinicians have expectations for cervical dilation rates that are faster than physiologic reality, women with normal labor progression are at risk of being misdiagnosed with dystocia and receiving subsequent interventions aimed at expediting birth. The clinical tendency to over-diagnose dystocia explains, in part, why half of nulliparous women with spontaneous labor onset receive oxytocin augmentation.[7,19,51,52] This practice jeopardizes birth safety—oxytocin is the intervention most commonly associated with preventable adverse perinatal outcomes.[53-55]

Of even greater concern, dystocia is the indication for half of all cesarean births in nulliparous women.[49,56,57] At present, cesarean deliveries account for approximately one-third of all births in the United States.[58] The World Health Organization maintains that a cesarean rate higher than 10% is not associated with better health outcomes,[59,60] and higher rates may contribute to excess morbidity and mortality without benefit to mothers or babies.[61-64] Safely decreasing the cesarean rate is an ongoing national priority in the United States, and specific definitions for dystocia based on contemporary population-based criteria are needed to achieve this goal.

Pre-Active Labor Admission

The timing of when a woman is admitted for labor care significantly influences the labor process and outcomes; indeed, the timing of admission is one of the most important decisions that is made. Many women are inadvertently admitted prior to being in active labor, yet they are held to dilation expectations of active labor once admitted to a hospital setting. Women who are admitted early (e.g., less than 4 cm dilation) are approximately twice as likely to be augmented with oxytocin when compared to women admitted in active labor.[65-68] Indeed, the rate of oxytocin use is inversely related to cervical dilation at admission ($r = –0.79$, $P < 0.05$).[69] Moreover, the cesarean rate following early labor admission is reported to be more than twice as high as the reference group rate in most studies.[65-67,70-72] Cesarean births for dystocia are significantly more frequent in early-admission groups in studies reporting specific surgical indications.[65-68,71] Half of all cesarean births in nulliparous women for dystocia have historically occurred at 5 cm or less dilation.[73] This finding raises concern that many cesareans

may be performed prior to active-phase labor onset.

Zhang et al.'s findings can be used to illustrate the shortcomings of viewing cervical dilation linearly and to explain how this practice pertains to dystocia.[7] From 3 cm forward, calculations based on these authors' data demonstrated that dilation rates conceptualized as linear are faster than actual rates until some point after 5 cm dilation, when the linear rates become slower than actual rates, as described previously (**Figure 25-4**).[74] As a consequence, diagnoses of dystocia and interventions aimed at correcting "slow" labor are much more likely in earlier active labor—a point at which linear dilation expectations are less likely to be met. For this reason, it is important that clinicians base cervical dilation expectations on an evidence-based, hyperbolic labor curve.

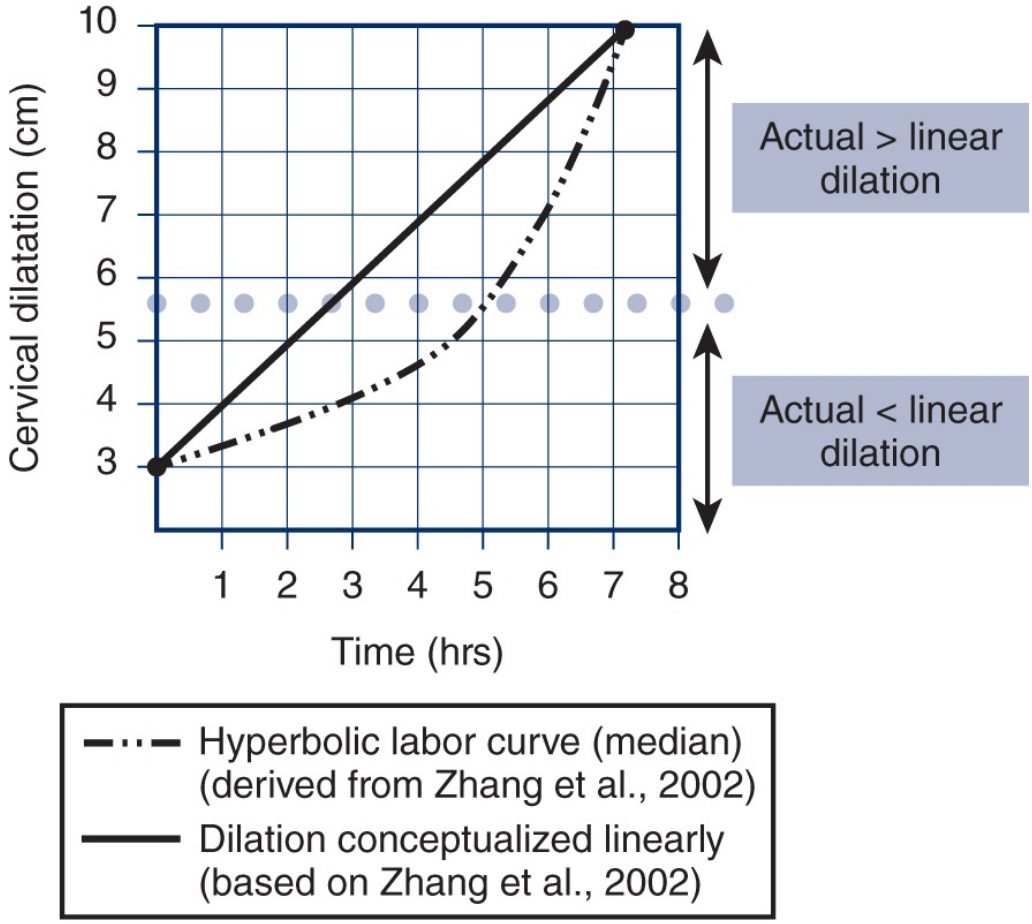


Figure 25-4 Hyperbolic, median nulliparous labor curve with linear conceptualization.

Data from Zhang J, Troendle JF, Yancey MK. Reassessing the labor curve in nulliparous women. *Am J Obstet Gynecol*. 2002;187(4):824-828.[19]

Reproduced with permission from Neal JL, Lowe NK, Patrick TE, Cabbage LA, Corwin EJ. What is the slowest-yet-normal cervical dilation rate among nulliparous women with spontaneous labor onset? *J Obstet Gynecol Neonatal Nurs*. 2010;39(4):361-369.[74]

Augmentation of Labor

Once a woman in spontaneous labor is in the active phase, attempts to accelerate labor may be justified if the dilation rate becomes slower than the expected minimum rate for the population.

Accelerative interventions are primarily used to decrease the number of dystocia-related cesarean births that are performed. However, the main interventions used by clinicians in an attempt to accelerate labor—that is, amniotomy and oxytocin augmentation—are used at surprisingly high rates in contemporary practice. For example, when Zhang et al. limited their analyses of the CSL database to only women with spontaneous labor onset, vertex presentation, vaginal delivery, and normal neonatal outcome (n = 62,415), approximately 45% were found to have received oxytocin augmentation during labor.[7] Oxytocin is a "high-alert medication" per the Institute for Safe Medication Practices,[54,75] Misuse of this drug is cited in half of all paid obstetric litigation claims.[55]

While studies comparing oxytocin use to no treatment are sparse, authors of a recent Cochrane Collaboration review found that early versus delayed oxytocin use results in a higher risk of uterine tachysystole associated with fetal heart rate changes requiring intervention (relative risk [RR] = 2.51; 95% confidence interval [CI] = 1.04–6.05).[76] Early oxytocin use reduces the mean duration of labor by approximately 2 hours. However, oxytocin was not effective in reducing the cesarean birth rate in this systematic review (RR = 0.88; 95% CI = 0.66–1.19).[76] This finding needs to be viewed with some caution. Oxytocin will induce uterine contractions and can be an important intervention for some women, but the decision to perform a cesarean section is affected by multiple factors and the finding that oxytocin does not decrease the cesarean rate is not conclusively related to an effect of the drug itself. With regard to amniotomy, there are no statistically significant differences in outcomes for women who undergo amniotomy and control groups in terms of first-stage labor duration or cesarean birth rates.[77]

Clinicians should bear in mind that the extent to which the relationship between prolonged labor and labor morbidity is causal is by no means certain; that is, it remains unclear whether the risks associated with longer labors are more strongly related to time in labor or to the interventions commonly used to shorten labor. Thus, prior to implementing interventions aimed at accelerating labor, any potential benefits must be weighed against the potential disadvantages, risks, iatrogenic consequences, and their attendant costs.

Midwifery Management During the First Stage of Labor

The dynamic nature of maternal and fetal responses to labor make the cyclic characteristics of the midwifery management process especially valuable as a structure for planning labor care. Continual reassessment with ongoing evaluation and modification of the management plan throughout the labor is expected as part of midwifery care.

Initial Evaluation of the Woman and Fetus

Initial evaluation of a woman presenting with signs and symptoms of labor includes review of history, physical assessment, and, often, laboratory investigations. The initial assessment of a woman who presents in labor not only seeks to ascertain the current physical well-being of the woman and fetus, but also assesses the encounter within the context of the woman's medical and obstetric history, social situation, and expectations. A comprehensive approach is necessary to identify actual and potential problems and to create a mutually agreeable and appropriate plan of care.

History

If the woman's prenatal record is available, it can be used as the source of information for much of the history. Specifically, personal information, past obstetric history, past medical and primary healthcare history, and family history should be reviewed. Present pregnancy history should be reviewed to confirm gestational age and estimated date of delivery, significant prenatal events, and presence of a personalized plan for birth. This spares the woman from having to reiterate history she has given before and from being disturbed at a time when she may be coping with the demands and stresses of labor. Critical items of the history should be double-checked with the woman to verify the existence of drug allergies, blood transfusions and reactions, and major obstetric or medical complications during her pregnancy. Documentation that the prenatal record was reviewed should be included in her intrapartum medical record. An interim history that includes any change in health status from the time of the last documented visit to the present encounter, chief complaint, and history of present illness, coupled with a brief review of pertinent systems, will complete the history database and give direction to the physical examination.

A woman presenting for labor evaluation without an available prenatal record presents a special challenge for caregivers. The woman may have been receiving regular prenatal care but simply not have records available for a variety of reasons, such as miscommunication about expected birth location or travel. However, women who have not received adequate prenatal care are at increased risk of unexpected adverse obstetric complications including preterm birth and stillbirth, giving birth to infants who are large or small for gestational age, and early neonatal death.[78] In the absence of a prenatal record, the midwife must elicit essential information, beginning with those data most relevant to the acuity of the clinical scenario. **Table 25-5** outlines the essential components of the health history when evaluating a woman presenting for labor evaluation and identifies their significance.

Table 25-5	Essential Components of Health History When Evaluating a Woman Presenting with Signs and Symptoms of Labor
History Element	**Significance**
Age	Birth at the extremes of the reproductive lifespan is associated with an increased risk for adverse perinatal outcomes
Parity	Influences labor progress and labor duration (e.g., progress is typically slower in nulliparous women compared to multiparous women)
Estimated date of delivery and estimated weeks of gestational age	Identifies the potential for newborn complications related to prematurity or postmaturity; establishes a baseline for evaluating fetal size as related to gestational age
Complications of current pregnancy, including group B *Streptococcus* status	Identifies existing or potential problems that may influence management of the current labor and birth
Major complications of previous pregnancies, including prenatal, intrapartum, and postpartum periods	Identifies potential recurring problems that may affect the current labor and birth
Previous labor experience, including duration	Previous labor experience influences expectations for labor progress and makes known previously used labor coping strategies
Mode of previous births/deliveries	Identifies previous operative vaginal deliveries or cesarean sections, which may influence management of the current labor and birth
Size of previous babies	Suggests an estimate of pelvic adequacy
Fetal movement pattern	Reflects fetal well-being
Vaginal bleeding	Differentiated from bloody show, frank vaginal bleeding is abnormal and typically contraindicates performance of a digital cervical examination; it indicates necessity for further evaluation including physician consultation, collaboration, or referral
Status of membranes	Duration of ruptured membranes and characteristics of amniotic fluid will influence management decisions
Time of onset of contractions, and character of contractions from onset to the present, including frequency, duration, intensity, and aggravating and relieving factors	Establishes the probable start of labor and helps discriminate prelabor contractions from true labor
Last oral intake	Provides a baseline from which to assess energy reserves and fluid status; also useful in anesthesia management in the event of surgery

Physical Examination

Essential components of the physical and pelvic examination performed at the initial labor evaluation are presented in **Table 25-6**. A physical examination that was performed prenatally can be reviewed as a baseline for the current physical examination when a prenatal record is available. An abbreviated examination can then be performed that focuses on any problem areas identified and areas where new information is needed to make management decisions. Documentation that the previous examination was reviewed should be included in the woman's healthcare record. A comprehensive physical examination is indicated when a woman has no prenatal records available or has received inadequate prenatal care.

Table 25-6 **Essential Components of the Physical Examination When Evaluating a Woman Presenting with Labor Signs and Symptoms**

Components of Examination	Significance
Physical Examination	
Vital signs: blood pressure, temperature, pulse, respirations	Elevated blood pressure may indicate a hypertensive disorder, while lowered blood pressure may indicate shock. In the presence of a normal diastolic measurement, elevation in systolic blood pressure usually indicates anxiety or pain. Elevated temperature indicates an infectious process or dehydration. Elevated pulse and/or respirations may indicate infection, shock, dehydration, or anxiety.
Auscultation of heart and lungs	Screens for and identifies acute or previously unrecognized conditions that could potentially adversely affect the current labor and birth.
Abdominal palpation to determine contraction pattern and fetal lie, presentation, position, and engagement	The contraction pattern helps determine labor status. Fetal lie, presentation, and position influence the course of labor and guide care decisions regarding mode of delivery and necessity for consultation, collaboration, or referral. Engagement of the fetal presenting part is reassuring for pelvic adequacy.
Abdominal palpation to determine estimated fetal weight and fundal height	In relation to gestational age, identifies the possibility of inaccurate dating, smaller or larger than expected fetus or multifetal gestation, oligohydramnios or polyhydramnios, or other abnormalities and their associated complicating factors.
Visual inspection for abdominal scars	Confirms surgical history or identifies previously unidentified surgical procedures, including previous cesarean sections.
Assessment for presence of peripheral or facial edema	Facial edema alone or in association with peripheral edema is a classic sign of preeclampsia.
Pelvic and Cervical Examination	
Cervical effacement and dilatation	Progressive cervical effacement and dilatation is the mark of true labor.
Position of the cervix	Location of the cervix in an anterior or middle position is associated with greater readiness for labor compared to a cervix that is posterior.
Station of fetal presenting part	Progressive fetal descent is indicative of progressive labor and pelvic adequacy.
Presence of molding or caput succedaneum	The presence or absence of molding and/or caput indicates fetal adaptation to the maternal pelvis and indirectly reflects the degree of pelvic adequacy.
Fetal lie, presentation, and position	Digital examination findings confirm and enhance abdominal examination findings relative to fetal lie, presentation, and position.
Tone and elasticity of vagina and length of perineum	The potential for perineal lacerations may be suggested by assessment of the tone and elasticity of the vagina and length of perineal body.
Confirmation of membrane status	Palpation of intact fetal membranes or amniotic fluid expulsion upon cervical examination is suggestive of membrane status.
Visual inspection of perineum	Identification of lesions, vulvar varicosities, and/or vaginal discharge including frank bleeding will impact the management plan.
Assessment of fetal heart rate	Indicates absence or presence of risk factors for fetal acidemia based on fetal heart rate characteristics and response to uterine contractions.
Optional or Supplemental Examinations	

Measurement of maternal weight	Relevant when compared to prepregnancy weight to ascertain total weight gain during pregnancy, or when compared to previous prenatal visit measurements to assess interval weight gain. Very rapid weight gain is an indirect measure of edema and possibly preeclampsia.
Clinical pelvimetry	Supports clinical judgment when estimating pelvic adequacy.
Evaluation of reflexes and determination of the presence of clonus	Hyperreflexia and clonus are signs of severe preeclampsia/eclampsia.
Speculum examination	Provides visualization of the cervix and vaginal vault to confirm rupture of membranes, collect laboratory specimens, and estimate cervical dilation and effacement.

Physical examination findings or vital signs indicative of a potential or actual complication require further evaluation. For example, an elevated blood pressure is an indication for eliciting and evaluating reflexes and for assessing for the presence of clonus.

Controversy exists about the role of digital cervical examinations in the initial and ongoing evaluation of a woman with labor signs and symptoms. The unique clinical situation of the laboring woman and her desires, along with the clinical judgment and expertise of the midwife, are taken into consideration when considering the necessity of performing a digital cervical examination.

In institutions that have continuous electronic fetal monitoring capability, it is common practice to obtain a baseline fetal heart rate (FHR) recording (e.g., 20 minutes) as the means of initial evaluation of fetal well-being for most, if not all, women presenting for evaluation of labor. This practice, when applied to women who do not have an obstetric or medical risk for fetal acidemia, does not improve maternal or neonatal outcomes and, when compared to initial assessment by intermittent monitoring, may be associated with an increase in intrapartum interventions including continuous electronic fetal monitoring (RR = 1.30; 95% CI = 1.14–1.48) and cesarean delivery (RR = 1.20; 95% CI = 1.00–1.44).[79] National guidelines are silent on the preferred method of evaluating fetal heart rate for women presenting for evaluation with labor signs and symptoms, but are clear regarding the appropriateness of intermittent auscultation for ongoing evaluation of low-risk women in labor.[2,80,81]

Laboratory Investigation

Available prenatal records can be reviewed to identify the woman's blood type and Rh status, anemias, glucose tolerance testing, and specific perinatal infections including GBS carrier status, hepatitis B infection or carrier status, and HIV status. In the case of a woman presenting with no prenatal record, all routine prenatal laboratory tests will be obtained to provide a baseline for development of a management plan.

Clinical judgment and institutional policy will influence the decision to order further laboratory studies. If the woman will be having an intravenous infusion, blood specimens can be obtained concurrently with intravenous line placement. **Table 25-7** identifies laboratory studies commonly performed for evaluation of a woman with labor signs and symptoms.

Table 25-7	**Commonly Performed Laboratory Investigations for a Woman Presenting with Labor Signs and Symptoms**

Laboratory Test	Significance
Complete blood count (CBC)	Provides baseline measures of hemoglobin and hematocrit, thereby evaluating for anemia (a risk factor for postpartum hemorrhage)
	Provides a baseline for the white blood cell count, which can indicate infection—although transiently high values are common with labor
	Provides a baseline platelet count, which may be used by anesthesia personnel when determining anesthesia eligibility
Blood type, Rh status, and antibody screen[a]	Confirms prenatal blood group and Rh status
	Provides a comparative value to prenatal results, allowing for assessment of maternal antibodies development
	Provides basic information for a blood bank in the event that blood transfusion is needed
Urinalysis	Identifies the presence of protein, a marker for preeclampsia
	Identifies the presence of glucose, a marker for poorly controlled diabetes
	Identifies the presence of ketones, a reflection of fat metabolism and current nutrition status
	Indicates hydration status (i.e., specific gravity measurement)
	Identifies abnormal components in the urine that may direct differential diagnosis toward urinary tract infection

Confirmation of Labor Status

A diagnosis of labor warrants the woman's admission to the birth center/hospital or continuous midwife attendance when a home birth is planned. Because cervical dilation rates indicative of active labor do not begin for many women until cervical dilatation reaches 6 cm or more,[7] labor status determinations must be based on at least two adequately spaced cervical examinations—for example, 2 to 4 hours apart. In the presence of regular, painful contractions and complete or near-complete effacement, it is reasonable to consider a woman to be in active labor at 4 cm or 5 cm dilation *if that state of dilatation is immediately preceded by cervical change over time* (i.e., 1 cm or more in a 2-hour or shorter window), or at 6 cm or more regardless of the rate of previous cervical change. Unless labor is clearly advanced, a single-point cervical dilation measurement does not reliably differentiate labor phases. When a diagnosis of labor cannot be made with relative certainty, observation before admission to the birthing unit is prudent, assuming the woman is planning to give birth in the hospital.

It may take several hours of ongoing assessment to differentiate between prelabor, latent labor, and active labor. Between assessments and after fetal well-being is confirmed, the woman may be encouraged to walk, rock, sit on a birthing ball, or simply rest. In hospital and birth center settings, when no change in dilation is noted at the next examination before active labor, the woman may safely go home if she has no transportation problems and wants to go home.

In making a diagnosis of labor, clinicians must carefully differentiate not only between true labor and prelabor, but also between labor and any obstetric or non-obstetric discomforts or complications that can masquerade or be misinterpreted as labor, such as urinary tract infections or placental abruption.

Components of Midwifery Care for Laboring Women

Labor Support and Pain Management

Labor support and pain management are intertwined. Labor support consists of techniques and interventions that support the physical and emotional experiences of the woman in labor and includes recognizing the therapeutic value of human presence—a hallmark of the art and science of midwifery. Reviewed in detail in the *Support for Women During Labor* chapter, the inclusion of labor support and pain management as a basic component of midwifery care reflects the importance of intentionally and continually evaluating and modifying the midwifery care provided throughout labor. Women who receive continuous labor support are more likely to have a spontaneous vaginal birth, use less pain medication, have slightly shorter labors, and be more satisfied with their birth experiences than women who do not receive such support.[82]

Although some practice environments essentially preclude the midwife from personally acting as the exclusive continuous labor support provider, midwives remain in the unique position of advocating for this essential care. Midwives can engage with and support local doulas and/or other labor support caregivers so that they are engaged in the labor support process.

Maternal Position and Level of Activity

The level of physical activity and positions used during labor are ideally those chosen by the laboring woman. It is rare that a woman would have a contraindication to assuming any position that she prefers. When given a choice, many women prefer an upright position or ambulation throughout much of their labor. However, the birth environment may affect the level of empowerment a woman has to assert her desires, and many hospital labor settings have a culture of lying in bed during the first stage of labor. While laboring in bed is considered to be convenient to some providers, it may not be optimal for the progress of physiologic labor or for the comfort of the laboring woman.

Women assuming upright positions have shorter labor durations by approximately 1 hour and are less likely to have an epidural or cesarean birth, as compared to women who maintain recumbent positions.[83] Creative use can be made of furniture, pillows, birthing balls, or an adjustable bed to support a laboring woman in a variety of upright positions, including hands and knees, sitting, standing, and squatting. The midwife can support unlimited activity and freedom of choice of positions for low-risk women in labor by routine use of intermittent fetal auscultation and becoming comfortable with and skilled at the process of assessing fetal heart rate and contractions with women in a variety of positions.

When resting in bed is necessary or desired, lateral recumbent positions are preferred to supine positions because they reduce the potential for aortic/venae cavae compression with resulting maternal hypotension and potential fetal compromise. Lateral positions also facilitate kidney function and do not interfere with coordination and efficiency of uterine contractions.

Rupture of the fetal membranes is not usually a contraindication to ambulation and upright positions. Once fetal well-being is assured, if the fetus is in a cephalic presentation and the fetal presenting part is engaged in the pelvis or well applied to the cervix, there is no reason to

restrict ambulation or upright positions. These activities may be desirable to the woman, as the intensity of the contractions often increases after rupture of membranes and upright positions tend to facilitate maternal comfort. Ambulation or upright positions may be contraindicated upon rupture of membranes if the fetal head is unengaged or in the case of a malpresentation because of the heightened risk of umbilical cord prolapse.

Women with medical or obstetric conditions such as severe preeclampsia, placental abruption, or acute infections will necessarily have their activity restricted due to their physiologic instability, the effect of medications, or increased fetal risk requiring continuous electronic fetal monitoring. Women who have physical mobility disabilities may require additional modifications to facilitate comfort and labor progression. Finally, women who have a history of sexual or physical abuse may find any position such as lithotomy, or having providers stand over her, a trigger of previous trauma. For these women, if a plan has not been established prenatally for labor management, discussion early in labor to identify positions of comfort will be of value.

Hydration and Nutrition

Hydration and nutrition are potentially important variables for optimal uterine perfusion and myometrial function and, therefore, may be related to uterine efficiency during labor. Contemporary management of labor in the United States typically involves limited oral nutritive intake and noncaloric intravenous fluid administration. The primary rationale cited for withholding food and fluid during labor is the decreased risk of gastric content aspiration during general anesthetic induction—an extremely rare but serious syndrome first described by Mendelson in 1946.[84] Of course, obstetric anesthesia has changed considerably since the 1940s, with greater use of regional anesthesia and better general anesthetic technique. Moreover, fasting does not guarantee an empty stomach or less acidity. Overall, modern evidence shows no benefits or harms associated with oral intake during labor, so there is no justification for the restriction of fluids and food in labor for women at low risk for complications. Adequate hydration during labor would seemingly assist in the delivery of oxygen and nutrients as well as facilitate the elimination of waste from the contracting uterus, akin to how proper hydration benefits the skeletal muscle of athletes.

Four randomized studies have been conducted to investigate maternal hydration as a potential variable in labor progress, all focusing on nulliparous women.[85-88] In the earlier studies, Garite et al.[85] and Eslamian et al.[86] randomized low-risk, nulliparous women in spontaneous labor to receive 125 or 250 mL/hour of intravenous isotonic fluids while receiving little or nothing by mouth. In the 250 mL per hour groups, Eslamian et al. reported a shorter first stage of labor duration (3.9 ± 1.4 versus 6.1 ± 1.8 hours, respectively; $P < 0.0001$),[86] while Garite et al. demonstrated a strong nonsignificant trend toward a shorter first-stage labor.[85] Both research teams found fewer prolonged labors in the 250 mL per hour group, and Eslamian et al. reported that oxytocin augmentation was significantly less likely in the women who received more fluids (8.1% versus 20.4%, respectively; $P < 0.001$).[86]

Coco et al.[87] studied whether giving low-risk, nulliparous women more intravenous fluid reduced labor duration when oral intake was unrestricted (e.g., water, juice, or even

carbonated soft drinks). This team found that increased intravenous hydration does not decrease labor duration when women are permitted to drink freely, nor does it decrease rates of oxytocin augmentation or cesarean births. Likewise, Kavitha et al.[88] found no significant differences in labor duration, oxytocin augmentation, or mode of delivery between women receiving oral fluids only or intravenous fluids at a rate of either 125 or 250 mL/hour, although those women who received intravenous hydration were less likely to vomit during labor. A significant shortcoming in all of these randomized hydration studies was that none measured maternal hydration by scientific means (e.g., urine specific gravity). Thus, the women's actual hydration status remained unknown.

Nutritional needs during labor are not well understood, although it has been reported that women in the third trimester of pregnancy exhibit a state of "accelerated starvation," characterized by rapid rises in plasma beta-hydroxybutyric acid (the principal labor ketone) and a concomitant fall in plasma glucose. This process of accelerated lipolysis conserves glucose for the fetus. Ketones are produced in the liver as a by-product of fat catabolism when glycogen stores are unavailable (e.g., in starvation states or during exercise) and are normally oxidized to carbon dioxide, water, and alternative forms of energy that are usable by tissues such as a contracting uterus.[89,90] Production of ketones in quantities above the level of need results in ketosis and may have deleterious effects on uterine function.[89]

Authors of a Cochrane review published in 2013 found no significant differences in duration of labor, augmentation of labor, cesarean births, or Apgar scores between women with and without oral fluid and food restriction during labor.[91] Given the current state of evidence, women should be free to drink and eat in labor as they wish and encouraged to maintain their fluid intake.

Intravenous Access

Routine prophylactic insertion of intravenous access is a practice that is unnecessary in the provision of normal labor and birth care for women who have no significant obstetric or medical risks. Instead, the decision to initiate and maintain intravenous access during labor should be based on actual or potential risk factors for each woman. Women who cannot tolerate oral fluid intake may require intravenous fluids. Intravenous access is also necessary for administration of some medications, such as antibiotic prophylaxis for women who are carriers of GBS, pain medications, or oxytocin augmentation. Prior to initiation of epidural anesthesia, establishing intravenous access allows for administration of isotonic fluid blood volume expanders to mitigate epidural-related hypotension; continuing access after epidural placement allows for ongoing fluid administration and medication administration should complications develop. Institutional policies that require intravenous access exist in many hospital birth settings, and eliminating these policies is one example of changes needed so that policies are in line with existing evidence to support normal physiologic labor and birth.

Membrane Management

Historically, being born "in the caul" or with intact membranes was considered a sign of good

fortune and occasionally magic gifts. Yet artificial rupture of membranes (AROM) during labor has commonly been believed to accelerate the progress of labor and is used frequently in contemporary practice. AROM can be employed to induce labor either alone or with other agents, used routinely during labor in an effort to speed progress, or used selectively as a treatment for dystocia. Because AROM is associated with adverse outcomes, however, the effectiveness of AROM for each of these situations must be examined. Risks associated with AROM include umbilical cord compression with resultant fetal heart rate decelerations, umbilical cord prolapse, maternal discomfort from the procedure, increased risk of infection, and, rarely, rupture of fetal vessels (vasa previa).[77]

Two trials have evaluated the effectiveness of early amniotomy (during latent labor) as an adjunct during induction. Macones et al. evaluated outcomes for nulliparous women who were induced at term and who had an amniotomy before 4 cm dilation compared to outcomes for women who had an amniotomy after they were at 4 cm dilation.[92] More women in the early amniotomy group gave birth within 24 hours (68% versus 56%, $P = 0.002$) and there was a mean 2-hour shorter labor in the early amniotomy group.

The use of AROM to treat dystocia has also been investigated. Rouse et al. randomized women with active-phase arrest to amniotomy with oxytocin or oxytocin alone ($n = 108$).[93] No significant differences in mode of birth or duration of labor were observed between the two groups. The women in the amniotomy group had a nonsignificant trend toward more chorioamnionitis and endometritis. Because they had internal uterine pressure catheters and fetal scalp electrodes placed at the time of amniotomy, it is not clear if AROM or the other interventions increased this risk for infection; however, there is known relationship between use of intrauterine pressure catheters and increased risk for infection.

Wei et al. evaluated early versus late use of amniotomy and oxytocin augmentation for both prevention and treatment of dystocia in a Cochrane meta-analysis ($n = 8033$).[94] In the prevention of dystocia trials, women in the early amniotomy/oxytocin groups had a modest reduction in cesarean birth rates (RR = 0.87; 95% CI = 0.77–0.99) and shorter labor (mean difference: –1.57 hours). In the treatment of dystocia trials, there was no reduction in cesarean birth rates (RR = 1.47; 95% CI = 0.73–2.96), nor was there a reduction in the duration of labor between women who had early versus late amniotomy and oxytocin augmentation. There were no differences in the rate of infection or adverse neonatal outcomes.

Current evidence suggests that when amniotomy is used in conjunction with oxytocin for induction or as a method of preventing dystocia in women who have mild labor delays, the decrease in labor duration associated with amniotomy may be statistically significant but not clinically relevant. The reduced risk of cesarean birth is significant but modest. In contrast, amniotomy alone does not appear to be a beneficial treatment for women with active-phase arrest.

The use of AROM as a routine measure to speed the progress of labor in women without dystocia has been evaluated in several randomized trials and a Cochrane meta-analysis. Unlike in the dystocia trials, AROM in women who do not have dystocia was associated with a trend toward an increased risk for cesarean birth (RR = 1.27; 95% CI = 0.99–1.63; $n = 5583$; 15 trials) without a concomitant shortening of the first stage of labor.[73] There was a similar trend toward more fetal heart rate abnormalities (RR = 1.09; 95% CI = 0.97–1.23) but no

differences in serious neonatal morbidity. The American College of Obstetricians and Gynecologists recently recommended that amniotomy should not be performed in women with normally progressing labor unless required to facilitate monitoring—a position that supports a long-standing midwifery practice.[2]

If there is a clinical rationale to perform an amniotomy, a cephalic presentation and engagement in the pelvis should be confirmed. Before performing AROM, the midwife carefully reassesses the fetal station and ensures the fetal head is well applied to the cervix. Keeping the fingers in the cervix, the membranes can be gently disrupted with the Amniohook. Care should be taken to avoid scratching the fetal head and the clinician's fingers should be left in place during the initial gush of fluid to ensure a prolapsed cord does not occur. The fetal heart rate should be assessed during the procedure and monitored frequently for a short time afterward.

In summary, routine amniotomy for women in spontaneous term labor may do more harm than good. This procedure should be reserved for treatment of dystocia, as an adjunct intervention for women who have a clear indication for induction of labor, and when internal monitoring is required. The woman should be reassured that aside from the potential discomfort associated with any vaginal examination, there is no pain associated with AROM for her or her fetus. Finally, the woman should give consent for this procedure and a formal shared decision-making process that reviews the benefits, risks, and alternatives should be conducted.

Fetal Heart Rate Monitoring

Fetal monitoring does not necessarily imply "continuous electronic fetal monitoring," but rather reflects an attitude of heightened sensitivity on the part of the midwife to fetal well-being, via ongoing evaluation of the fetal heart rate via the method most appropriate to the individual woman and fetus. The *Fetal Assessment During Labor* chapter details the methods and indications for intermittent auscultation of the fetal heart rate as well as continuous external and continuous internal fetal heart rate assessment.

Uterine Contraction Monitoring

Uterine activity can be evaluated by correlating the woman's perceptions of contractions with observation and abdominal palpation and/or one of two electronic monitoring methods: an external tocodynamometer or an intrauterine pressure catheter.

Labor progress is the best indicator of adequate contractions. Nevertheless, it is generally accepted that three contractions within 10 minutes is the minimum frequency necessary to achieve progressive cervical change in active labor. "Adequate" labor contractions typically do not allow indentation of the uterine fundus when palpated abdominally at their acme. The maternal response to contractions provides insights into the frequency, duration, and intensity of contractions, but it is important to keep in mind that women's responses to labor vary widely. The intensity of a woman's external response to a contraction is influenced by environment, culture, and individual coping abilities.

When considering application of an electronic uterine monitor, the need for uterine activity data must be weighed against the need of the woman for mobility and freedom of movement. The rise of the uterus within the abdomen during a contraction can be easily visualized on a woman of average or low BMI. This change in abdominal shape is the mechanism by which an external electronic tocodynamometer functions; that is, this device captures the graphic image of a contraction as the abdomen lifts against the pressure transducer, which is held in place by the resistance of an abdominal band. External tocodynamometry can present graphically the frequency and duration of uterine contractions, but it relies on correct placement and calibration and may not work well for women who are overweight or obese. In addition, the tocodynamometer is unable to provide accurate information regarding the intensity of contractions. It is highly sensitive to maternal movement, predisposing the graphic data to artifacts and errors.

To counter limitations of the traditional tocodynamometer, researchers are investigating the use of electrical uterine myography. This emerging method of electronic uterine monitoring uses noninvasive electrodes placed on the maternal abdomen to report electrical activity of the uterine muscle, similar to how an electrocardiogram records the electrical conduction of the heart. This promising technology is able to graphically present the same information as that provided by a tocodynamometer, while having the advantage of being less sensitive to maternal body type and movement.

Women generally sense contractions when the contractions exert approximately 15 mm Hg of intrauterine pressure; this is also the minimum strength of contraction that can be detected by an observer's hand. In contrast, internal uterine monitoring detects the increase in tonus slightly sooner than the woman or her attendants[95] (**Figure 25-5**). Internal uterine monitoring is the most accurate but most invasive method of evaluating uterine activity (**Figure 25-6**). The intrauterine pressure catheter requires membranes to be ruptured and has a low—albeit real—risk of uterine or placental perforation and infection. When placed correctly (i.e., within the amniotic fluid compartment), the intrauterine pressure catheter provides information regarding frequency, duration, and intensity of contractions. The intrauterine pressure catheter has the added feature of enabling the instillation of fluid into the uterus (amnioinfusion) to treat recurrent variable fetal heart rate decelerations associated with fetal umbilical cord compression.

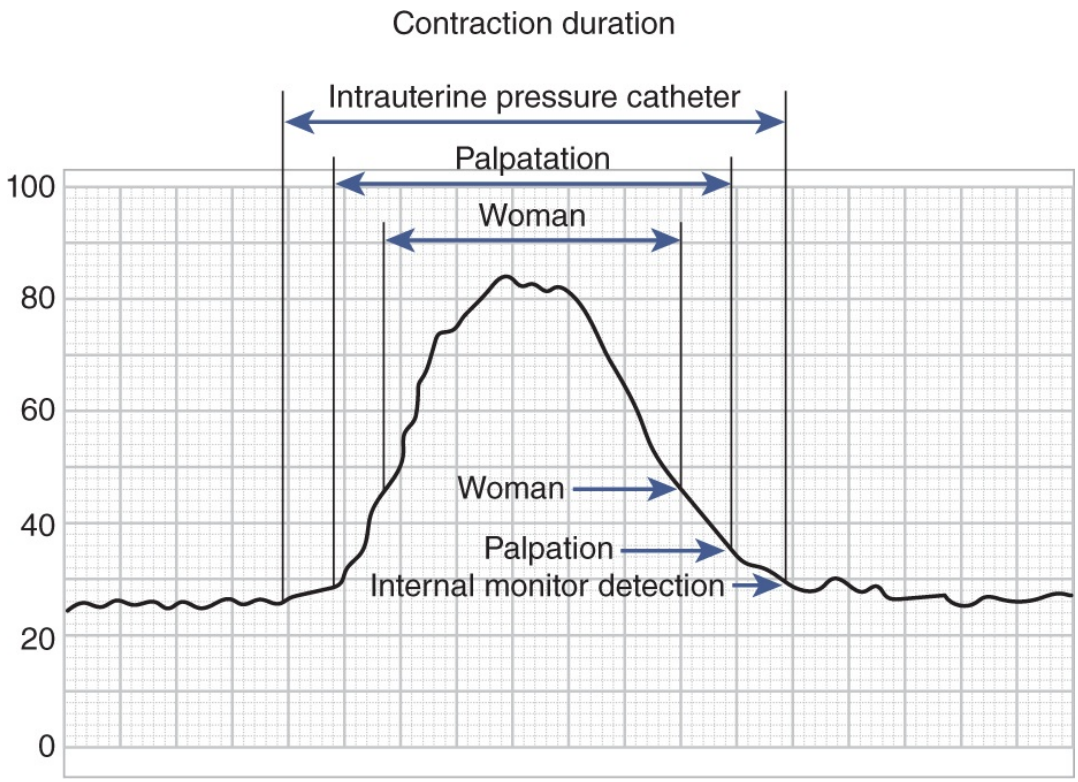


Figure 25-5 Relative sensitivity of various methods available for detection of uterine contractions.

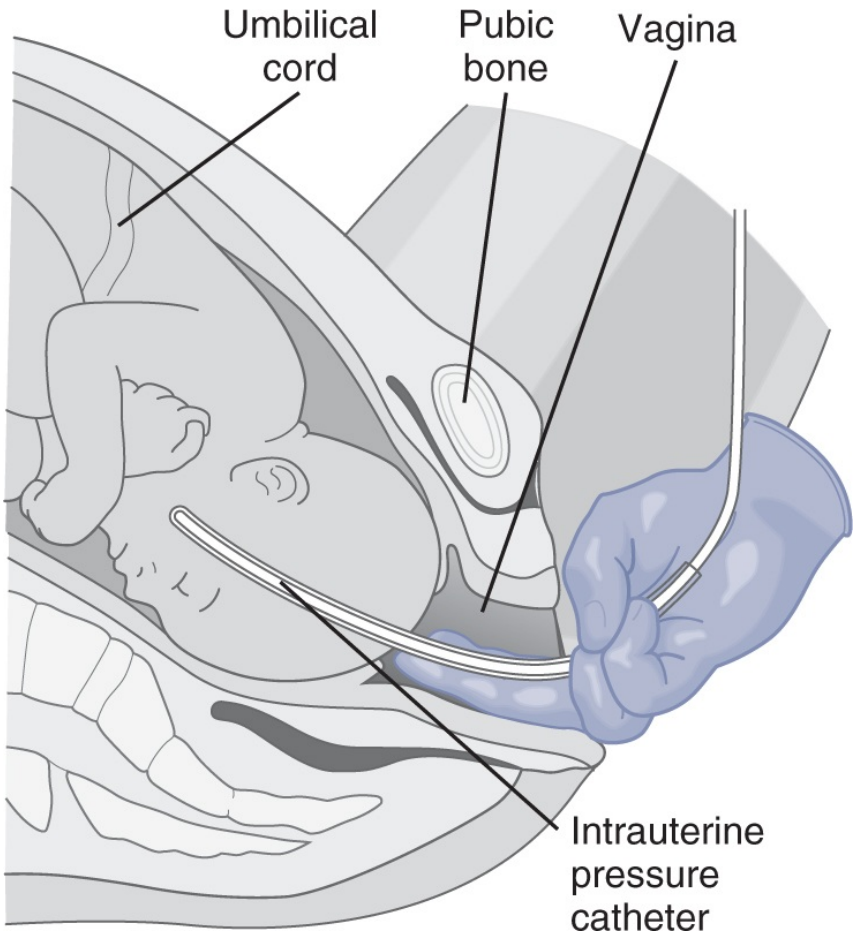


Figure 25-6 Insertion of an intrauterine pressure catheter.

Montevideo units (MVUs) are used to assess the effectiveness of uterine contractions measured via an intrauterine pressure catheter. MVU levels are determined by subtracting the numeric amplitude of each contraction that occurs in a 10-minute window from the baseline uterine tonus amplitude (in millimeters of mercury [mm Hg]) and then summing the total calculated rise in amplitude over the 10-minute time period. Historically, 200 to 250 MVUs has been used to define “adequate” contractions based on Calderyo-Barcia’s studies from the 1960s, which determined the minimum MVU values that reliably predict vaginal birth.[96]

Introduction of an intrauterine pressure catheter may be indicated during labor augmentation when labor dystocia is suspected. However, this device has no place in the management plan for labor in the absence of fetal or obstetric complications.

Continuing Evaluation During Labor

Continuing evaluation during labor has three primary focuses: (1) maternal well-being, (2) fetal well-being, and (3) labor progress. Assessing maternal status includes monitoring maternal vital signs, urinary output, coping status, and general well-being. Continuing evaluation of the fetus includes the monitoring of fetal heart rate. In addition to monitoring cervical dilation over time, evaluation of fetal presentation and position and fetal adaptation to the maternal pelvis is an essential component of evaluating labor progress.

Maternal Well-Being

Maternal Vital Signs

The frequency with which vital signs are assessed in the absence of complications may vary among settings, but is typically detailed in an institutional policy to ensure adherence to a minimum standard. Unfortunately, the standards for the frequency of vital signs for low-risk women in labor may be based in nursing and institutional tradition rather than on evidence of clinical relevance in laboring women. The following schedule for checking vital signs is frequently encountered as policy for a woman (without epidural anesthesia) during the first stage of labor who does not have a specific condition that would require more frequent monitoring:

- Blood pressure, pulse, and respirations: every hour
- Temperature: every 2 to 4 hours when the temperature is normal and the membranes are intact, and every 1 to 2 hours if the temperature is abnormal and/or after the membranes have ruptured

As a woman's status may change over the course of her labor, the frequency of assessing vital signs should be adjusted to match her unique situation.

Urinary Output

The bladder is a pelvic organ. A distended bladder can impede the progress of labor by preventing fetal descent as well as increasing the discomfort and pain in the lower abdomen that women frequently experience during labor. A woman in labor should be encouraged to empty her bladder at least every 2 hours during the active phase of the first stage of labor. This provides an opportunity for the clinician to evaluate maternal hydration status. Presence of ketones or protein in the urine may be helpful in developing the management plan and can be assessed as indicated.

With deepening descent of the fetal presenting part into the true pelvis during labor, the bladder may be compressed to the point at which even small urine volumes cause distension. In the third stage of labor, a distended bladder can inhibit the ability of the uterus to contract effectively, increasing the risk of postpartum hemorrhage secondary to uterine atony. Bladder hypotonicity, urine stasis, and infection during the postpartum period can result from traumatic

pressure exerted on a distended bladder during labor.

A distended bladder may appear as a bulge above the symphysis pubis and, in severe cases, may extend as high as the umbilicus. When the fetus is in a posterior position, the contour of the woman's abdomen may look as though she has a full bladder; bladder distension must then be ruled out.

In the event of bladder distension, the first step is to facilitate spontaneous voiding. The best method is for the woman to walk to the toilet if there are no contraindications to ambulation. If she is unable to be out of bed and if the common methods (having her listen to the sound of running water; running warm water over her perineum; applying light suprapubic pressure; and having her practice perineal relaxation) do not initiate urination, then catheterization may be considered.

Bladder distension can occur in any laboring woman, but is especially likely in women with epidural anesthesia who receive a bolus of fluid prior to initiating the epidural. Once the epidural is active, the woman cannot feel the urge to urinate and may not have the muscle control needed to void. Intermittent or indwelling insertion of a urinary catheter to drain the bladder should be considered to minimize the risk of urinary retention or infection for these women, especially if they are remote from delivery.

General Maternal Condition

Continuing evaluation of maternal status includes evaluation of several areas that interrelate and overlap: the woman's level of fatigue and physical depletion, her behavior and responses to labor, her perception of pain, and her ability to cope with labor.

A woman's level of fatigue and physical depletion is affected by her state at the start of labor, maintenance of hydration during labor, length of labor, and her ability to cope with the demands placed upon her by labor. This can be a vicious cycle: A woman's lack of ability to cope may increase fatigue, and fatigue can decrease her ability to cope. Occasionally a woman enters labor truly exhausted and dehydrated from days of the general aches and symptoms characteristic of the end of pregnancy or from a prolonged latent phase. A mutually agreed-upon hospital-based management plan for such a woman might include considering an early epidural and intravenous fluids.

A woman's behavior changes throughout labor, and these changes can be used in evaluating her response to labor. Her expressions and behaviors are also affected by her degree of self-efficacy, anxiety or fear, expectations, and the amount of pain she is experiencing. In another cycle of interplay, these factors affect her coping ability, and her ability to cope in turn affects her self-efficacy, anxieties, and fears. Evaluation of the woman's behavior during labor will help determine the support and comfort measures needed, including consideration for medication. That said, interpretation of another persons expressions or behavior is heavily influenced by cultural expectations and can be easily misjudged. Therefore, the midwife must evaluate a laboring woman in the context of her world view, and be careful to avoid making judgements based on the midwife's cultural dictates.

Fetal Well-Being

Ongoing and standardized fetal heart rate assessment is essential to evaluating fetal well-being throughout labor regardless of the method chosen. The *Fetal Assessment During Labor* chapter provides details on fetal assessment in labor.

Labor Progress

Digital Cervical Examinations

Internal cervical examinations are intrusive and often uncomfortable for women; however, appropriately timed digital cervical dilation examinations inform labor management. Use of contemporary guidelines that account for normal duration of labor and rate of cervical dilation is important. While it is appropriate to wait several hours prior to doing a cervical examination as the cervix changes in early active labor (e.g., from 4 cm to 5 cm), it may be inappropriate to wait for such a long period of time later in labor (e.g., between 8 cm and 9 cm).

Supplemental indicators of labor progress in addition to cervical dilation can and should be obtained from each digital cervical examination that is performed during labor, including (1) cervical effacement, (2) position (posterior, mid-position, anterior), (3) consistency (firm or soft), and (4) fetal station and position. The frequency with which cervical examinations are performed depends both on the woman's condition and on the midwife's ability to use other parameters for evaluating progress in labor. It is not always necessary to perform an internal examination to gain insights into the progress of labor. The traditional practice of conducting cervical examinations every 1 to 2 hours simply subjects the woman to unnecessary discomfort, intrusion, and increased risk of infection. Astute observation of the woman—including her behavior, contraction pattern, signs and symptoms of transition into second-stage labor, change of location of back pain, change in location of maximum intensity of fetal heart tones, and change in position of fetal heart tones—can give valuable insights into labor progression. This information does not, however, negate performance of a cervical examination if a question arises regarding labor progress.

During normal first-stage labor, a cervical examination may be indicated in the following situations:

- To establish an informational baseline that can be used for appropriately timing further examinations to establish labor status prior to admission or labor interventions (prelabor, latent, or active labor)
- As an appropriately timed second examination to determine the woman's labor state prior to labor admission (prelabor, latent, or active labor)
- To inform management decisions related to management of labor pain
- To verify complete dilation
- To check for a prolapsed cord after spontaneous rupture of membranes if a prolapsed cord is a suspected risk (e.g., ballottable presenting part or fetal heart rate decelerations that do not resolve with usual maneuvers)

Unfortunately, digital cervical examinations rely on the clinical experience and

proprioceptive skill of the examiner. Labor care providers accurately determine actual cervical dilation in only half of all cases,[97-100] but are accurate to within 1 cm (plus or minus 1 centimeter) of actual cervical dilatation in approximately 90% of cases.[97,99,100] This imprecision is a significant shortcoming for accurately evaluating labor progress, considering that a laboring woman who is truly dilated to 4 cm may be determined to be 3 cm to 5 cm by a labor care clinician, and so on, for subsequent examinations. Thus, true cervical change over time is only *estimated* by cervical examinations. Because of this shortcoming in labor progress evaluation, it is recommended that cervical examinations be performed by the same professional whenever possible, and be interpreted conservatively so that unnecessary interventions aimed at accelerating labor are not imposed on women who are adequately progressing in labor.

Fetal Presentation, Position, and Station

Assessment of descent and internal rotation of the fetal presenting part is as important as assesment of progressive cervical dilation when monitoring labor progress. The studies that have determined normal time frames for labor progress are largely based cohorts of women whose fetus is in a vertex presentation. Most commonly the fetus enters the pelvis in an occiput transverse position and then internally rotates to occiput anterior at the level of the pelvic ischial spines. Thus, it is common to find no cervical dilation between one digital examination and the next, yet to identify progressive changes in fetal descent or internal rotation over the same interval. In contrast to slow dilation as the fetus in an occiput position descends and rotates, arrest of descent is an important finding that may indicate true cephalopelvic disproportion.

Because cephalic presentations are by far the most common, it is vital that a midwife be proficient in identifying the essential landmarks of the fetal skull (as described in the *Anatomy and Physiology During Labor and Birth* chapter). Cervical examination provides information regarding (1) fetal presentation; (2) fetal position; (3) fetal station; (4) the adaptation of the fetus to the pelvis—specifically, synclitism or asynclitism of the fetal head; (5) the presence or absence of molding; and (6) the presence or absence of caput succedaneum. Each of these elements should be determined at every cervical examination conducted during active labor.

The formation of a few millimeters of caput succedaneum is not unusual or abnormal. A small caput succedaneum may be indicative of a somewhat prolonged labor resulting from uterine inertia with weak contractions. Formation of extensive caput succedaneum, which makes the identification of fetal sutures and fontanels difficult, combined with severe amounts of molding, is usually seen only when intrauterine pressure has been great and labor prolonged. Cephalopelvic disproportion must be suspected when significant molding or overriding sutures are noted. A sizable caput may also be seen from positional pressure when the fetal position is occipitoposterior.

Recently, point-of-care ultrasound has been explored as a mechanism for diagnosing malpositions and asynclitism. Ultrasound appears promising for this use; however, treatments for specific fetal malpositions have not been the subject of sufficient research to establish effectiveness.[101] Use of differing maternal positions and movements for treatment of fetal

malpositions is reviewed in the *Complications During Labor and Birth* chapter. Care of women who have a fetus in a persistent occiput posterior position is reviewed in the *Second Stage of Labor and Birth* chapter, as interventions are rarely needed for this fetal position prior to the second stage.

Conclusion

Normal physiologic labor is an intricate interplay of physiologic and psychological forces within the woman. Caring for women during labor involves attention to three different but equally important variables: the well-being of the woman, the well-being of the fetus, and labor progress. Historical measures for evaluating labor progress have been found to be unreliable, but new indices are not yet available for all populations. Given the differences in labor patterns noted in Friedman's traditional findings and contemporary research, it is inappropriate to base clinical expectations of cervical dilation during labor on Friedman's work. That is, contemporary patterns of labor should be used, as this body of research continues to grow and evolve.

Resources

White Papers and Journal Articles
King TL, Pinger W. Evidence-based practice for intrapartum care: pearls of midwifery. *J Midwifery Womens Health*.
Romano AM, Lothian JA. Promoting, protecting, and supporting normal birth: a look at the evidence. *J Obstet Gynec*
Sakala C, Corry MP. *Evidence-Based Maternity Care: What It Is and What It Can Achieve.* New York, NY: Milbank M

Organization	Description	Webpage
American College of Nurse-Midwives (ACNM)	*Evidence-Based Practice: Pearls of Midwifery*: PowerPoint presentation and printable checklists that review the evidence for these labor management practices.	http://www.midwife.org/Evidence-Based-Practice-Pearls-of-Midwifery
	Healthy Birth Initiative resources for consumers, midwives, and maternity care institutions.	http://www.midwife.org/ACNM-Healthy-Birth-Initiative
American College of Nurse-Midwives (ACNM), Midwives Alliance North America (MANA), National Association of Certified Professional Midwives (NACPM)	Supporting healthy and normal physiologic childbirth: a consensus statement by ACNM, MANA, and NACPM, 2012.	https://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/00000000%20FINAL%20May%2018%202012%20FINAL.pdf
American College of Nurse-Midwives (ACNM), Association of Women's Health, Obstetric and	This website is a joint venture among ACNM, AWHONN, NCAP, and Lamaze. It has tools for clinicians to	www.BirthTOOLS.org

Neonatal Nurses (AWHONN), National Association of Certified Professional Midwives (NCAP), Lamaze	help them optimize the outcomes of labor safely. Content includes tools for coping in labor, promoting spontaneous onset of labor, nutrition and hydration, and physiologic second-stages, to name just a few topics covered.	
American College of Obstetricians and Gynecologists (ACOG) and Society for Maternal-Fetal Medicine	*Safe Prevention of the Primary Cesarean: Obstetric Consensus #1* reviews recommended labor management practices that can reduce the incidence of cesarean birth.	https://www.acog.org/-/media/Obstetric-Care-Consensus-Series/oc001.pdf?dmc=1&t
Childbirth Connection	Blueprint for action: steps toward a high-quality, high-value maternity care system.	http://transform.childbirthconnection.org/blueprint
National Institute for Health and Care Excellence (NICE)	Information about intrapartum care for healthy women and babies.	https://www.nice.org.uk/guidance/cg190/chapter/Recommendations
World Health Organization (WHO), Department of Reproductive Health and Research	*Care in Normal Birth: Practical Guide.*	www.who.int/reproductivehealth/publications/maternal_perinatal_health/MSM_96_24_/



References

1. American College of Nurse-Midwives, Midwives Alliance, National Association of Certified Professional Midwives. Supporting healthy and normal physiologic childbirth: a consensus statement by ACNM, MANA, and NACPM. 2012. Available at: https://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000272/Physiological%20Birth%%20FINAL%20May%2018%202012%20FINAL.pdf. Accessed November 12, 2017.
2. American College of Obstetricians and Gynecologists. Committee Opinion No. 687: approaches to limit intervention during labor and birth. *Obstet Gynecol*. 2017;129(2):e20-e28.
3. American College of Nurse-Midwives. Position statement: premature rupture of membranes at term. Updated 2012. Available at: http://midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000233/PROM%20Mar%202012.pdf. Accessed September 12, 2017.
4. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin No 172: premature rupture of membranes. *Obstet Gynecol*. 2016;128(4):e165-e177.
5. Centers for Disease Control and Prevention. Prevention of perinatal group B streptococcal disease. Revised guidelines, 2010. Available at: https://www.cdc.gov/mmwr/pdf/rr/rr5910.pdf. Accessed July 15, 2017.
6. Abdelazim IA, Makhlouf HH. Placental alpha microglobulin-1 (AmniSure® test) for detection of premature rupture of feta membranes. *Arch Gynecol Obstet*. 2012;285(4):985-989.
7. Zhang J, Landy HJ, Branch DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcomes. *Obst Gynecol*. 2010;116(6):1281-1287.
8. Friedman E. The graphic analysis of labor. *Am J Obstet Gynecol*. 1954;68(6):1568-1575.
9. Friedman EA. Primigravid labor: a graphicostatistical analysis. *Obstet Gynecol*. 1955;6(6):567-589.
10. Friedman EA. Labor in multiparas: a graphicostatistical analysis. *Obstet Gynecol*. 1956;8(6):691-703.
11. Friedman EA, Kroll BH. Computer analysis of labour progression. *J Obstet Gynaecol Br Commonw*. 1969;76(12):1075-1079.
12. Friedman EA, Kroll BH. Computer analysis of labor progression. III. Pattern variations by parity. *J Reprod Med*. 1971;6(4):179-183.
13. Friedman EA, ed. *Labor: Clinical Evaluation and Management*. 2nd ed. New York, NY: Appleton-Century-Crofts; 1978.
14. Cunningham FG, Leveno KJ, Bloom SL, et al., eds. *Williams Obstetrics*. 24th ed. New York, NY: McGraw-Hill; 2014.
15. Peisner DB, Rosen MG. Transition from latent to active labor. *Obstet Gynecol*. 1986;68(4):448-451.
16. Kilpatrick SJ, Laros RKJ. Characteristics of normal labor. *Obstet Gynecol*. 1989;74(1):85-87.
17. Zhang J, Troendle J, Mikolajczyk R, Sundaram R, Beaver J, Fraser W. The natural history of the normal first stage of labo *Obstet Gynecol*. 2010;115(4):705-710.
18. Laughon SK, Branch DW, Beaver J, Zhang J. Changes in labor patterns over 50 years. *Am J Obstet Gynecol*. 2012;206(5):419.e1-419.e9.
19. Zhang J, Troendle JF, Yancey MK. Reassessing the labor curve in nulliparous women. *Am J Obstet Gynecol*. 2002;187(4):824-828.
20. Albers LL, Schiff M, Gorwoda JG. The length of active labor in normal pregnancies. *Obstet Gynecol*. 1996;87:355-359.
21. Albers LL. The duration of labor in healthy women. *J Perinatol*. 1999;19(2):114-119.
22. Jones M, Larson E. Length of normal labor in women of Hispanic origin. *J Midwifery Womens Health*. 2003;48(1):2-9.
23. Neal JL, Lowe NK, Ahijevych KL, Patrick TE, Cabbage LA, Corwin EJ. "Active labor" duration and dilation rates among low-risk, nulliparous women with spontaneous labor onset: a systematic review. *J Midwifery Womens Health*. 2010;55(4):308-318.
24. Perl FM, Hunter DJ. What cervical dilatation rate during active labour should be considered abnormal? *Eur J Obstet Gynecol Reprod Biol*. 1992;45(2):89-92.
25. Hildingsson I, Blix E, Hegaard H, et al. How long is a normal labor? Contemporary patterns of labor and birth in a low-risk sample of 1,612 women from four Nordic countries. *Birth*. 2015;42(4):346-348.
26. McPherson JA, Tuuli M, Odibo AO, Roehl KA, Zhao Q, Cahill AG. Labor progression in teenaged women. *Am J Perinatol*. 2014;31(9):753-758.
27. Tuuli MG, Odibo AO, Cauhey AB, Roehl K, Macones GA, Cahill AG. Are there differences in the first stage of labor between black and white women? *Am J Perinatol*. 2015;32(3):233-238.
28. Kominiarek MA, Zhang J, Vanveldhuisen P, Troendle J, Beaver J, Hibbard JU. Contemporary labor patterns: the impact of maternal body mass index. *Am J Obstet Gynecol*. 2011;205(3):244.e1-244.e8.
29. Harper LM, Caughey AB, Odibo AO, Roehl KA, Zhao Q, Cahill AG. Normal progress of induced labor. *Obstet Gynecol*. 2012;119(6):1113-1118.
30. Carlson NS, Lowe NK. Intrapartum management associated with obesity in nulliparous women. *J Midwifery Womens*

Health. 2014;59(1):43-53.
31. Neal JL, Lowe NK, Schorn MN, et al. Labor dystocia: a common approach to diagnosis. *J Midwifery Womens Health*. 2015;60(5):499-509.
32. Lavender T, Hart A, Smyth RMD. Effect of partogram use on outcomes for women in spontaneous labour at term. *Cochrane Database Syst Rev*. 2013;7:CD005461.
33. Philpott RH, Castle WM. Cervicographs in the management of labour in primigravidae. I. The alert line for detecting abnormal labour. *J Obstet Gynaecol Br Commonw*. 1972;79(7):592-598.
34. Philpott RH, Castle WM. Cervicographs in the management of labour in primigravidae. II. The action line and treatment o abnormal labour. *J Obstet Gynaecol Br Commonw*. 1972;79(7):599-602.
35. Drouin P, Nasah BT, Nkounawa F. The value of the partogramme in the management of labor. *Obstet Gynecol*. 1979;53(6):741-745.
36. World Health Organization, Division of Family Health, Maternal Health and Safe Motherhood Programme. The partograpl the application of the WHO partograph in the management of labour. Report of a WHO multicentre study 1990–1991. 1994;WHO/FHE/MSM/94.4.
37. Lavender T, Alfirevic Z, Walkinshaw S. Partogram action line study: a randomised trial. *Br J Obstet Gynaecol*. 1998;105(9):976-980.
38. Lavender T, Wallymahmed AH, Walkinshaw SA. Managing labor using partograms with different action lines: a prospective study of women's views. *Birth*. 1999;26(2):89-96.
39. Pattinson RC, Howarth GR, Mdluli W, Macdonald AP, Makin JD, Funk M. Aggressive or expectant management of labou a randomised clinical trial. *BJOG*. 2003;110(5):457-461.
40. Lavender T, Alfirevic Z, Walkinshaw S. Effect of different partogram action lines on birth outcomes: a randomized controlled trial. *Obstet Gynecol*. 2006;108(2):295-302.
41. Mathews JE, Rajaratnam A, George A, Mathai M. Comparison of two world health organization partographs. *Int J Gynaecol Obstet*. 2007;96(2):147-150.
42. Orji E. Evaluating progress of labor in nulliparas and multiparas using the modified WHO partograph. *Int J Gynaecol Obstet*. 2008;102(3):249-252.
43. van Bogaert L. Revising the primigravid partogram: does it make any difference? *Arch Gynecol Obstet*. 2009;279(5):643-647.
44. Dujardin B, De Schampheleire I, Sene H, Ndiaye F. Value of the alert and action lines on the partogram. *Lancet*. 1992;339(8805):1336-1338.
45. Neal JL, Lowe NK. Physiologic partograph to improve birth safety and outcomes among low-risk, nulliparous women with spontaneous labor onset. *Med Hypotheses*. 2012;78(2):319-326.
46. Neal JL, Lowe NK, Nacht AS, Koschoreck K, Anderson J. Pilot study of physiologic partograph use among low-risk, nulliparous women with spontaneous labor onset. *J Midwifery Womens Health*. 2016;61(2):235-241.
47. Cohen WR, Friedman EA. Misguided guidelines for managing labor. *Am J Obstet Gynecol*. 2015;212(6):753.e1-753.e3.
48. Cohen WR, Friedman EA. Perils of the new labor management guidelines. *Am J Obstet Gynecol*. 2015;212(4):420-427.
49. Spong CY, Berghella V, Wenstrom KD, Mercer BM, Saade GR. Preventing the first cesarean delivery: summary of a join Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, and American College of Obstetricians and Gynecologists workshop. *Obstet Gynecol*. 2012;120(5):1181-1193.
50. Barber EL, Lundsberg LS, Belanger K, Pettker CM, Funai EF, Illuzzi JL. Indications contributing to the increasing cesarean delivery rate. *Obstet Gynecol*. 2011;118:29-38.
51. Tracy SK, Sullivan E, Wang YA, Black D, Tracy M. Birth outcomes associated with interventions in labour amongst low risk women: a population-based study. *Women Birth*. 2007;20(2):41-48.
52. Walsh J, Foley M, O'Herlihy C. Dystocia correlates with body mass index in both spontaneous and induced nulliparous labors. *J Matern Fetal Neonatal Med*. 2011;24(6):817-821.
53. Selin L, Almstrom E, Wallin G, Berg M. Use and abuse of oxytocin for augmentation of labor. *Acta Obstet Gynecol Scand*. 2009;88(12):1352-1357.
54. Clark SL, Simpson KR, Knox GE, Garite TJ. Oxytocin: new perspectives on an old drug. *Am J Obstet Gynecol*. 2009;200(1):35.e1-35.e6.
55. Clark SL, Belfort MA, Dildy GA, Meyers JA. Reducing obstetric litigation through alterations in practice patterns. *Obstet Gynecol*. 2008;112(6):1279-1283.
56. Zhang J, Troendle J, Reddy UM, et al. Contemporary cesarean delivery practice in the United States. *Am J Obstet Gynecol*. 2010;203(4):326.e1-326.e10.
57. Boyle A, Reddy UM, Landy HJ, Huang C, Driggers RW, Laughon SK. Primary cesarean delivery in the United States. *Obstet Gynecol*. 2013;122(1):33-40.
58. Martin JA, Hamilton BE, Osterman MJK, Driscoll AK, Mathews TJ. Births: final data for 2015. *Natl Vital Stat Rep*. 2017;66(1):1-69.
59. World Health Organization, Department of Reproductive Health and Research. WHO statement on caesarean section

rates. 2015;WHO/RHR/15.02.
60. World Health Organization. Appropriate technology for birth. *Lancet*. 1985;326(8452):436-437.
61. Villar J, Valladares E, Wojdyla D, et al. Caesarean delivery rates and pregnancy outcomes: the 2005 WHO global survey on maternal and perinatal health in Latin America. *Lancet*. 2006;367(9525):1819-1829.
62. Villar J, Carroli G, Zavaleta N, et al. Maternal and neonatal individual risks and benefits associated with caesarean delivery multicentre prospective study. *BMJ*. 2007;335(7628):1025-1025.
63. Althabe F, Sosa C, Belizán JM, Gibbons L, Jacquerioz F, Bergel E. Cesarean section rates and maternal and neonatal mortality in low-, medium-, and high-income countries: an ecological study. *Birth*. 2006;33(4):270-277.
64. Curtin SC, Gregory KD, Korst LM, Uddin SF. Maternal morbidity for vaginal and cesarean deliveries, according to previous cesarean history: new data from the birth certificate, 2013. *Natl Vital Stat Rep*. 2015;64(4):1-13, back cover.
65. Neal JL, Lowe NK, Phillippi JC, et al. Likelihood of cesarean delivery after applying leading active labor diagnostic guidelines. *Birth*. 2017;44(2):128-136.
66. Bailit JL, Dierker L, Blanchard MH, Mercer BM. Outcomes of women presenting in active versus latent phase of spontaneous labor. *Obstet Gynecol*. 2005;105(1):77-79.
67. Wood AM, Frey HA, Tuuli MG, et al. Optimal cervical dilation in spontaneously laboring women. *Am J Perinatol*. 2016;33(2):188-194.
68. McNiven PS, Williams JI, Hodnett E, Kaufman K, Hannah ME. An early labor assessment program: a randomized, controlled trial. *Birth*. 1998;25(1):5-10.
69. Incerti M, Locatelli A, Ghidini A, Ciriello E, Consonni S, Pezzullo JC. Variability in rate of cervical dilation in nulliparous women at term. *Birth*. 2011;38(1):30-35.
70. Impey L, Hobson J, O'Herlihy C. Graphic analysis of actively managed labor: prospective computation of labor progress in 500 consecutive nulliparous women in spontaneous labor at term. *Am J Obstet Gynecol*. 2000;183(2):438-443.
71. Rahnama P, Ziaei S, Faghihzadeh S. Impact of early admission in labor on method of delivery. *Int J Gynaecol Obstet*. 2006;92(3):217-220.
72. Mikolajczyk R, Zhang J, Chan L, Grewal J. Early versus late admission to labor/delivery, labor progress and risk of caesarean section in nulliparous women. *Am J Obstet Gynecol*. 2008;199(6 suppl A):S49.
73. Gifford DS, Morton SC, Fiske M, Keesey J, Keeler E, Kahn KL. Lack of progress in labor as a reason for cesarean. *Obstet Gynecol*. 2000;95(4):589-595.
74. Neal JL, Lowe NK, Patrick TE, Cabbage LA, Corwin EJ. What is the slowest-yet-normal cervical dilation rate among nulliparous women with spontaneous labor onset? *J Obstet Gynecol Neonatal Nurs*. 2010;39(4):361-369.
75. Institute for Safe Medication Practices. ISMP list of high-alert medications in acute care settings. Updated 2017. Available at: http://www.ismp.org/Tools/institutionalhighAlert.asp. Accessed September 12, 2017.
76. Bugg GJ, Siddiqui F, Thornton JG. Oxytocin versus no treatment or delayed treatment for slow progress in the first stage of spontaneous labour. *Cochrane Database Syst Rev*. 2013;6:CD007123.
77. Smyth RM, Markham C, Dowswell T. Amniotomy for shortening spontaneous labour. *Cochrane Database Syst Rev*. 2013;6:CD006167.
78. Balayla PS. Inadequate prenatal care utilization and risks of infant mortality and poor birth outcome: a retrospective analysis of 28,729,765 U.S. deliveries over 8 years. *Am J Perinatol*. 2012;29(10):787-794.
79. Devane D, Lalor JG, Daly S, McGuire W, Cuthbert A, Smith V. Cardiotocography versus intermittent auscultation of fetal heart on admission to labour ward for assessment of fetal wellbeing. *Cochrane Database Syst Rev*. 2017;1:CD005122.
80. Intermittent auscultation for intrapartum fetal heart rate surveillance: American College of Nurse-Midwives. *J Midwifery Womens Health*. 2015;60(5):626-632.
81. American College of Obstetricians and Gynecologists. Practice Bulletin No. 116: management of intrapartum fetal heart rate tracings. *Obstet Gynecol*. 2010;116:1232-1240.
82. Hodnett ED, Gates S, Hofmeyr GJ, Sakala C. Continuous support for women during childbirth. *Cochrane Database Syst Rev*. 2013;7:CD003766.
83. Lawrence A, Lewis L, Hofmeyr GJ, Styles C. Maternal positions and mobility during first stage labour. *Cochrane Database Syst Rev*. 2013;10:CD003934.
84. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anesthesia. *Am J Obstet Gynecol*. 1946;52:191.
85. Garite TJ, Weeks J, Peters-Phair K, Pattillo C, Brewster WR. A randomized controlled trial of the effect of increased intravenous hydration on the course of labor in nulliparous women. *Am J Obstet Gynecol*. 2000;183(6):1544-1548.
86. Eslamian L, Marsoosi V, Pakneeyat Y. Increased intravenous fluid intake and the course of labor in nulliparous women. *Int J Gynaecol Obstet*. 2006;93(2):102-105.
87. Coco A, Derksen-Schrock A, Coco K, Raff T, Horst M, Hussar E. A randomized trial of increased intravenous hydration in labor when oral fluid is unrestricted. *Fam Med*. 2010;42(1):52-56.
88. Kavitha A, Chacko KP, Thomas E, et al. A randomized controlled trial to study the effect of IV hydration on the duration of labor in nulliparous women. *Arch Gynecol Obstet*. 2012;285(2):343-346.

89. Foulkes J, Dumoulin JG. The effects of ketonuria in labour. *Br J Clin Pract*. 1985;39(2):59-62.
90. Watanabe T, Minakami H, Sakata Y, et al. Effect of labor on maternal dehydration, starvation, coagulation, and fibrinolysis *J Perinat Med*. 2001;29(6):528-534.
91. Singata M, Tranmer J, Gyte GM. Restricting oral fluid and food intake during labour. *Cochrane Database Syst Rev*. 2013;8:CD003930.
92. Macones GA, Cahill A, Stamilio DM, Odibo AO. The efficacy of early amniotomy in nulliparous labor induction: a randomized controlled trial. *Am J Obstet Gynecol*. 2012;207(5):403.e1-403.e5.
93. Rouse DJ, McCullough C, Wren AL, Owen J, Hauth JC. Active-phase labor arrest: a randomized trial of chorioamnion management. *Obstet Gynecol*. 1994;83(6):937-940.
94. Wei S, Wo BL, Qi HP, et al. Early amniotomy and early oxytocin for prevention of, or therapy for, delay in first stage spontaneous labour compared with routine care. *Cochrane Database Syst Rev*. 2013;8:CD006794.
95. Freeman RK, Garite TJ, Nageotte MP, Miller LA. *Fetal Heart Rate Monitoring*. 4th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2012.
96. Krapohl AJ, Myers GG, Caldeyro-Barcia R. Uterine contractions in spontaneous labor: a quantitative study. *Am J Obstet Gynecol*. 1970;106(3):378-387.
97. Buchmann EJ, Libhaber E. Accuracy of cervical assessment in the active phase of labour. *BJOG*. 2007;114(7):833-837.
98. Huhn KA, Brost BC. Accuracy of simulated cervical dilation and effacement measurements among practitioners. *Am J Obstet Gynecol*. 2004;191(5):1797-1799.
99. Phelps JY, Higby K, Smyth MH, Ward JA, Arredondo F, Mayer AR. Accuracy and intraobserver variability of simulated cervical dilatation measurements. *Am J Obstet Gynecol*. 1995;173(3):942-945.
100. Tuffnell DJ, Bryce F, Johnson N, Lilford RJ. Simulation of cervical changes in labour: reproducibility of expert assessment. *Lancet*. 1989;2(8671):1089-1090.
101. Bellussi F, Ghi T, Youssef A, et al. The use of intrapartum ultrasound to diagnose malpositions and cephalic malpresentations. *Am J Obstet Gynecol*. 2017 Dec;217(6):633-641.

APPENDIX

25A

Evaluation and Diagnosis of Ruptured Membranes

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When evaluating a woman whose history suggests rupture of membranes, the differential diagnoses include urinary incontinence, vaginal or cervical discharge, semen, and (rarely) rupture of the chorion alone.

Several laboratory tests can confirm or support a diagnosis of rupture of membranes (ROM), including the arborization test (fern test), pH testing (nitrazine test), ultrasound assessment of amniotic fluid volume, and testing for the presence of placental alpha microglobulin-1 (PAMG-1) or fetal fibronectin in vaginal secretions. Several of these tests can be directly performed on samples of fluid obtained during the physical examination.

Tests for and Classic Signs of Rupture of Membranes

1. Observation of fluid coming from the cervical os is diagnostic for ROM.
2. The *fern test* is a classic method of assessing for ROM and is considered diagnostic in many settings when the classic pattern of ferning is clearly seen (**Figure 25A-1**). During a sterile speculum examination, a sterile cotton swab is used to obtain a specimen of the fluid from the posterior vaginal fornix. Care must be taken not to touch the cervical os to avoid collecting cervical mucus. The specimen is spread thinly onto a microscope slide and allowed to dry thoroughly. The slide is then inspected without using a cover slip using a microscope at 10× power for a fern-like pattern (arborization) caused by crystallization due to the high sodium chloride and protein concentrations in amniotic fluid.

   The sensitivity and specificity of the fern test among nonlaboring women are 51.4% and 70.8%, respectively; for women in labor, these results improve to 98% and 88.2%, respectively.[1] Cervical mucus and semen can also show fern-like patterns, which may

cause a false-positive result. The results of a fern test must be viewed within the context of the history and physical examination and may best be viewed as a supportive test rather than a conclusive one if active leaking from the cervical os is not visible.

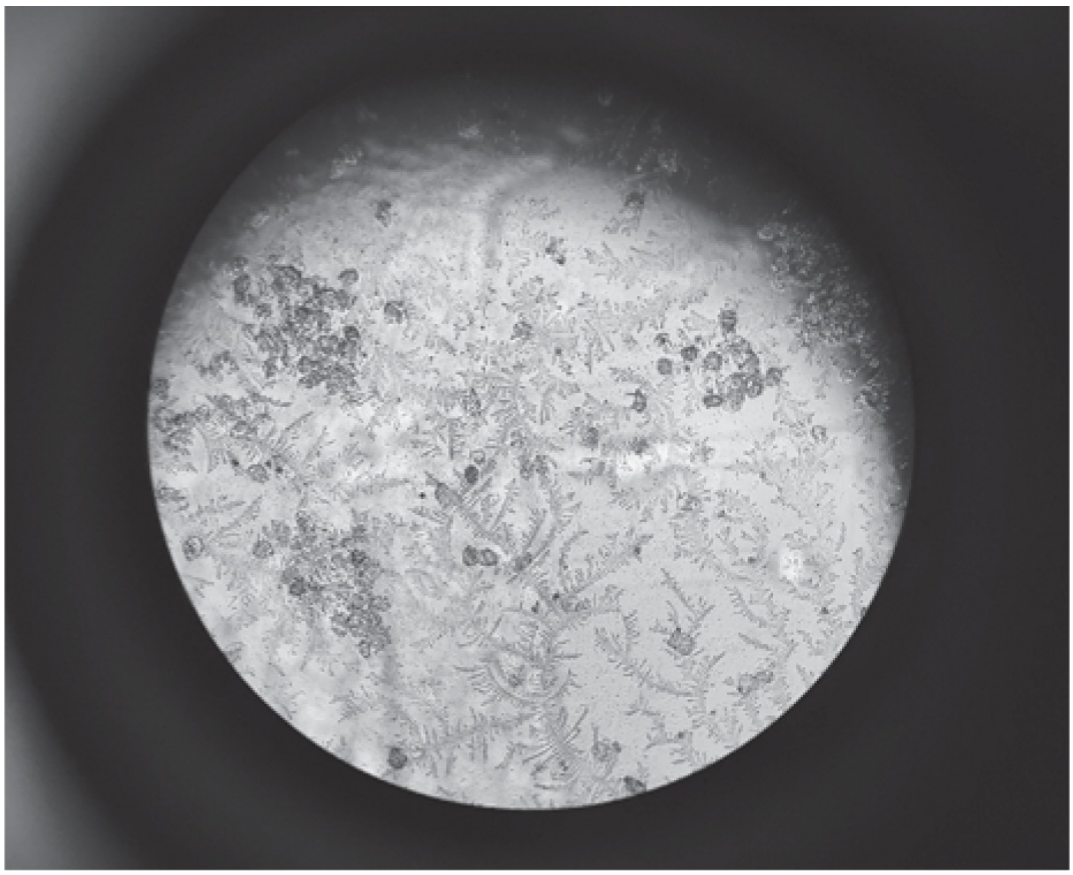


Figure 25A-1 Fern pattern of amniotic fluid.

Courtesy of Philapeter. Creative Commons license available at https://creativecommons.org/licenses/by-sa/4.0/deed.en.

3. The *nitrazine test* uses limited-range pH paper or a commercially prepared swab to detect the rise in pH in vaginal discharge associated with the presence of amniotic fluid. The normal pH of the vagina of most women is acidic (approximately 4.5), whereas amniotic fluid is neutral to slightly alkaline (7.0–7.5). The mustard-gold nitrazine will turn dark blue in the presence of alkaline material. When using nitrazine paper, the procedure is to collect a sample of fluid from the posterior fornix with a sterile cotton swab and then touch the nitrazine paper with the saturated swab. A well-saturated cotton swab can be used to prepare a slide for the fern test, then touched to the nitrazine paper. Alternatively, the paper can be touched in the fluid collected at the tip of the sterile speculum when it is withdrawn from the vagina. Commercially prepared nitrazine swabs are available and are accompanied by manufacturer instructions that must be carefully followed.

   The sensitivity and specificity of the nitrazine method in confirming ROM are approximately 87% and 81%, respectively.[2] False-positive results can occur in the

presence of vaginal infections, blood, semen, or other alkaline substances, while false-negative results can be caused by a "high" or minimal leak of amniotic fluid.

4. Pooling of fluid in the posterior fornix of the vagina is a helpful sign, but it is not diagnostic for ROM without confirmation via ferning or nitrazine testing.
5. Ultrasound quantification may also be used, although a woman can have ruptured membranes and still have a normal amount of amniotic fluid, especially if the membranes are only leaking. Ultrasound documentation of oligohydramnios does not confirm ROM but may be helpful in making a management plan.

Diagnosis of Rupture of Membranes

The diagnosis of ruptured membranes is typically made via the constellation of history, physical examination, and positive testing. When two or more tests are positive, the accuracy of the diagnosis is approximately 93%.[3] This method is generally considered acceptable for women at term. For women who are preterm, accurate diagnosis is more critical. Physician consultation is needed if the diagnosis is inconclusive for a woman who is at a preterm gestation.

Evaluation for Ruptured Membranes

History

1. Inquire about the time, amount, color, consistency, odor, and pattern of leaking (e.g., large gush, continued trickling).
   a. These data are especially important for development of a management plan because the length of time from rupture of membranes to delivery is directly correlated with risk of maternal–fetal infection. The characteristic of the fluid can reveal clues to fetal well-being.
   b. ROM typically will cause a large gush of fluid, followed by a continuous watery discharge necessitating use of sanitary pads or even washcloths or towels.
   c. In some instances of ruptured membranes, the only symptom the woman may notice is a feeling of moistness on her undergarments from a small, continuous discharge. Assessing the woman's ability to control the leakage with contraction of the pelvic floor muscles (Kegel) helps to differentiate PROM from urinary incontinence.
2. Inquire about any recent fever, abdominal pain, vaginal bleeding, abnormal discharge, urine symptoms, and last intercourse.
   a. Semen expelled from the vagina can sometimes be mistaken for amniotic fluid.
3. Inquire about signs of labor: contractions, bloody show, fetal movement, recent cervical assessments, or intercourse.
4. Confirm pregnancy dating (this is especially important if less than 37 weeks' gestation).
5. Review the prenatal record for past obstetric history, prenatal issues, or current medical

problems.

Physical Examination

The earlier an examination is performed after the rupture of membranes occurs, the easier it is to diagnose ruptured membranes. When more than 6 to 12 hours passes, many of the diagnostic observations become unreliable because of lack of fluid.

1. Measure temperature, pulse, respirations, and blood pressure.
2. Perform heart and lung auscultation.
3. Palpate the abdomen for tenderness.
4. Perform Leopold's maneuvers to assess fetal position, estimated fetal weight, and presenting part.
    a. Ultrasound confirmation of the presenting part may be required.
5. Perform fetal assessment with Doppler or electronic fetal heart rate monitoring per institutional or practice guidelines. Continuous electronic fetal heart rate monitoring is required for women who are between 24 to 37 weeks' gestation.
6. Perform a sterile speculum examination.
    a. Note the color, consistency, and amount of any fluid leaking from the vaginal introitus.
    b. As the speculum is carefully inserted, be alert for any evidence of prolapsed cord, bulging forebag, or protruding fetal parts.
    c. Visualize the cervical os and note any pooling of fluid in the vaginal vault or fluid leaking directly from the os.
        i. Normal amniotic fluid can be clear, straw colored, or cloudy. Flecks of white or creamy vernix may be noted in the amniotic fluid of preterm or near-term infants. Dark yellow or green fluid indicates the presence of meconium in the amniotic fluid. Meconium-stained fluid increases the risk for chorioamnionitis and can be an indication of fetal compromise.
        ii. Amniotic fluid has a distinct musty odor, which differentiates it from urine, while foul-smelling fluid can be an indicator of infection.
        iii. If there is no visible fluid leaking from the os:
            1. Have the woman perform a Valsalva maneuver or cough.
            2. Alternatively, consider having an assistant apply gentle fundal pressure or gently elevate the presenting part abdominally to allow fluid to pass by the presenting part and flow through the cervical os.
            3. Another option is to have the woman remain semi-reclining for 30 to 60 minutes and then repeat the sterile speculum examination.
    d. Obtain sterile swab specimens of any fluid or discharge seen, avoiding the cervical mucus.
        i. Using a sterile swab, collect a sample of fluid for 10–15 seconds from the vaginal pool at the posterior fornix or along the vaginal wall. Avoid the cervix.
        ii. If a nitrazine swab is being used, the color change can be read directly from the

swab.

iii. If nitrazine paper is being used, apply the swab to pH paper before proceeding. A pH of 6.5 or higher is suggestive of amniotic fluid rupture.

iv. Immediately roll the swab across a dry, clean slide to create a thin film. Thick specimens may obscure ferning. Set aside for 10 minutes.

1. Ferning is based on crystallization of the sodium chloride in amniotic fluid. This occurs as the liquid evaporates, so false-negative results are possible if the slide is examined before it is completely dry.

v. Obtain a wet mount of any discharge.

e. Obtain a specimen for gonorrhea and chlamydia culture per protocols as required.

f. Obtain GBS culture if the woman's status is unknown or if it has been more than 5 weeks since the last GBS result.

g. Visualize the cervix for dilation and length/effacement.

h. *Do not* perform a digital vaginal/cervical examination unless signs of active labor are present.

Microscopy Evaluation

1. Examine the dry slide under low power (10×) for the presence of ferning (see **Figure 26A-1**).
2. Examine the wet mount slide for the presence of yeast, bacterial vaginosis, or trichomoniasis.

References

1. de Haan HH, Offermans PM, Smits F, Schouten HJ, Peeters LL. Value of the fern test to confirm or reject the diagnosis of ruptured membranes is modest in nonlaboring women presenting with nonspecific vaginal fluid loss. *Am J Perinatol*. 1994;11(1):46-50.
2. Abdelazim IA, Makhlouf HH. Placental alpha microglobulin-1 (AmniSure® test) for detection of premature rupture of fetal membranes. *Arch Gynecol Obstet*. 2012;285(4):985-989.
3. Canavan TP, Simhan HN, Caritis S. An evidence-based approach to the evaluation and treatment of premature rupture of membranes: Part I. *Obstet Gynecol Surv*. 2004;59(9):669-677.

CHAPTER

26

Fetal Assessment During Labor

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Introduction

The fetal assessment techniques used during labor are intended to identify the fetus experiencing acute impairment of gas exchange. None of the current methods that monitor the fetal heart directly measures fetal oxygenation or acid–base balance in fetal tissue. Therefore, it is important to know the value of the fetal assessment technologies that are in use and the extent of their capabilities. This chapter reviews fetal acid–base physiology, continuous electronic fetal heart rate monitoring (EFM), intermittent auscultation techniques, umbilical cord gas assessments that reflect fetal acid–base balance immediately prior to birth, and midwifery management of fetal heart rate (FHR) patterns.

To date, no single indicator has been identified that has a high positive predictive value for detecting clinically significant fetal acidemia. It may be that the threshold for permanent damage differs among individual fetuses; perhaps today's instruments are not individually or collectively sensitive enough; or perhaps both of these considerations play a role.[1,2]

History of Fetal Heart Rate Monitoring

The electronic fetal monitor was introduced as an instrument for monitoring women with high-risk pregnancies in the 1960s. Clinicians originally anticipated that EFM would be a screening test that would identify fetal asphyxia early enough to allow interventions before fetal damage occurred. Unfortunately, continuous EFM was rapidly adopted in clinical practice before adequate research assessing the relationship between FHR characteristics and newborn outcomes had been conducted.

The first observational studies of FHR monitoring were historical in nature. These studies compared newborn outcomes in settings before the introduction of FHR monitoring to those after widespread adoption of FHR monitoring. Those studies documented a significant decrease in the incidence of intrapartum stillbirth in women who were continuously monitored during labor—an outcome that remains one of the key benefits of EFM.[3]

No randomized controlled trials (RCTs) have compared continuous EFM to no monitoring. RCTs conducted in many developed nations, comparing continuous EFM to intermittent auscultation of the fetal heart with a Pinard stethoscope or a handheld Doppler device were performed in the 1990s. Most of these trials included one-to-one nursing, and several included women at high risk for adverse outcomes. Neither the RCTs nor the subsequent meta-analyses found that EFM improved outcomes when compared to intermittent auscultation (IA) with regard to Apgar scores, cerebral palsy, or perinatal mortality.[4,5] However, continuous EFM was associated with an approximately 50% increase in rates of cesarean section (relative risk [RR], 1.63; 95% confidence interval [CI], 1.29–2.07) and a slight increase in operative vaginal birth (RR, 1.15; 95% CI, 1.01–1.33).[6]

The largest RCT, which was performed in Dublin, Ireland, found EFM resulted in a 50% decrease in early neonatal seizures (1.8% in the EFM group versus 4.1% in the IA group). Newborn seizures were used as an indicator of long-term neurologic dysfunction and perinatal mortality. When the cohort of newborns in this trial who had seizures and survived were reexamined at age 4 years, those in the EFM group and those in the IA group had equal proportions of cerebral palsy—a finding that suggests EFM was not effective in decreasing the rate of cerebral palsy.[7,8] However, it is not possible to draw clinically relevant conclusions about the effect of EFM from these data and the small number of affected children. Nonetheless, EFM may have a protective effect in decreasing the incidence of early newborn seizures, although the long-term implications are unclear.

The negative findings of the meta-analysis of the RCTs evaluating EFM resulted in many clinicians concluding that EFM has no clinical value. However, the RCTs' failure to detect a benefit of EFM occurred secondary to several methodologic problems; thus, the results are not considered conclusive or reliable for practice today.[9] For example, when these studies were conducted, cerebral palsy was believed to result from intrapartum asphyxia. Subsequent studies have shown that most children with cerebral palsy acquire a brain injury in the prenatal or neonatal period, so intrapartum interventions such as EFM are not likely to prevent this disorder. The marked increase in cesarean sections noted in the EFM-recipient groups may be at least partially attributed to the criteria used to define fetal distress in the RCTs. Although it was known at the time that transient decreases in oxygenation cause late decelerations, the

importance of FHR variability as the best indicator of cerebral oxygenation was not recognized. Some of the FHR patterns (e.g., late decelerations with moderate FHR variability) that signaled fetal distress and resulted in cesarean section in the trials are known today to be poorly predictive of fetal acidemia. Finally, perinatal mortality is a rare enough event that more than 37,000 participants in an RCT would be needed to detect a change in perinatal mortality rates.

In short, the RCTs did not answer the question of whether EFM improves neonatal outcomes because they had the wrong outcome measures, they did not monitor for FHR patterns that are known today to indicate fetal compromise, and the studies were not large enough to detect meaningful changes in perinatal mortality. Thus, it is as inappropriate to claim EFM has no value as it is to advocate for universal EFM.

One of the difficulties encountered by researchers investigating EFM, especially in the older studies, was lack of a standardized lexicon. Moreover, clinicians often had difficulty communicating with one another without a common language. In 1997, the National Institute of Child Health and Human Development (NICHD) convened an expert panel charged with developing a standard terminology for interpreting FHR patterns. This panel created mutually exclusive definitions for each FHR characteristic that is used in clinical practice today; the same definitions have been used in FHR research over the last decade (**Table 26-1**).[10]

Table 26-1 Terminology for Fetal Heart Rate Characteristics

Term	Definition
Baseline rate	Mean FHR rounded to increments of 5 bpm during a 10-minute segment excluding periodic or episodic changes, periods of marked variability, and segments of baseline that differ by > 25 bpm. Duration must be ≥ 2 minutes.
Bradycardia	Baseline rate < 110 bpm.
Tachycardia	Baseline rate > 160 bpm.
Variability	Fluctuations in the baseline FHR ≥ 2 cycles/min.
Absent variability	Amplitude from peak to trough undetectable.
Minimal variability	Amplitude from peak to trough > undetectable and ≤ 5 bpm.
Moderate variability	Amplitude from peak to trough 6–25 bpm.
Marked variability	Amplitude from peak to trough > 25 bpm.
Acceleration	Visually apparent abrupt increase (onset to peak < 30 seconds) of FHR above baseline. Peak ≥ 15 bpm. Duration ≥ 15 bpm and < 2 minutes. In gestations < 32 weeks, peak ≥ 10 bpm above baseline and duration ≥ 10 seconds is an acceleration.
Prolonged acceleration	Acceleration ≥ 2 minutes and < 10 minutes' duration. An acceleration ≥ 10 minutes is a baseline change.
Early deceleration	Visually apparent *gradual* decrease (onset of deceleration to nadir ≥ 30 seconds) of FHR below baseline. Return to baseline associated with a uterine contraction. The nadir of the deceleration occurs at the same time as the peak of the contraction. Generally, the onset, nadir, and recovery of the deceleration occur at the same time as the onset, peak, and recovery of the contraction, respectively.

Late deceleration	Visually apparent *gradual* decrease (onset of deceleration to nadir ≥ 30 seconds) of FHR below baseline. Return to baseline associated with a uterine contraction. The nadir of the deceleration occurs *after* the peak of the contraction. Generally, the onset, nadir, and recovery of the deceleration occur after the onset, peak, and recovery of the contraction, respectively.
Variable deceleration	Visually apparent *abrupt* decrease (onset of deceleration to nadir < 30 seconds) in FHR below baseline. Decrease ≥ 15 bpm below baseline. Duration ≥ 15 seconds and < 2 minutes from onset to return to baseline.
Prolonged deceleration	Visually apparent decrease in FHR below baseline. Decrease ≥ 15 bpm below baseline. Duration ≥ 2 minutes but < 10 minutes from onset to return to baseline.

Abbreviations: bpm, beats per minute; FHR, fetal heart rate.

Based on National Institute of Child Health and Human Development Research Planning Workshop. Electronic fetal heart rate monitoring: research guidelines for interpretation. *Am J Obstet Gynecol.* 1997;17:1385-1390; *JOGNN.* 1997;26:635-640.[10]

In 2008, an NICHD expert panel met again to reevaluate the prior decade of research. The 1997 terminology was reaffirmed and new definitions for uterine tachysystole were devised. In addition, the panel recommended a 3-tier system for interpreting FHR patterns as normal (Category I), indeterminate (Category II), or abnormal (Category III); they remain the three categories used in clinical practice today (**Table 26-2**).[11]

Table 26-2 Fetal Heart Rate Interpretive Categories

Category	FHR Patterns Assigned to This Category
Category I (Normal)	
Baseline rate 110–160 bpm, moderate variability, and absence of late or variable decelerations must *all* be present.	Baseline rate: 110–160 bpm Baseline FHR variability: moderate Late or variable decelerations: absent Early decelerations: present or absent Accelerations: present or absent
Category II (Indeterminate)	
All FHR tracings not categorized as Category I or Category III. Includes *any* of these patterns.	Baseline rate: Bradycardia not accompanied by absent baseline variability Tachycardia Baseline FHR variability: Minimal baseline variability Absent baseline variability Marked baseline variability Accelerations: Absence of induced accelerations after fetal stimulations Periodic or episodic decelerations: Recurrent variable decelerations accompanied by minimal or moderate baseline variability Prolonged deceleration > 2 minutes but < 10 minutes Recurrent late decelerations with moderate baseline variability

	Variable decelerations with other characteristics, such as slow return to baseline, “overshoots,” or “shoulders”
Category III (Abnormal)	
Includes *either* pattern.	Absent baseline FHR variability and any of the following: Recurrent late decelerations Recurrent variable decelerations Bradycardia Sinusoidal pattern

Abbreviations: bpm, beats per minute; FHR, fetal heart rate.

Based on Macones GA, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development Research Workshop report on electronic fetal heart rate monitoring. *Obstet Gynecol.* 2008;112:661-666; *JOGNN.* 2008;37:510-515.[11]

Despite the controversy about the value of continuous EFM, this form of fetal surveillance is used in almost all hospital birthing units. Many possible reasons exist for this enigma. It has been suggested that continuous EFM reassures women and caregivers alike, whereas the risks associated with cesarean section births are not considered as significant in comparison. In addition, FHR monitoring allows for remote surveillance without the need for a continuous bedside provider. Thus, it is important that midwives are familiar with the current evidence regarding the relationship between FHR patterns and fetal acidemia, was well as the benefits and limitations of this technology. First, a brief review of fetal oxygenation and acid–base physiology is in order.

Fetal Physiology

The aspects of fetal physiology that affect transport of oxygen and nutrients between the mother and the fetus include uteroplacental circulation, gas exchange across the placenta, factors that affect the FHR, and the fetal response to hypoxia, which is markedly different than the adult response.

Uteroplacental Circulation and Gas Exchange

Optimal transfer of oxygen, carbon dioxide, and nutrients from the maternal circulation to the fetal circulation can occur only when the following five components function well: (1) adequate maternal blood flow into the intervillous space, (2) a large enough placental area to facilitate gas and nutrient exchange, (3) efficient diffusion of gases and nutrients across the three membranes that separate the maternal and fetal circulations, (4) unimpaired umbilical vein circulation into the fetus, and (5) adequate oxygen transport capacity in the fetus.

In women without preexisting medical disorders, placental growth is usually adequate, and gas and nutrient exchange across the placental membranes is efficient. Furthermore, the fetus has enhanced oxygen-carrying capacity relative to an adult because of several physiologic mechanisms:

- Fetal hemoglobin binds to oxygen more easily than does adult hemoglobin, which favors transfer of oxygen from the maternal circulation to the fetal circulation.
- The fetus has more hemoglobin in circulation than do adults (the average fetal hematocrit ranges from 43% to 63%).
- The fetus has a higher cardiac output and heart rate for an overall faster circulation.
- Organs that need oxygen such as the heart and brain are over-perfused in the fetus.

These physiologic differences allow the fetus to survive well at a PO_2 of approximately 35 mm Hg, which is approximately the same as the maternal venous system. The same mechanisms also provide a buffer during the intermittent decreases in maternal blood flow into the intervillous space that occur during uterine contractions. Therefore, the components of uteroplacental function most likely to adversely affect fetal gas exchange are decreased perfusion to the placenta (e.g., uterine tachysystole, maternal supine positioning) and decreased umbilical vessel perfusion (e.g., cord compression).

Approximately 500 to 900 mL of maternal blood flows to the uterus each minute at term.[12,13] The majority of this blood (70–90%) enters the maternal spiral arteries, which run perpendicular through the uterine endometrium. During active labor, uterine contractions constrict these arteries, which temporarily interrupts blood flow in and out of the intervillous space.

As reviewed in the *Anatomy and Physiology of Pregnancy* chapter, the uterine spiral arteries become deinnervated during pregnancy as their endothelial linings are replaced with trophoblastic tissue. As a consequence, the arteries become maximally dilated so as to facilitate blood flow into the intervillous space.[14] In addition, the arteries lose their ability to

constrict if pressure in the vessel drops.

The clinical significance of this placental structure is that maternal hypotension, hypertension, and uterine contractions can all decrease blood flow into the intervillous space. Very little can be done physiologically to increase uterine blood flow if it is needed (**Figure 26-1**). Maximal arterial flow to the spiral arteries can be supported via use of side-lying positions to prevent compression of the inferior vena cava and administration of tocolytics that stop uterine contractions for a short time.

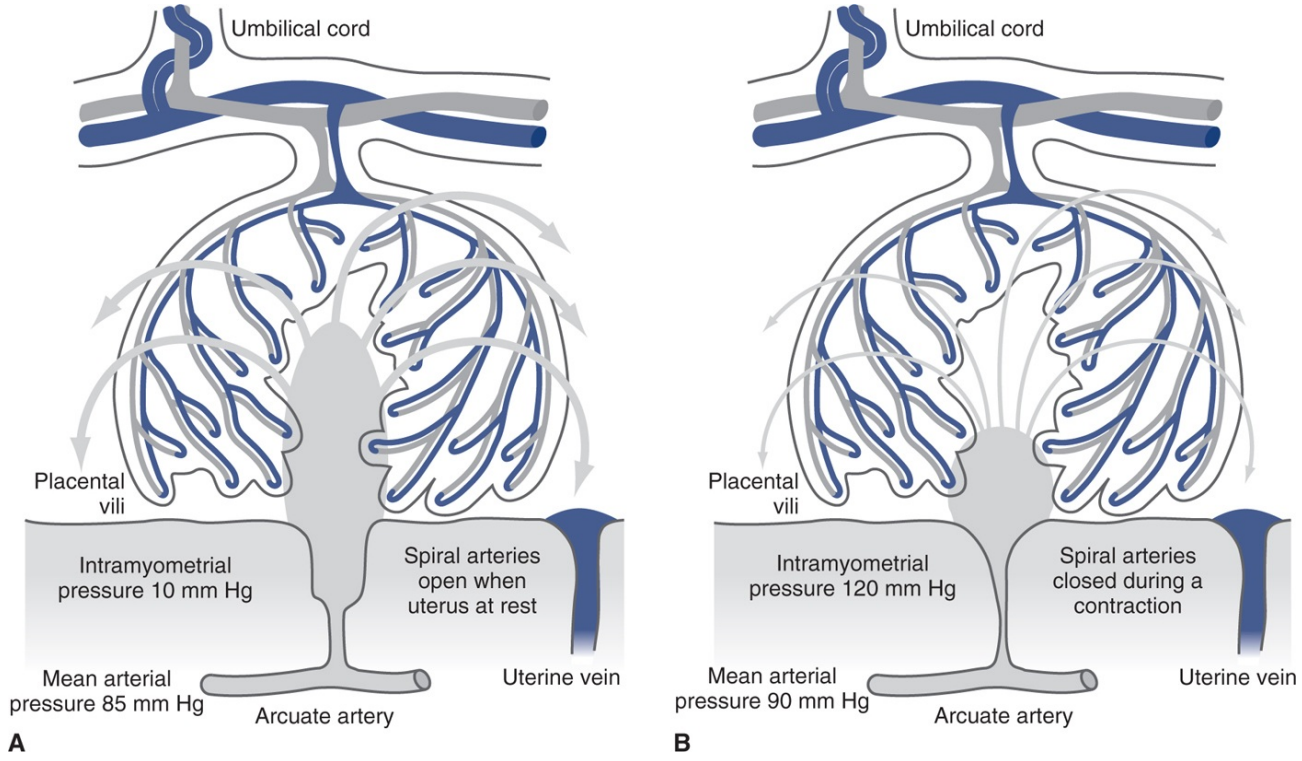


Figure 26-1 Effect of uterine contraction on blood flow into the intervillous space in the placenta. **A.** Blood flow into the intervillous space between uterine contractions. **B.** Blood flow into the intervillous space stops or is reduced during myometrial contraction.

Factors That Control the Fetal Heart Rate

The fetal heart rate is controlled by sympathetic, parasympathetic, chemoreceptor, baroreceptor, and central nervous system (CNS) inputs.

Sympathetic and Parasympathetic Input

The average baseline heart rate in the normal term fetus before labor is 140 bpm (range: 120–160 bpm). Early in pregnancy, the FHR is even higher, albeit not significantly higher. The FHR of a preterm fetus is likely to be on the higher end of this range.

The sympathetic fibers that innervate the myocardium respond to catecholamine stimulation by increasing the FHR. As the parasympathetic nervous system matures during the second trimester, parasympathetic input—which is mediated via the vagus nerve—becomes dominant over sympathetic stimulation; in turn, the baseline heart rate gradually slows.

The fetal heart is similar to the adult heart in that it has intrinsic pacemaker activity. The sinoatrial node, in the right atrium, has the highest intrinsic rate; thus, the sinoatrial node sets the pace for the fetal heart beats.[15(p33)] The vagus nerve originates in the medulla oblongata and terminates in the sinoatrial node of the fetal heart, where vagal stimulation changes the FHR by causing the interval between successive beats to vary.[16]

Chemoreceptors and Baroreceptors

Chemoreceptors found in the aortic arch and central nervous system are sensitive to changes in oxygen and carbon dioxide content within the blood. Increased carbon dioxide causes the chemoreceptors to signal the medulla oblongata, which stimulates the vagus nerve and slows the FHR. In the adult, central chemoreceptor detection of hypercarbia produces tachycardia, whereas peripheral chemoreceptor detection of hypercarbia produces bradycardia. The responses to chemoreceptor stimulation in the fetus are not totally understood, but the result of transient hypercarbia is clearly a slowing of the FHR. Baroreceptors in the aortic and carotic arches rapidly detect changes in blood pressure. When blood pressure rises, a quick reflex occurs (via the vagal nerve) to slow the fetal heart rate (**Figure 26-2**).

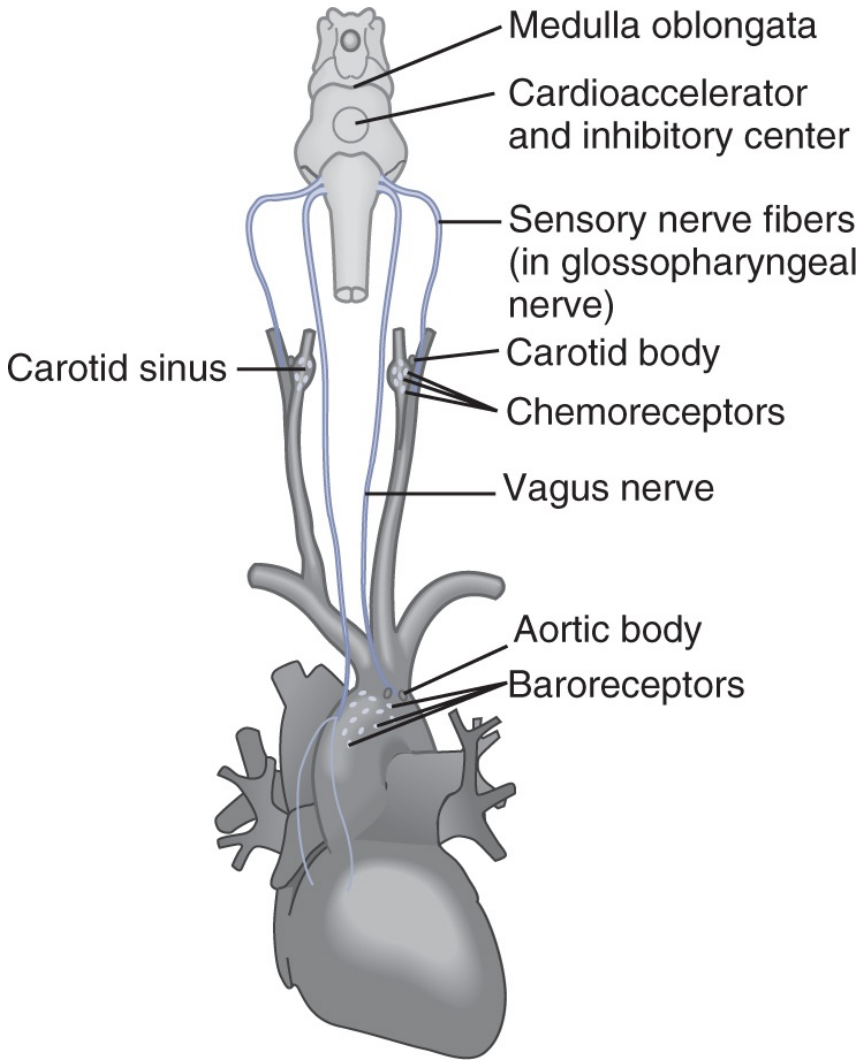


Figure 26-2 Chemoreceptors and baroreceptors.

Fetal Response to Hypoxia

Hypoxia is a reduced or insufficient level of oxygen in body tissues; definitions related to hypoxia are listed in **Table 26-3**.[15,17] The fetus responds to hypoxia in three ways: (1) a transient bradycardia that decreases oxygen consumption by as much as 60%, (2) redistribution of blood to vital organs, and (3) use of anaerobic metabolism.[15(p58)]

Table 26-3 Definitions of Terms Related to Hypoxia

Term	Definition
Acidemia	Increased concentration of hydrogen ions in the blood.
Acidosis	Increased concentration of hydrogen ions in the tissues of the body.
Aerobic metabolism	Metabolism of glucose using oxygen.
Anaerobic metabolism	Metabolism of glucose without the use of oxygen.
Asphyxia	Comes from the Greek word for "pulseless." A decrease in O_2 and increase in CO_2 secondary to an interference with gas exchange. Asphyxia is a continuum described by degrees of acidosis; this term is used clinically when tissue damage or death occurs.
Base	A substance that is capable of accepting hydrogen ions, thereby decreasing acidity.
Base excess (BE)	The amount of base or HCO_3^- that is available for buffering hydrogen ions. As metabolic acidosis increases, the base excess decreases.
Bicarbonate (bicarb)	HCO_3^- is the base or hydrogen acceptor that is part of the primary buffering system within the blood.
Buffer	A chemical substance that is both a weak acid and a salt. Buffers can absorb or give up hydrogen ions, thereby maintaining a constant pH value. The primary buffer involved in fetal oxygenation is HCO_3^-.
Hypercarbia	Excessive carbon dioxide in blood.
Hypoxemia	Decreased oxygen in blood.
Hypoxia	Decreased oxygen in tissue.
Metabolic acidemia	Low bicarbonate (negative base excess).
pH	The concentration of hydrogen ions in blood. The term pH refers to *puissance hydrogen*, which is French for "strength (or power) of hydrogen." A pH of 7.0 is the same as 0.0000001 (10^{-7}) mole per liter of hydrogen ions.
Respiratory acidemia	High PCO_2.

Reproduced with permission from Parer JT, King TL, Ikeda T. *Electronic Fetal Heart Rate Monitoring: The 5-Tier System*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2018.[15]

During normal *aerobic metabolism*, the fetus produces carbonic acid (H_2CO_3), which dissociates into water and carbon dioxide (CO_2). Both CO_2 and water diffuse across the placenta very quickly. When oxygen is not available for aerobic metabolism, anaerobic

metabolism occurs. *Anaerobic metabolism* utilizes glucose and glycogen to produce energy, with the final dissociation product being lactic acid ($C_3H_6O_3$). Unlike water and CO_2, lactic acid does not cross the placenta quickly. When anaerobic metabolism persists, the accumulating lactic acid will overwhelm the buffering capacity within the fetal circulation and lower the pH, resulting in a state of metabolic acidosis.

Mechanism of Asphyxia

Asphyxia is a state of extreme deficiency of oxygen and carbon dioxide excess. Severe asphyxia results in metabolic acidosis and ultimately organ failure. Because asphyxia is a continuum that cannot be directly measured, and because the individual response to asphyxia varies, there is no clear threshold at which one can say metabolic acidosis will have long-term adverse effects in the fetus/newborn. Thus, the term "asphyxia" is not used clinically or in written documentation when describing the newborn's status.

Respiratory acidosis in a fetus refers to a condition of elevated carbon dioxide. *Metabolic acidosis* refers to a loss of physiologic buffering capability and low pH in tissue, most often secondary to a buildup of lactic acid. When metabolic and respiratory acidosis become severe, normal metabolic processes begin to fail. Neurons have a high metabolic rate and the brain does not store glucose, so the fetal brain is particularly vulnerable to asphyxial damage. In addition, oxygen deficiency in brain tissue results in both acidemia and ischemia because the cardiovascular adaptive mechanisms that preserve cerebral perfusion become overwhelmed when the arterial O_2 falls below a critical threshold. Autoregulation of cerebral blood flow fails, such that cerebral perfusion becomes pressure dependent and then, in the presence of hypotension, the brain becomes ischemic.[18-24] Cellular metabolism is quickly compromised (**Figure 26-3**). Thus, the final pathway to brain injury is termed "hypoxic–ischemic."

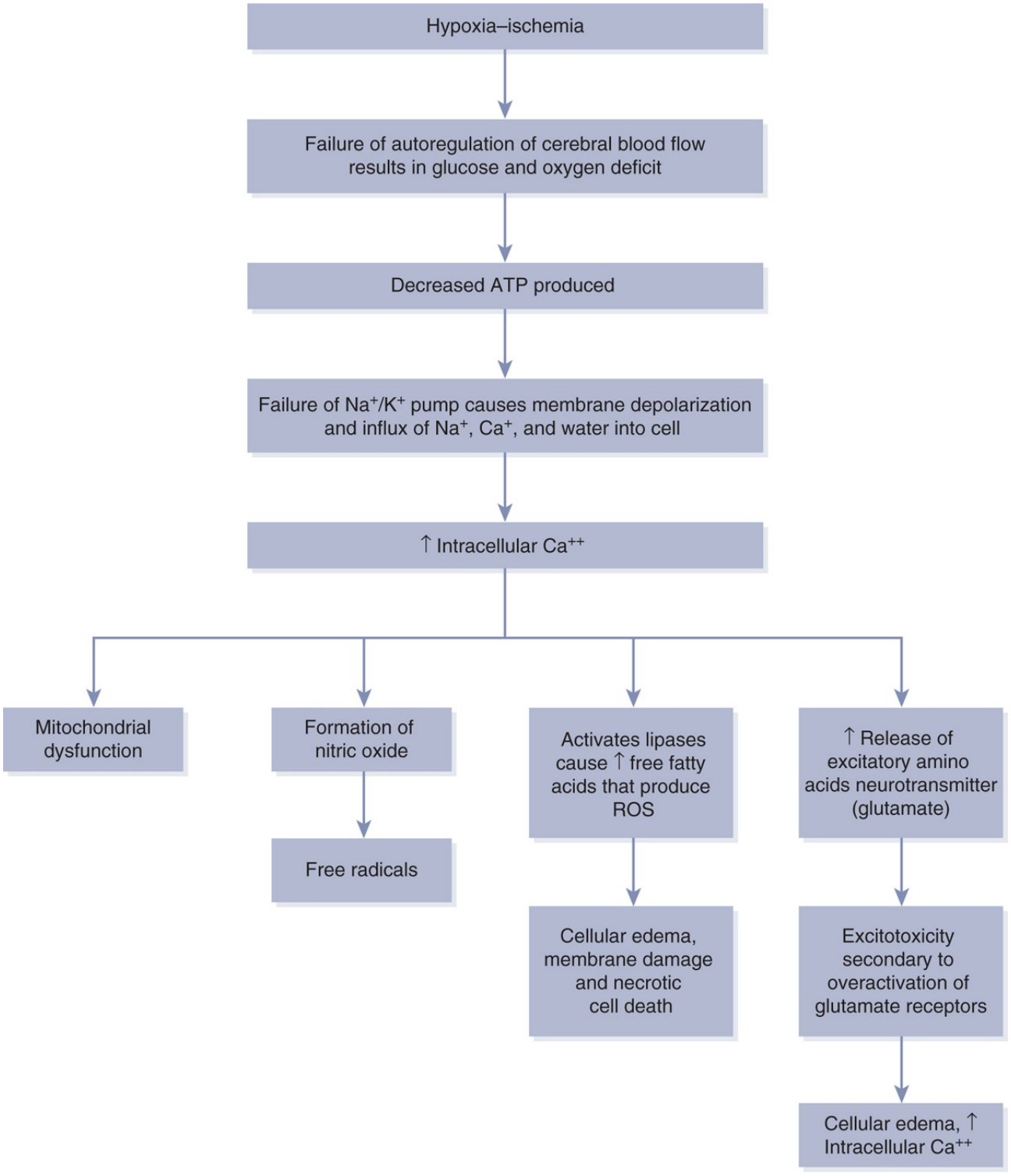


Figure 26-3 Primary energy failure: mechanism of hypoxic–ischemic injury.

Abbreviations: ATP, adenosine triphosphate; ROS, reactive oxygen species.

Reproduced with permission from Parer JT, King TL, Ikeda T. *Electronic Fetal Heart Rate Monitoring: The 5-Tier System.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2018.[15]

Hypoxic–Ischemic Encephalopathy and Neonatal Encephalopathy

Hypoxic–ischemic encephalopathy (HIE) is a descriptive term that describes the type of

lesion seen in the brains of newborns who survive a severe asphyxial insult. HIE occurs in approximately 1.5 per 1000 term births.[24] Approximately 66% of newborns with severe HIE will die or experience long-term sequelae. Conversely, the majority of infants with mild HIE have no adverse effects.[25]

Neonatal encephalopathy is a clinically defined syndrome of impaired central nervous system function in a newborn that includes disturbed neurologic function in an infant born at or after 35 weeks' gestation and with a subnormal level of consciousness or seizures.[26] Neonatal encephalopathy occurs in approximately 2 per 1000 live births. The criteria necessary to assign the etiology of neonatal encephalopathy to intrapartum asphyxia are listed in **Table 26-4**.[26,27]

Table 26-4 **Acute Peripartum or Intrapartum Events Sufficient to Cause Neonatal Encephalopathy**

Neonatal Characteristic	Description
Apgar score	< 5 at 5 and 10 minutes after birth.
Umbilical artery acidemia	pH < 7.0 and/or base deficit ≥ 12 mmol/L.
Magnetic resonance imaging	MRI abnormalities that appear after 24 hours of life. MRI obtained between 24 and 96 hours after birth is the most sensitive for determining the timing of cerebral injury. Conventional qualitative MRI findings of diffusion abnormalities, deep nuclear gray matter, or watershed cortical injury are most likely to indicate hypoxic–ischemic injury.
Multisystem organ failure	Can include renal injury, hepatic injury, hematologic abnormalities, gastrointestinal injury, cardiac dysfunction, metabolic derangements, or a combination of these abnormalities.
Sentinel hypoxic or ischemic event	A hypoxic or ischemic event that occurred immediately before or during labor and delivery (e.g., ruptured uterus, placental abruption, vasa previa, and amniotic fluid embolism).
Fetal heart rate patterns	Category II tracing identified on presentation or for longer than 60 minutes that includes minimal/absent variability and no accelerations; suggests a previously compromised fetus.
	Category I FHR pattern that develops into a Category III pattern over the course of labor.
	Additional FHR patterns consistent with hypoxic ischemic events: tachycardia with recurrent decelerations and persistent minimal variability with recurrent decelerations.
Other proximal or distal factors	No evidence of other proximal or distal factors that could contribute to neonatal encephalopathy.

Abbreviations: FHR, fetal heart rate; MRI, magnetic resonance imaging.

Based on American College of Obstetricians and Gynecologists Task Force on Neonatal Encephalopathy. *Neonatal Encephalopathy and Neurologic Outcome*. 2nd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2014.[26]

Hypoxic–ischemic brain injury is a subset of neonatal encephalopathy, in that it is one of many etiologies that cause neonatal encephalopathy. Because neither hypoxia nor ischemia can be assumed to have been the unique initiating causal mechanism for a specific newborn with neurologic impairment, the term "hypoxic–ischemic encephalopathy" is not used clinically and has been replaced by "neonatal encephalopathy."[26]

The region of the brain that is damaged depends on the gestational age of the fetus, as

hypoxic–ischemic injury typically occurs in border zones between the ends of major cerebral vessels. Spastic quadriplegia cerebral palsy may result from hypoxic–ischemic injury in term infants because the regions of the brain that are most likely to suffer insult at that point of gestation are those involved in motor cordination.[26]

The relationship between hypoxic–ischemic injury secondary to oxygen deprivation during labor and subsequent development of cerebral palsy is complex. Cerebral palsy has multiple etiologies. Many of the symptoms of brain injury that are evident in the newborn reflect a common pathway that several different pathologic events exhibit. For example, placental lesions, intrauterine infection, and intrauterine stroke are all potential inciting events that may result in fetal brain damage and subsequent cerebral palsy.

Methods of Fetal Assessment During Labor

Fetal well-being can be assessed during labor via intermittent auscultation of the FHR as well as continuous or intermittent EFM. Fetal scalp stimulation may be used as an adjunctive test to determine whether the fetal pH is in the normal range.

Intermittent Auscultation

Auscultation of the fetal heart rate may be performed with a fetoscope, a Doppler device, or even the external ultrasound transducer of the electronic fetal monitor. The DeLee-Hillis fetoscope and the Allen fetoscope use bone conduction in the listener's cranium to amplify the sounds of the FHR. Because fetoscopes that do not use Doppler all require use of bone conduction and close proximity of the examiner's head to the pregnant abdomen, maternal position, habitus, and activity can limit assessment of the FHR with a fetoscope.

Doppler ultrasound technology projects an ultrasound wave into the uterus. This wave is reflected off the moving fetal ventricle, and the waveform changes as it is reflected back to the Doppler transducer. The transducer detects the heart movement of each heartbeat and emits an electronically created sound that can be counted or displayed digitally. Doppler devices can detect the fetal heart movement when the woman is in most positions, and some models can be safely used underwater, allowing for their use during hydrotherapy in labor and birth. Auditory counting of the fetal heart rate by Doppler technology and by fetoscope has been demonstrated to be equivalent in pregnant women who are not in labor.[28,29]

Use of the EFM Doppler transducer for intermittent documentation is somewhat controversial. Advocates find use of the EFM ultrasound transducer to be convenient, and a printout of the tracing can be made. Caution is warranted, however: When an FHR tracing is obtained, data including the FHR variability and possible accelerations or decelerations are evident and must be interpreted according to continuous EFM guidelines. No standard exists in this regard; each institution and individual clinician must consider the pros and cons of relying on these data. The technique for performing intermittent auscultation is presented in **Appendix 26A.**

Electronic Fetal Heart Rate Monitoring

The electronic fetal monitor displays the fetal heart rate pattern and the uterine contraction pattern. It can be used with devices that are placed externally on the maternal abdomen or internally into the uterus through the vagina. The fetal monitor record has both upper and lower display areas. The upper portion displays the fetal heart rate pattern, with increments of 10 bpm graphed on the vertical axis. The lower portion displays the uterine contractions, with gradations for 0 to 100 Montevideo units on the vertical axis. The horizontal axis is divided into 10-second and 1-minute segments. Therefore, the FHR pattern, the contraction pattern, and the timing and relationship between the two are displayed on the device.

External Monitoring

For external fetal heart rate monitoring, a Doppler device that has a transducer and a receiver is secured onto the maternal abdomen, over the area of the fetal heart. The Doppler transducer transmits a high-frequency ultrasound pulse of approximately 2.5 MHz, then records a number of points on the changed wavelength that is reflected off the moving fetal heart during each beat. A replica from these points is constructed using a technique called autocorrelation.

In brief, the monitor counts the interval between fetal heartbeats and calculates a beat-per-minute (bpm) rate. It then plots this rate on the graph paper, which scrolls continuously so that the individual plot points merge into a jagged line. This *fetal heart rate variability* line is jagged because the time interval between each heart beat typically varies. Because the vagus nerve is responsive to input from the central nervous system, and because frequent changes in timing between beats occur secondary to vagus input, the variability seen on EFM primarily reflects central nervous system function and well-being.

Fetal heart rate monitoring technology has improved over the last decade such that the FHR recorded using an external Doppler transducer now closely approximates the exact FHR recorded on an electrocardiogram (ECG).[30] Early fetal monitors did not have an autocorrelation function, which meant that they often displayed too much interference to allow accurate interpretation of baseline variability. Today's external fetal monitoring offers the same reliability as internal monitoring for assessment of all FHR characteristics.

External fetal monitoring is appropriate for the majority of women for whom continuous EFM during labor is indicated. In most situations, with careful placement of the abdominal transducer and tocodynamometer, the FHR and contractions will be visually displayed adequately for interpretation. The equipment and its electrical cords limit the distance a woman can move about unless a telemetry unit is available. Telemetry units currently in use allow women to walk long distances, shower, or be in a tub. However, maternal movement can cause interference in the signal, occasionally obscuring or making the data being collected unreadable.

Internal Fetal Heart Rate Monitoring

Internal monitoring is used when the fetal heart rate or the contractions need to be monitored continuously and an adequate tracing is not available via external monitoring, such as when a woman with a high body mass index (BMI) is in labor. Internal monitoring requires ruptured membranes. A fetal scalp electrode (FSE) is placed a few millimeters into the fetal scalp. The wires extending from the electrode are entrapped in an electronic transmission device that is secured to the woman's thigh with an elastic or Velcro belt. The fetal scalp electrode transmits the fetal electrocardiogram to the monitor, which displays a digital signal of the FHR as well as a continuous graph of the heart rate. The procedure for placing an FSE is described in **Appendix 26B.**

Uterine Activity Monitoring

Uterine contractions can be monitored via maternal perception, manual palpation, external tocodynamometer attached to an electronic fetal monitor, or internal placement of an

intrauterine pressure catheter (IUPC).

Although there is significant variability between individuals, maternal perception of a painful contraction usually occurs when the intrauterine pressure reaches a minimum of 15 mm Hg.[31] This pressure is the amount required to distend the lower uterine segment and create pressure on the cervix. A uterine contraction is first detectable via manual palpation when the intrauterine pressure exceeds 10 mm Hg.[31]

A uterine pressure tocodynamometer (toco) is placed on the maternal fundus and secured with a belt that goes around the woman's abdomen. The toco records changes in pressure that occur when the fundus tightens during a contraction. Although this device records contraction duration and frequency, it does not record intensity accurately. Moreover, tightening the monitor straps or changing the position of the toco can cause the device to show a very different apparent intensity of contractions.

Internal uterine monitoring is accomplished by placing an intrauterine pressure catheter through the cervix and into the uterine cavity. The IUPC has the advantage of providing specific information about the resting tone of the uterus, the actual pressure generated by the contractions, and accurate timing of the onset, peak, and completion of a contraction. If amnioinfusion is needed, it can be done through the same catheter used to perform internal uterine monitoring. Disadvantages of this technology include an increased risk of infection if the IUPC is left in place for an extended period of time and the requirement that the woman remain tethered to a bed.

If the progress of labor is slower than expected, an IUPC may be used to provide a quantitative measurement of uterine contraction intensity in Montevideo units—a concept first introduced by Caldeyro-Barcia in 1957.[32] The Montevideo unit is calculated by adding together the peak pressures of all contractions that occur in a 10-minute window. Montevideo units of approximately 200 when subtracting the baseline tone, or 240 when the baseline tone is included, are considered adequate for labor that progresses normally.

Fetal Heart Rate Patterns

The characteristics of the fetal heart rate are classified as baseline, periodic, or episodic.[10] The baseline features, which include the heart rate, variability, and accelerations, are assessed between uterine contractions. Periodic changes occur in association with uterine contractions. Episodic changes are not clearly associated with uterine contractions.

Baseline Fetal Heart Rate

The baseline fetal heart rate at term ranges from 110 to 160 bpm. Bradycardia is defined as an FHR of less than 110 bpm; tachycardia is defined as a baseline rate of more than 160 bpm. Fetal tachycardia alone is not usually associated with poor outcomes in the term fetus. Mild tachycardia is common in the fetus of less than 28 weeks' gestation because parasympathetic (vagal) tone does not predominate until the third trimester. Thus, heart rates of 140 to 160 bpm are normal baseline rates in a preterm fetus.

Sustained tachycardia in a term fetus may be caused by developing acidemia,[33,34] maternal or fetal infection,[35] or cardiac arrhythmia,[36] or it may be idiopathic. Other causes include administration of beta-mimetic drugs or ephedrine given to correct maternal hypotension. More rarely, fetal tachycardia occurs secondary to fetal anemia (Rh isoimmunization), acute fetal blood loss (placental abruption), an abnormal fetal conduction system (fetal arrhythmia), or poorly controlled maternal hyperthyroidism.

Short periods of tachycardia are a normal compensatory response to transient hypoxemia. Sustained tachycardia, especially if accompanied by minimal variability and recurrent decelerations, may indicate that the fetus's ability to compensate for repeated hypoxia is limited.[33,37] This pattern can appear after recurrent decelerations are present for some period of time but before a terminal bradycardia becomes evident, and is characteristic of FHR pattern evolution when acidemia is increasing.

Both asphyxial and non-asphyxial causes of fetal bradycardia are possible. Non-asphyxial causes include fetal heart block and maternal hypothermia. A mild bradycardia in the 100–120 bpm range can be idiopathic and is often seen in the postmature fetus, as the heart rate progressively slows over the course of gestation and early childhood. Bradycardia can also develop during rapid descent of the fetal presenting part or at the end of the second stage of labor when fetal head compression causes increased intracranial pressure, which then initiates a vagal response and bradycardia. In such a case, the fetus will maintain cerebral oxygenation at an FHR higher than 80 bpm and variability will be retained. Fetal bradycardia also can occur after administration of intrathecal opioids or local anesthetics for epidural analgesia. This etiology is usually attributed to maternal hypotension, secondary to the sympathetic block, which causes a decrease in uteroplacental blood flow. These transient bradycardias or prolonged decelerations usually retain normal FHR variability and are rarely associated with adverse outcomes. Fetal bradycardia that is accompanied by moderate variability is not associated with fetal acidemia. The presence of moderate variability during a mild bradycardia can reassure the midwife that no intervention is required.[37]

Asphyxial causes of bradycardia include acute emergency events such as prolapsed cord,

placental abruption, uterine rupture, or vasa previa.[38] An FHR of less than 60 bpm is an emergency. At this heart rate, the fetus is unable to increase stroke volume to sustain adequate circulation through the heart and supply the coronary arteries. Thus, when an acute bradycardia occurs, the depth, duration, and the presence or absence of variability are the key factors associated with subsequent neonatal morbidity.

Fetal Heart Rate Variability

The presence of moderate FHR variability signifies that the fetus has an intact cerebral cortex, midbrain, vagus nerve, and cardiac conduction system. Moderate variability is the single best indicator that the fetus does not have cerebral tissue hypoxia or ischemia.[37,39] Approximately 98% of all fetuses with moderate variability will not have clinically significant acidemia at the time the variability is observed, even if recurrent fetal heart rate decelerations are present (**Figure 26-4**).[37]

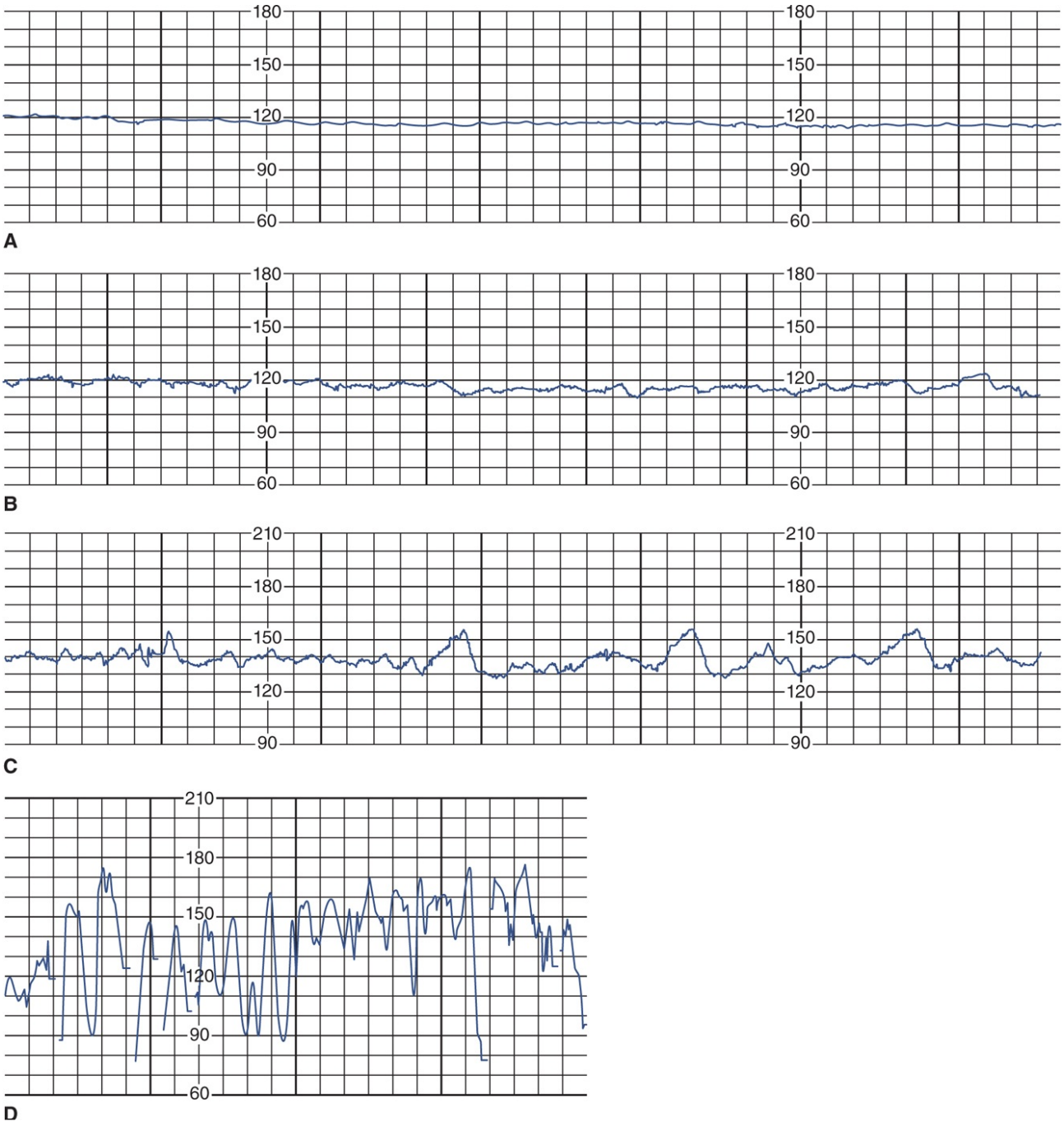


Figure 26-4 Fetal heart rate variability. **A.** Absent variability. **B.** Minimal variability. **C.** Moderate variability. **D.** Marked variability.

Minimal variability is interpreted based on the presence or absence of accelerations or recurrent decelerations. If the variability is minimal and FHR decelerations are not present, this condition has no correlation with fetal acidemia.[37] Fetal rest cycles are associated with uncomplicated minimal variability, with these periods of minimal variability typically lasting 20 to 40 minutes but sometimes persisting for as long as 75 to 80 minutes.[40] Administration of opiates (e.g., morphine, butorphanol, fentanyl), promethazine (Phenergan), or beta-adrenergic agents such as terbutaline (Brethine) to the mother will also decrease the amplitude of FHR variability.

Like minimal variability, absent variability is interpreted based on the presence or absence of decelerations. Absent variability without decelerations can be idiopathic and not consistently associated with fetal acidemia. In contrast, minimal or absent variability in the presence of recurrent late or variable decelerations is one of the FHR patterns most consistently associated with fetal acidemia and requires urgent assessment and intervention.[14,33,37,41-43] The evolution of moderate to minimal to absent variability in the presence of recurrent decelerations that become progressively deeper is the classic pattern evolution that reflects developing fetal acidemia over time.[37,41-43]

Marked variability is relatively rare. It is hypothesized that this FHR characteristic is a response to a short period of hypoxia. Marked variability is not associated with adverse newborn outcomes.

Accelerations

Accelerations are transient increases of the fetal heart rate above the baseline (**Figure 26-5**). They are a reassuring indicator of fetal well-being, in that they are associated with a normal pH at the time the acceleration occurs.[44] Accelerations are often associated with fetal movement. Fetal scalp stimulation and acoustic stimulation can be used to induce an acceleration.

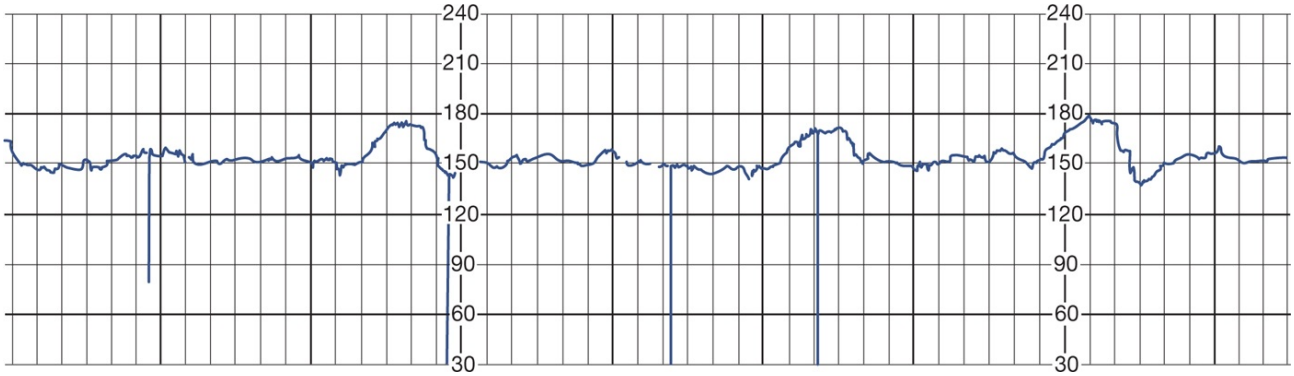


Figure 26-5 Accelerations of the fetal heart rate.

Early Decelerations

The mechanism underlying early decelerations has not been determined. These decelerations appear in active labor and are associated with moderate variability. Although one theory suggests that early decelerations are caused by head compression, head compression results in a vagal response and variable decelerations.[15(p5)] Regardless of the underlying physiologic mechanism, early decelerations are benign and do not require intervention, although it is important to carefully differentiate between early and late decelerations (**Figure 26-6**).

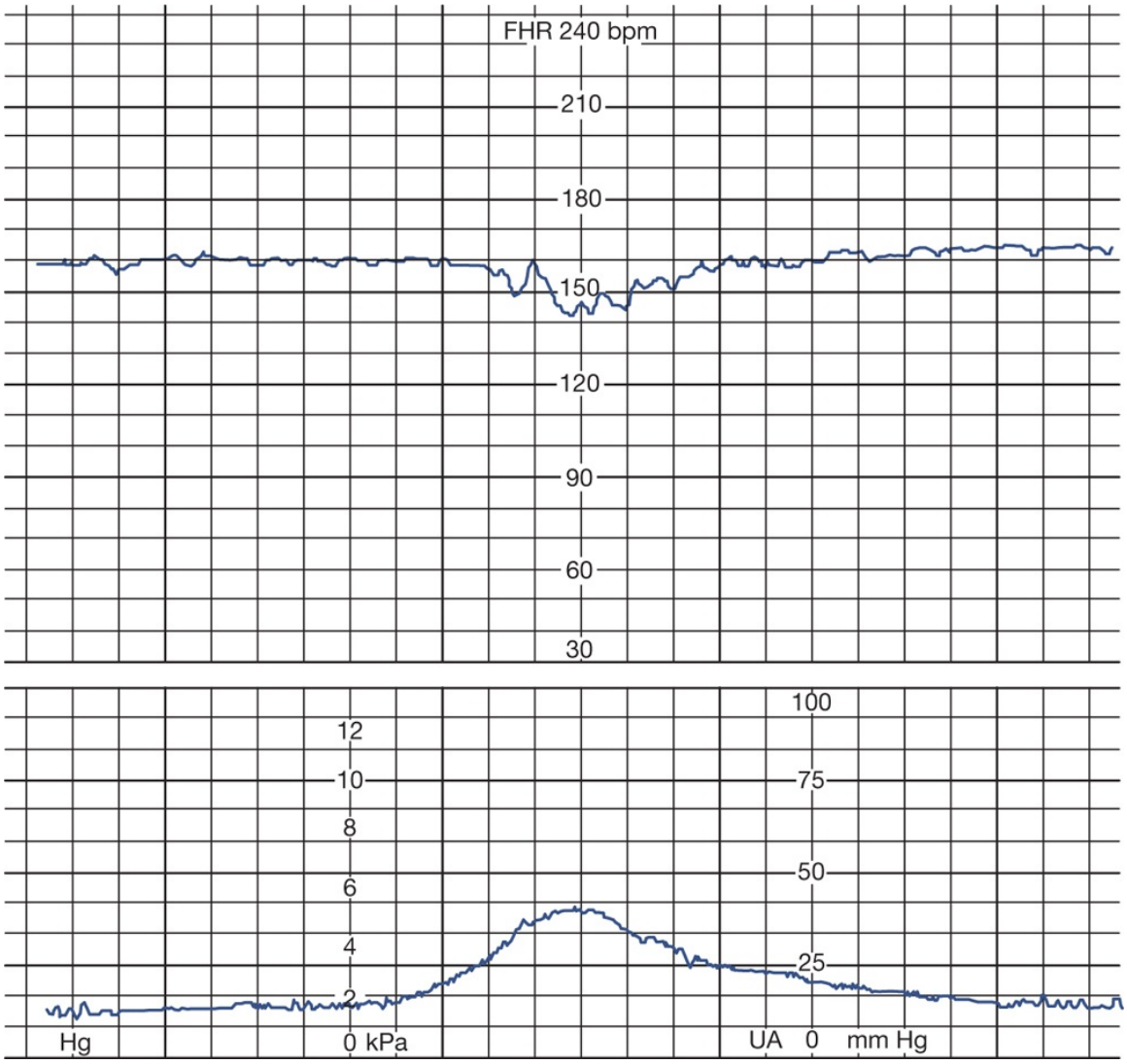


Figure 26-6 Early decelerations.

Abbreviations: bpm, beats per minute; FHR, fetal heart rate; UA, uterine activity.

Late Decelerations

Late decelerations are a response to transient decreases in oxygen tension. This mechanism starts with a uterine contraction that causes a decrease in uteroplacental perfusion, which is followed by lower oxygen levels in the fetal circulation. The hypoxemia is detected by chemoreceptors in the fetal carotid artery, carotid sinus, and aortic arch. Chemoreceptor activation signals a vagally mediated drop in fetal heart rate. The nadir of the fetal bradycardia is "late" relative to the peak of the uterine contraction, because the physiologic pathway that results in chemoreceptor stimulation takes time. The relatively deoxygenated blood must first traverse the umbilical vein before reaching the chemoreceptors (**Figure 26-7**).

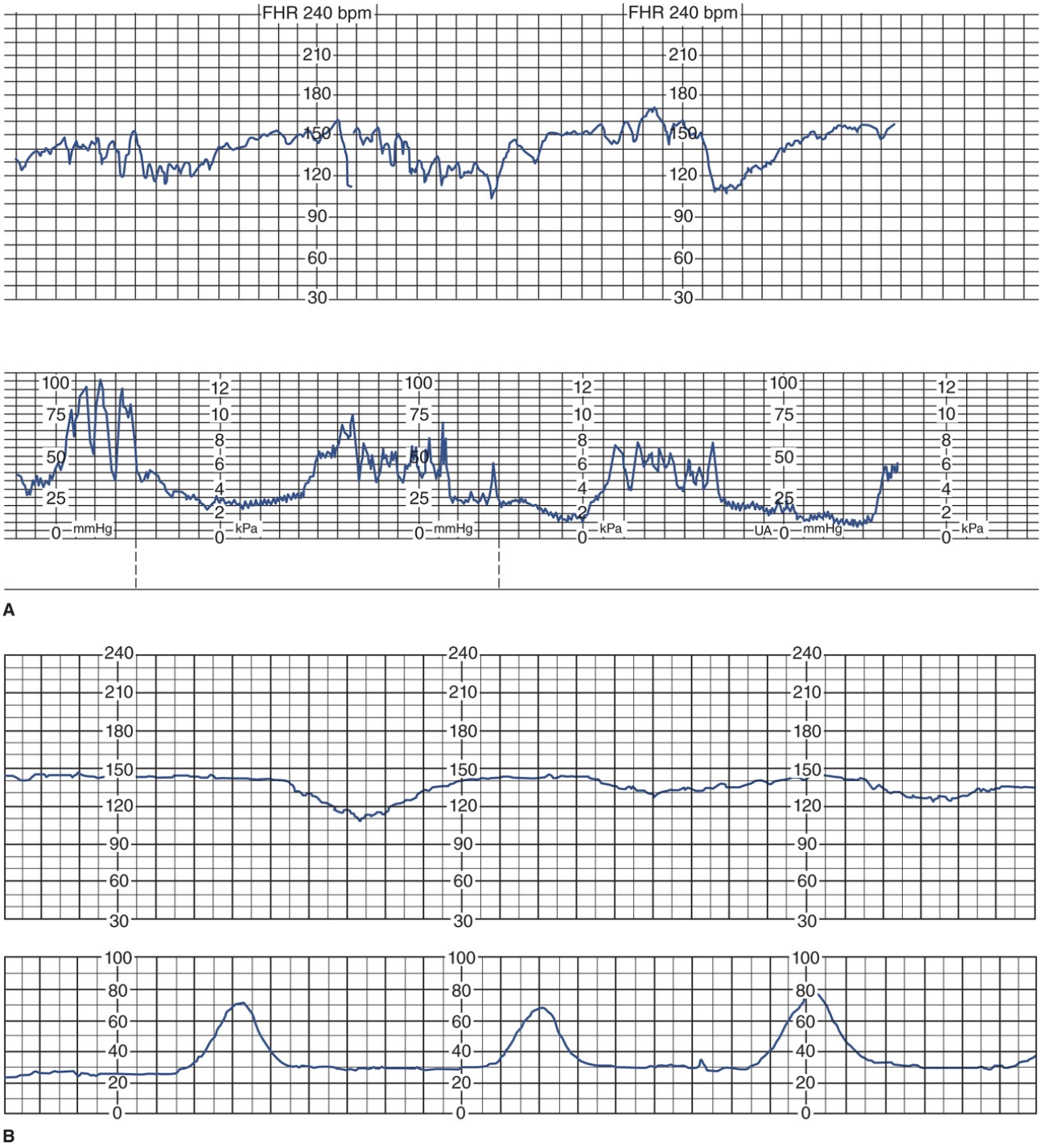


Figure 26-7 Late decelerations. **A.** Late deceleration with moderate variability. **B.** Late deceleration with absent variability.

Abbreviations: bpm, beats per minute; FHR, fetal heart rate.

Recurrent late decelerations are associated with uteroplacental insufficiency. *Uteroplacental insufficiency* refers to any condition in which the placenta is not able to supply sufficient oxygen or nutrients to the fetus. It can be chronic or acute. Causes of chronic uteroplacental insufficiency include maternal hypertensive disorders, diabetes, hyperthyroidism, autoimmune disorders such as lupus, and abnormal placentation. All of these disorders are associated with narrowed spiral arteries, small placentas, and/or placental

infarcts that decrease the placental area available for transfer of oxygen and nutrients. In addition, uteroplacental insufficiency is associated with postmaturity. Acute uteroplacental insufficiency can develop in the setting of uterine tachysystole or maternal hypotension. Finally, fetal anemia secondary to fetal–maternal hemorrhage, Rh sensitization, or nonimmune hydrops will effectively lower the oxygen-carrying capacity of the fetus, so that the transient decreases in oxygen present in the intervillous spaces associated with uterine contractions result in significant declines in fetal oxygen tension.

Late decelerations in the presence of moderate variability represent a physiologic response to transient hypoxemia, rather than an indicator of the presence of acidemia.[37] Conversely, recurrent late decelerations in the presence of minimal or absent variability is one of the FHR patterns most commonly associated with fetal acidemia.[11,37]

Late decelerations can be classified as mild (≤ a 15-bpm drop from baseline to nadir), moderate (> a 15-bpm drop but less than a 45-bpm drop from baseline to nadir), or severe (≥ 45-bpm drop from baseline to nadir). Although these classifications are not part of the NICHD terminology, good evidence supports the observation that late decelerations become progressively deeper as fetal acidemia worsens.[37,45]

Variable Decelerations

Variable decelerations occur secondary to an interruption in umbilical blood flow. Numerous causes of umbilical cord compression have been identified. In vertex presentations, these etiologies include cord occlusion secondary to the cord around the fetus's neck or body, true knots in the cord, frank and occult prolapse of the cord, and compression due to decreased amniotic fluid that cushions the cord. Variable decelerations are also common in the second stage of labor, in which case they are presumed to be secondary to head compression and the direct effect of increased intracranial pressure on the vagus nerve. Variable decelerations can be either episodic (occurring at random times) or periodic (occurring with each uterine contraction).

Variable decelerations are the most common variant FHR pattern, seen in approximately 45% to 75% of all labors.[46,47] The association between variable decelerations and fetal acidemia depends on the frequency, depth, duration and associated baseline FHR variability. Variable decelerations that return to baseline in less than 60 seconds with concomitant normal baseline and moderate variability are not associated with fetal acidemia. Recurrent variable decelerations with minimal or absent variability, similar to recurrent late decelerations with minimal or absent variability, are associated with fetal acidemia and require urgent evaluation and consideration of intervention (**Figure 26-8**).

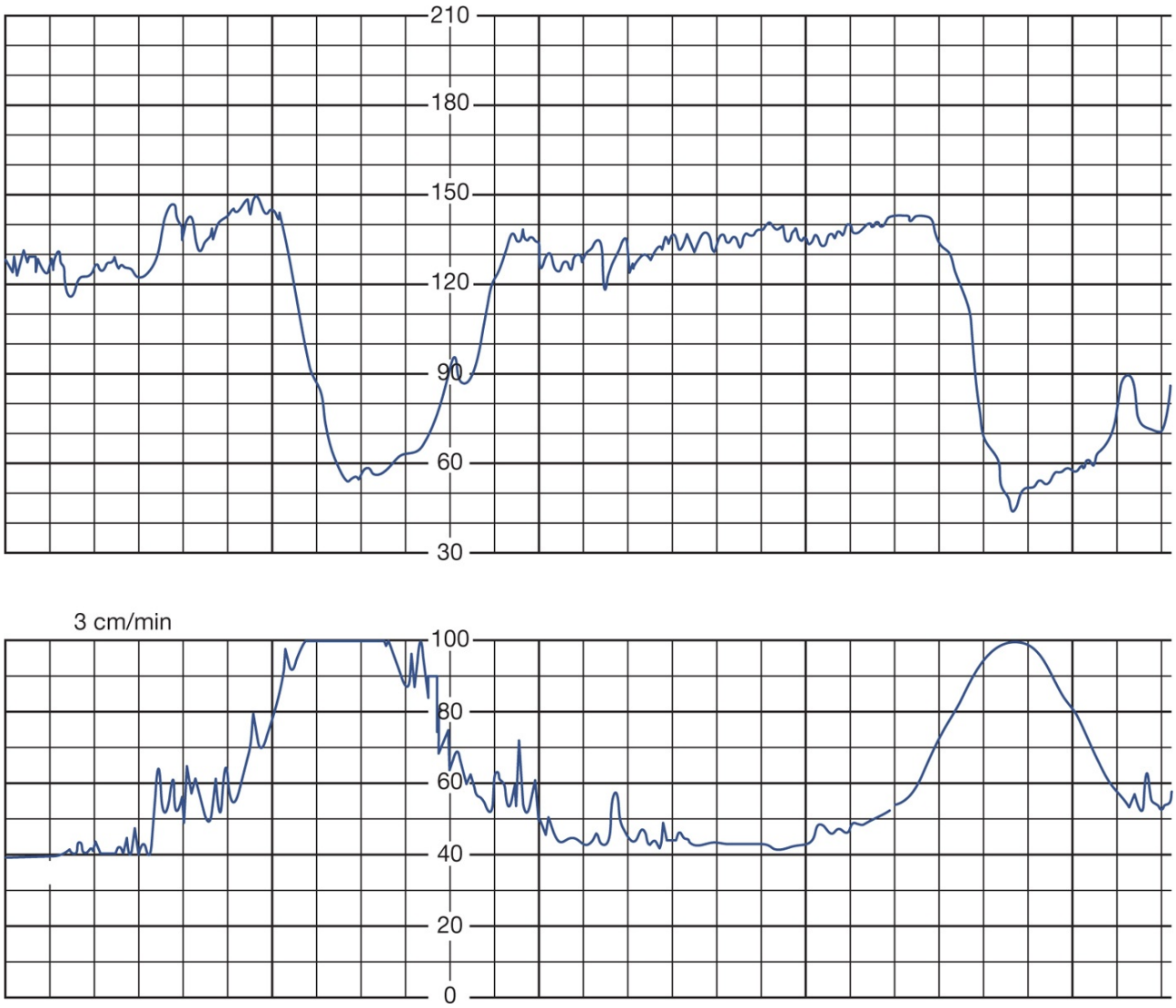


Figure 26-8 Variable decelerations.

Categorization of variable decelerations as mild, moderate, or severe has not proved to be clinically useful. Alternatively, variable decelerations may be categorized as typical or atypical based on their shapes. For example, variable decelerations often have a short increase in FHR above the baseline that precedes or is part of the final variable complex. These short increases are called shoulders and overshoots, respectively. Several studies have attempted to determine which characteristics of variable decelerations are most likely to be associated with acidemia, albeit without success.[47,48] Animal studies of umbilical cord occlusion that resulted in variable decelerations have shown that increasing depth, duration, and frequency are positively correlated with increasing acidemia, but specific deceleration shapes do not appear to be strongly correlated with specific degrees of fetal acidemia.[49]

Prolonged Decelerations

Prolonged decelerations have multiple causes. Known precipitating factors include umbilical cord compression; maternal hypotension related to supine positioning and epidural or spinal

anesthesia; paracervical anesthesia (which can cause decelerations by direct fetal uptake of local anesthetic, maternal hypotension, or uterine hypertonus); uterine tachysystole; maternal hypoxia associated with seizures or acute respiratory depression; and rapid descent of the fetal head (**Figure 26-9**).

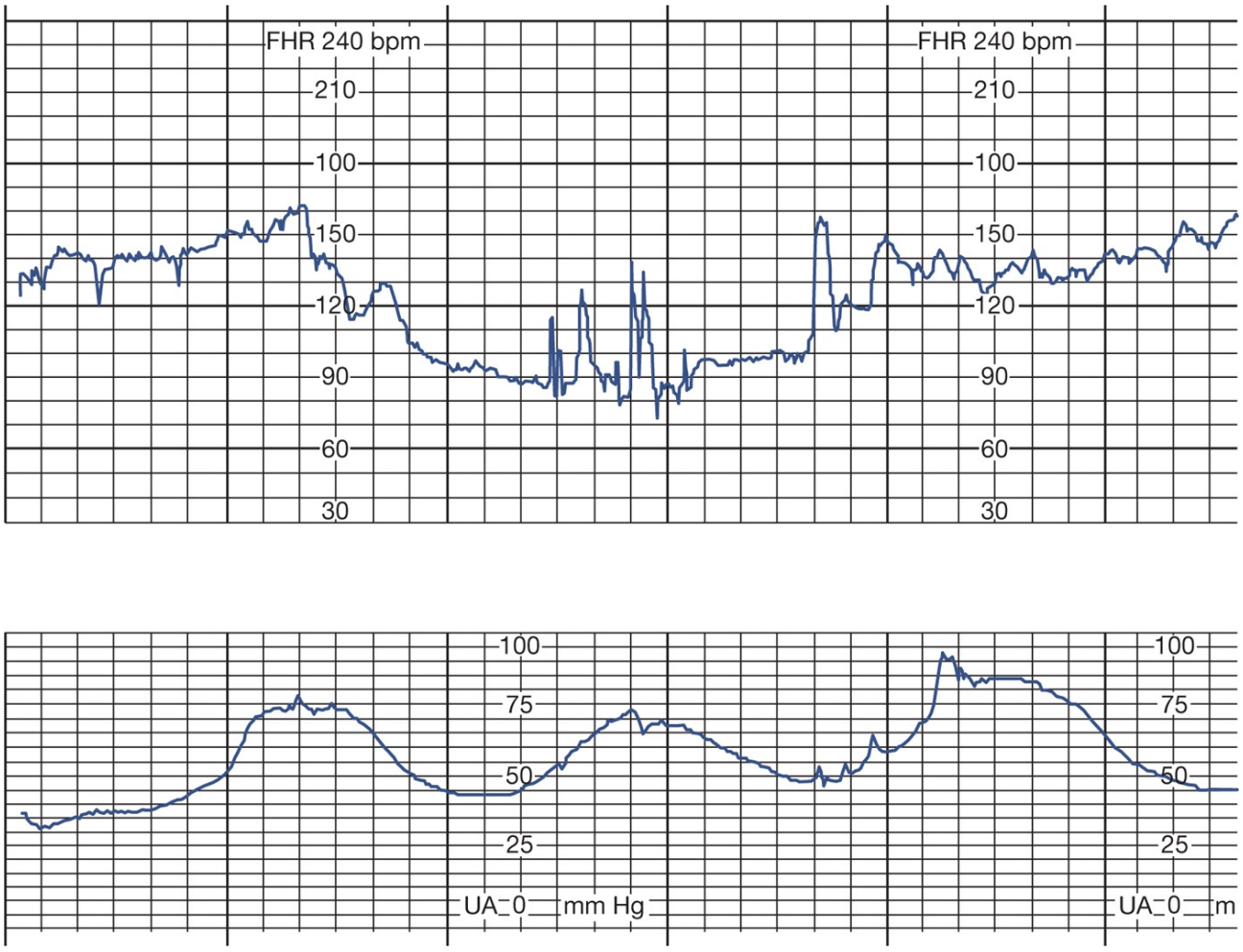


Figure 26-9 Prolonged decelerations.

Abbreviations: bpm, beats per minute; FHR, fetal heart rate; mm Hg, millimeters of mercury; UA, uterine activity.

Management of prolonged decelerations depends on the putative etiology, length of deceleration, baseline at nadir, and baseline variability. Most often, the fetus will recover with a period of tachycardia, decreased variability, or occasional late decelerations for a short time following a prolonged deceleration. If the precipitating cause is alleviated, the FHR generally returns to the previous baseline. Intermittent prolonged decelerations are not associated with an increased risk for fetal acidemia in the presence of moderate variability.

Sinusoidal Pattern

A sinusoidal pattern is characterized by an undulating, recurrent uniform fetal heart rate equally distributed 5 to 15 bpm above and below the baseline (**Figure 26-10**). The undulation occurs at a rate of 2 to 6 cycles per minute and is identified by an absence of short-term variability.

True sinusoidal patterns are extremely rare.

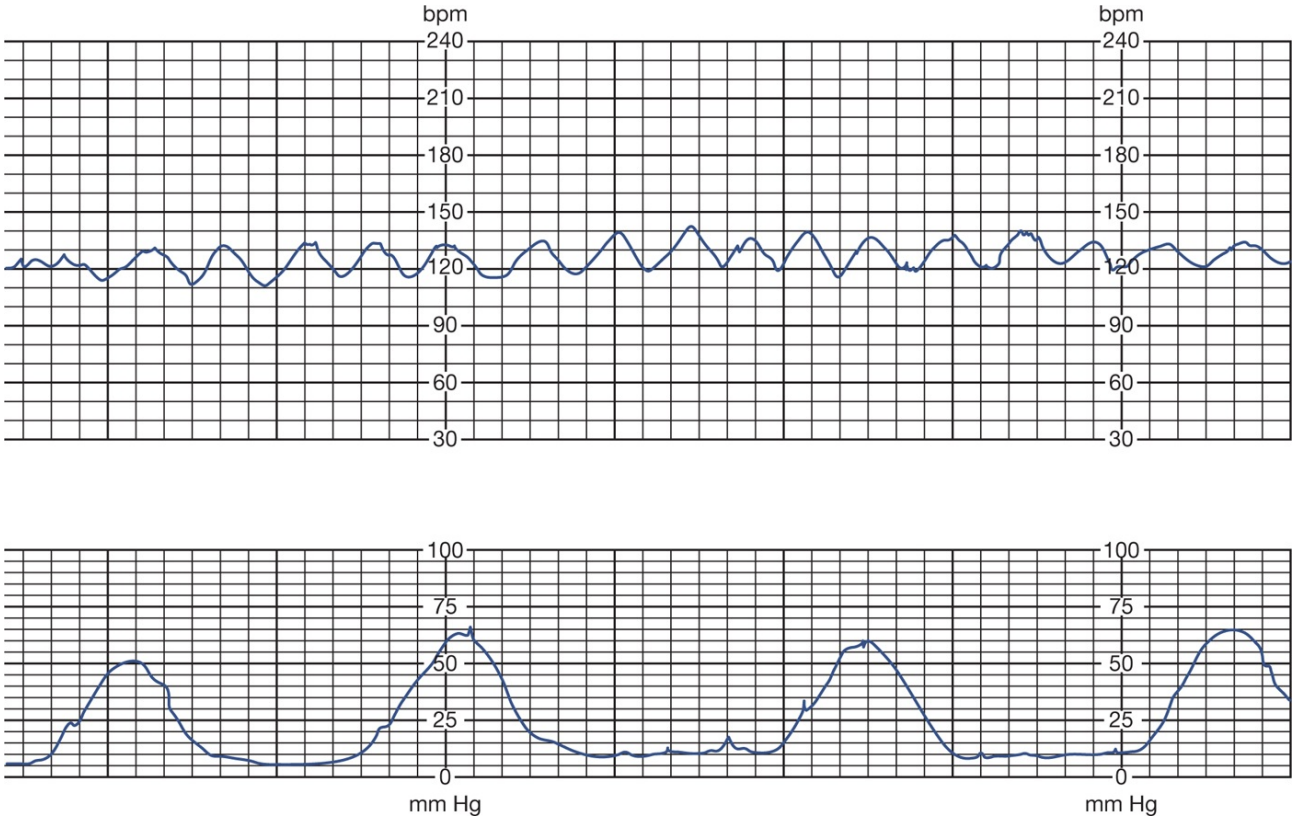


Figure 26-10 Sinusoidal pattern.

Abbreviation: bpm, beats per minute.

The sinusoidal FHR pattern is seen when the fetus is experiencing clinically significant anemia, such as occurs during a maternal-fetal hemorrhage following vasa previa, uterine rupture, or placental abruption. Historically, this pattern was seen in fetuses with anemia secondary to Rh isoimmunization. It may also appear shortly before death in a severely asphyxiated fetus. A sinusoidal heart rate pattern is always an ominous fetal heart rate pattern, and immediate intervention is indicated. The midwife should notify the consulting physician promptly and prepare the woman for emergency cesarean section. The neonatal team should be notified to prepare for a potentially anemic or hypovolemic newborn.

Pseudo-sinusoidal Fetal Heart Rate Pattern

The pseudo-sinusoidal pattern is not an NICHD-recognized FHR pattern because it is commonly the result of opioid administration, rather than being an intrinsic FHR characteristic. This clinically insignificant pattern is intermittent and bracketed by periods of moderate variability. A pseudo-sinusoidal FHR pattern differs from a real sinusoidal pattern in two ways—namely, variability is retained and there are more than 2 to 6 cycles per minute.

Wandering Baseline

Wandering baseline is not part of the official NICHD FHR terminology, but it continues to be

used in clinical practice and, therefore, deserves mention. A wandering baseline is a very late development in the progression of fetal deterioration. It is often within the normal baseline parameters of 120 to 160 bpm and is identifiable by absent variability. This pattern is an ominous indicator of fetal acidemia and requires immediate notification of a consulting physician and preparation for emergency operative birth if vaginal birth is not imminent. Neonatal/pediatric providers should be present at the birth. The key characteristic is the absence of variability, rather than the fact that the baseline changes over the course of several minutes.

Fetal Arrhythmias

Fetal arrhythmias occur among approximately 1% to 3% of fetuses at term.[50,51] Approximately 90% of fetal arrhythmias are transient and benign in nature. A consulting physician should evaluate any fetus with an arrhythmia, and a plan should be made for ongoing collaboration as long as the arrhythmia is present. Arrhythmias can be heard on audible Doppler devices and can be seen on FHR tracings, but cannot be accurately diagnosed via EFM (**Figure 26-11**). Thus, neonatal/pediatric attendance at birth may be recommended depending on the rate and persistence of the arrhythmia.

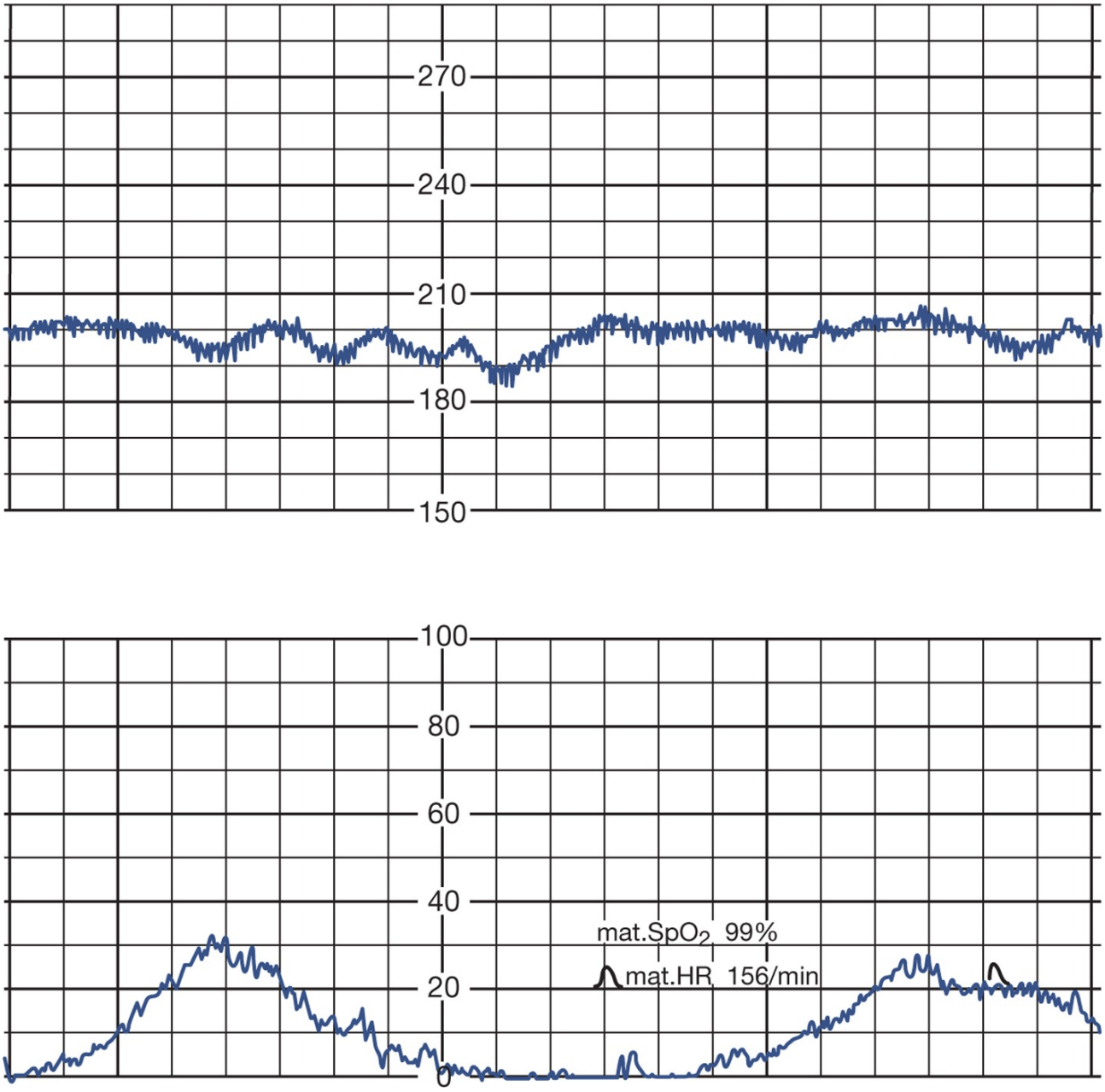


Figure 26-11 Fetal arrhythmia.

Fetal Heart Rate Pattens That Reflect Fetal Acidemia

Parer et al. reviewed the FHR research published between the 1960s and 2005 that evaluated the relationship between FHR patterns and newborn outcomes in term gestations.[15,37] In this review, the following themes were identified that capture the relationship between FHR patterns and clinically significant newborn acidemia:

1. Moderate variability is strongly predictive of neonatal vigor independent of the presence of variant patterns. Moderate FHR variability has a negative predictive value of 98% to 99% in a term fetus.
2. Minimal and absent variability in the presence of late or severe variable decelerations are the FHR patterns most likely to be associated with fetal acidemia, although the positive predictive value is only in the range of 10% to 30%.
3. A positive relationship can be found between the depth and severity of decelerations or bradycardia and the degree of acidemia.
4. Newborn acidemia with decreasing FHR variability in combination with decelerations develops over a period of approximately 1 hour in a previously healthy term fetus.

Clinically, significant metabolic acidosis in the fetus can develop in one of three ways:

- Acidosis may develop as the result of frequent, unremitting hypoxic insults (e.g., uterine contractions) that occur too frequently to allow transfer of respiratory and metabolic components across the placenta.
- Metabolic acidosis may develop rapidly following an acute emergency such as uterine rupture. An acute unremitting bradycardia may be both the cause and result of sudden marked decrease in oxygen delivery to the fetus.
- Prenatally, metabolic acidosis may develop slowly over time during the course of intrauterine growth restriction, fetal infection, or other fetal disorder.

Pattern Evolution During Labor

In a term healthy fetus, it takes approximately 1 hour to develop clinically significant metabolic acidemia once a pattern of recurrent late or variable decelerations and diminishing variability begins during labor.[37] Midwives, nurses, and physicians interpreting EFM results should recognize the typical pattern evolution that occurs as acidemia is developing (**Figure 26-12**) as well as the specific Category III FHR patterns that herald a significant risk for fetal acidemia. In all cases, this pattern starts with recurrent decelerations, and a terminal bradycardia will herald fetal demise if the problem is not remedied before the fetus is born. However, the onset of deeper decelerations, tachycardia, and diminution of variability can appear in any order. For example, tachycardia may develop before or after the variability starts to decrease. Similarly, the decelerations may not become visibly deeper and of longer duration until after the variability begins decreasing.

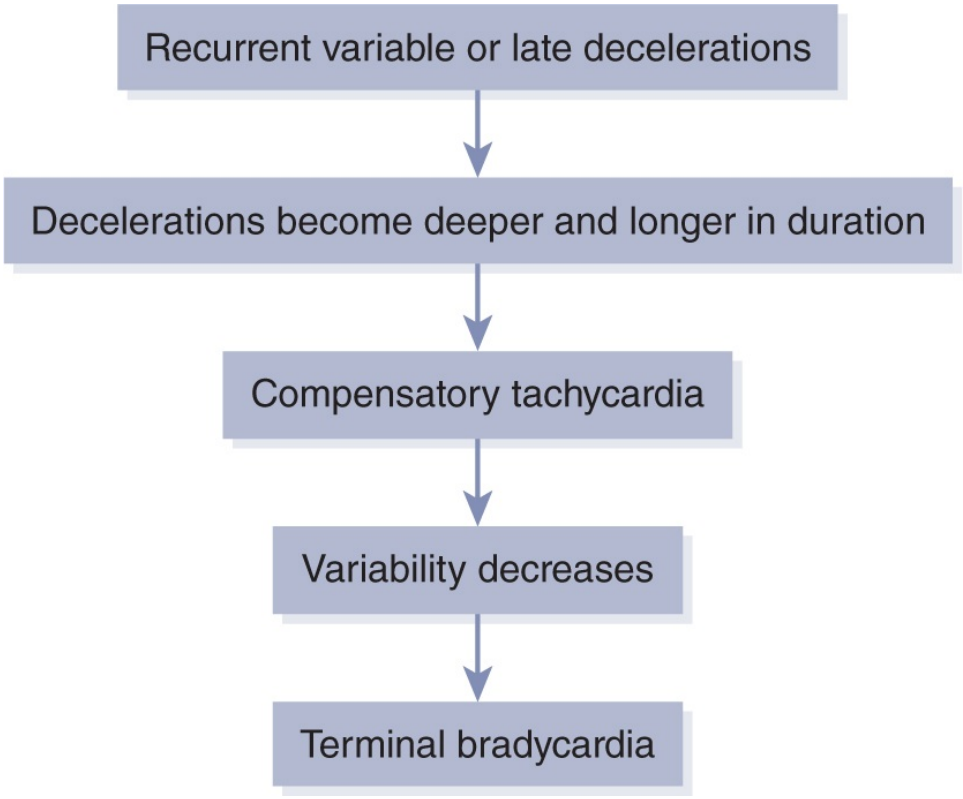


Figure 26-12 Fetal heart rate pattern evolution from normal to fetal acidemia.

Reproduced with permission from Parer JT, King TL, Ikeda T. *Electronic Fetal Heart Rate Monitoring: The 5-Tier System.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2018.[15]

Recent work using current technologies to more precisely measure both the depth and the duration of recurrent decelerations as a combined indicator have analyzed the "area under the curve" to calculate the total duration of time that an FHR remains below a specific bpm threshold during a defined period of time before birth.[48,52-54] These studies have correlated the total deceleration time to newborn outcomes. Although the results are preliminary, it appears that the total deceleration area has the best predictive power for identifying newborn acidemia.[48,52-54]

Acute Bradycardia

Sudden bradycardia can occur secondary to uterine rupture, placental abruption, cord prolapse, shoulder dystocia, maternal hypotension, amniotic fluid embolism, vasa previa, and other causes. In a retrospective analysis of cases of uterine rupture, Leung et al. found none of the newborns in the series had significant metabolic acidemia when the bradycardia-to-birth time was less than 18 minutes if a normal FHR without decelerations preceded the bradycardia.[55] By comparison, when the bradycardia was preceded by recurrent decelerations, metabolic acidemia occurred as early as 10 minutes after the bradycardia started. In general, if the FHR remains greater than 80 bpm, both coronary and cerebral oxygenation will be preserved and variability is often retained.[56] In contrast, when the FHR remains less than 80 bpm, the variability will diminish rapidly as a metabolic acidemia develops. In this scenario,

the fetus becomes unable to maintain circulation. If the bradycardia is 60 bpm or less, the fetus cannot maintain stroke volume and will be unable to oxygenate the coronary arteries.

Relationship Between Fetal Heart Rate Patterns and Newborn Outcomes

Several different newborn variables have been used in studies to identify the relationship between fetal heart rate patterns and neonatal outcomes, including Apgar scores, newborn seizures, umbilical cord blood gases, and—more recently—magnetic resonance imaging (MRI) findings in newborns with signs of neonatal encephalopathy.

Apgar Scores

The Apgar score was created to assess the adaptation of the infant to extrauterine life, rather than as a measure of prebirth acidemia. This distinction may appear trivial at first glance but, in fact, is an important one. While a 5-minute Apgar score of 7 or higher almost completely eliminates the possibility of fetal acidemia prior to birth, the converse is not true. Apgar scores can be markedly affected by drugs given prior to birth, infant resuscitation techniques, gestational age, and infection—all of which will result in a lower score.[57] Likewise, Apgar scores do not reliably predict newborn outcomes except in the extreme range of or less than 5 at 5 minutes. Therefore another index is needed that accurately reflects fetal status prior to birth and predicts newborn outcomes. Umbilical cord blood gases meet both of these requirements.

Umbilical Cord Blood Gases

Umbilical artery cord blood gases reveal the presence or absence of respiratory acidosis, metabolic acidosis, or mixed acidosis. pH is an indication of the degree of acidemia. The PCO_2 value is a reflection of the respiratory component, and the PO_2 value indicates the oxygen content. The base deficit (or base excess if negative numbers are used) indicates the severity of metabolic acidosis; it reflects how much buffer capability has been lost as lactic acid accumulates in the fetal circulation and tissue.[58] Umbilical artery cord pH and base deficit are the most useful measurements for determining the presence or absence of fetal acidemia at the time of birth. When they are in the normal range, intrapartum development of acidemia can be ruled out as the etiology of neonatal encephalopathy in a depressed or compromised newborn.[59]

Technique for Obtaining Umbilical Cord Blood Gases

Umbilical cord blood gases are obtained by collecting a sample of umbilical arterial blood and then a sample of umbilical venous blood in a heparinized syringe, from a section of umbilical cord that has been clamped at both ends. The fetal vessels on the surface of the placenta may be used if a length of umbilical cord is not available. The blood gases in the umbilical artery (UA) reflect the status of the fetus, whereas the blood gases in the umbilical vein (UV) reflect the status of the intervillous space. Thus, the values of import are those obtained from the umbilical artery. However, both UA and UV samples are needed so the

results can be correctly interpreted. When only one sample is obtained or when the samples yield the same results, one cannot be certain if the values are from the UA or the UV.

Umbilical cord blood gases should be obtained when a concern arises that the fetus might have experienced clinically significant acidemia. There are no standard guidelines that recommend indications for umbilical cord blood gas analysis. Clinical scenarios that are associated with an increased risk for metabolic acidemia in the newborn are listed in **Table 26-5**.[60]

Table 26-5	Selected Indications for Umbilical Cord Blood Gas Analysis
Cesarean section birth for fetal compromise	
Operative vaginal birth	
Preterm birth	
Fetal growth restriction	
Breech presentation	
Hypertension/preeclampsia	
Low 5-minute Apgar score	
Category III fetal heart rate pattern in last hour prior to birth	
Maternal thyroid disease	
Intrapartum fever	
Multifetal gestations	
Diabetes	
Maternal thyroid disease	
Meconium	
True knot in cord	
Substance use	

Newborns may benefit from delayed cord-clamping, and delayed cord-clamping is currently recommended for all newborns. The effect of delayed cord-clamping on umbilical cord blood gases is a current area of study.[61-63] The umbilical cord blood gas results may identify a higher P_aO_2 when samples are obtained from umbilical cords that are not immediately clamped and cut.[61-63] This phenomenon presumably occurs because there is ongoing circulation through the cord to the fetus at the time the sample is obtained. Other parameters such as pH and base excess do not appear to be affected by the few minutes of delay that allows for placental transfusion.

Interpretation of Umbilical Cord Blood Gases

The risk of neonatal morbidity is inversely related to newborn umbilical cord blood pH, although the precise level of acidemia that is pathologic for an individual newborn remains unknown. The result of fetal acidemia is an interaction between the magnitude of the stress and the responsiveness of the individual, with the latter being subject to a great deal of individual variation. In addition, although one might expect a small amount of asphyxia to cause a small amount of damage and larger amounts of asphyxia to have more adverse consequences, the

concept of a "continuum of causality" may not be correct when it comes to the fetal response to hypoxia. Instead, the fetal response to hypoxia is more likely a threshold effect—that is, damage becomes evident only after a certain threshold has been exceeded.

Despite these caveats, pathologic acidemia is generally defined as a UA pH value of less than 7.0 and a base deficit of greater than 12 mEq/L (**Table 26-6**).[64-68] This pH has consistently been identified as the inflection point at which the risk for neonatal encephalopathy becomes strongly significant statistically (RR, 16.86; 95% CI, 8.45–32.49).[65] The majority of infants with a pH less than 7.0 will be vigorous and have normal Apgar scores,[24] but approximately 20% to 24% will experience neonatal morbidity or mortality.[65] Many infants with a low pH have a respiratory acidosis that is quickly corrected following an initial few breaths. Newborns with a base deficit higher than 12 mEq/L are more likely to have significant neonatal morbidity or mortality.[64-68]

Table 26-6 Guidelines for Interpretation of Umbilical Arterial Cord Gases in Term Newborns

Diagnosis	pH	PCO_2 (mm Hg)	Bicarbonate (HCO_3)	Base Deficit[a] (mEq/L)
Normal,[b] mean (standard deviation)	7.26 (± 0.07)	53 (± 10)	22.0 (± 3.6)	4 (± 3)
Respiratory acidemia	Normal or low	High	Normal	Normal
Metabolic acidemia[c]	Low	Normal	Low	High
Mixed (metabolic–respiratory) acidemia[c]	Low	High	Low	High

[a] May be reported as base excess (negative values) or base deficit (positive value); however, the numeric value is the same.
[b] Data from term infants with an Apgar score ≥ 7 at 5 minutes.
[c] Clinical association exists with adverse neonatal outcomes when pH < 7 and base deficit > 12 mEq/L.
Based on Helwig JT, Parer JT, Kilpatrick SJ, Laros RK Jr. Umbilical cord blood acid–base state: what is normal? *Am J Obstet Gynecol.* 1996;174(6):1807-1814[58]; Gilstrap LC, Cunningham G. Umbilical cord blood acid–base analysis. In: *Supplement #4: Williams' Obstetrics.* 19th ed. Raritan, NJ: Ortho Pharmaceutical; 1994:1-24.[59]

Infants with a UA pH between 7.0 and 7.1 have an increased risk for short-term neonatal problems such as transient tachypnea, need for supplemental oxygen, or hypothermia. In some larger observational studies, although the absolute risk is still less than 2%, the relative risk for neonatal encephalopathy begins to be significant in newborns with a pH of 7.1 (RR, 5.18; 95% CI, 2.18-12.33).[65] Newborns with pH values in this range rarely experience long-term complications. A clinically significant metabolic acidemia is highly unlikely in newborns with a UA pH greater than 7.1.

Umbilical cord blood gases have an important limitation. Measurements of the UA pH and base deficit provide information about the severity of the asphyxial insult but do not provide any information about the duration, whether the insult was continuous or intermittent, or the ability of the individual fetus to compensate for the insult.

Interpretation and Management of Fetal Heart Rate Patterns

The recommended terminology and interpretation guidelines from the two NICHD expert panels apply to intrapartum FHR tracings for term fetuses. Although the same terminology is often used for preterm fetuses, there is a paucity of research on the relationship between fetal heart rate patterns and neonatal outcomes in preterm births.

Labor Admission Test with Fetal Heart Rate Monitoring

An initial EFM assessment of the fetus is commonly used in hospital birth settings to assess fetal status at the time of admission. The underlying concept assumes that if the FHR pattern shows a reactive nonstress test (NST) or a negative oxytocin challenge test (OCT), no decelerations, and moderate variability, this result can be interpreted as a reassuring sign of fetal well-being for the duration of the labor. However, the EFM assessment upon labor admission has not been shown to have adequate predictive value to warrant its use as an admission screening tool in women who are deemed to be either at high risk or at low risk for developing fetal acidemia during labor.[69] An admission EFM tracing does appear to increase the risk of having continuous monitoring throughout labor (RR, 1.30; 95% CI, 1.14–1.48) and a trend toward a higher risk for cesarean section (RR, 1.20; 95% CI, 1.00–1.44). The decision to perform an admission test does not affect rates of fetal or neonatal deaths.

FHR Interpretation During Labor: NICHD 3-Tier System

Overall, the positive predictive value of FHR monitoring is low, because the incidence of variant FHR characteristics is high and the incidence of neonatal acidemia is very low in the general population. The poor sensitivity and high false-positive rates found in studies that sought to correlate FHR patterns with newborn outcomes present a clinical conundrum that has not yet been solved.[70] In an effort to improve the predictive value of these data, several clinical algorithms that include three or more subcategories for interpretation have been developed.[11,71-76] Currently, the Parer–Ikeda 5-tier system appears to have the best accuracy in correlating FHR patterns to newborn acidemia.[77,78] Computer-aided models and models that incorporate risk screening are also being evaluated in research studies. None of these algorithms has been sufficiently tested in clinical settings.

Some data from this body of work do appear to be clinically useful. Category I FHR patterns signify an absence of acute fetal acidemia in 98% to 99% of fetuses. Thus, if no other risk factors are present, women with a Category I FHR pattern can be monitored with intermittent auscultation during labor. Conversely, category III includes the FHR patterns that likely reflect significant fetal acidemia; such a pattern requires urgent evaluation and preparations for emergent delivery.

Category II FHR patterns constitute a heterogenous group with differing risks for fetal acidemia and are an area of current research. When one of these patterns is present, management should be based first on the degree of FHR variability (and accelerations, if

present) and then on the presence or absence of recurrent decelerations (**Figure 26-13**). Management may also include use of auxiliary tests such as fetal scalp stimulation or vibroacoustic stimulation as well as intrauterine resuscitation techniques such as position change, intravenous fluid bolus, or oxygen administration. Because institutional resources must be taken into consideration when devising protocols, it is unlikely that one management paradigm for Category II FHR patterns will be applicable to all settings. Most importantly, when a Category II FHR pattern is present, it must be considered in conjunction with other factors in the clinical scenario that can independently reduce the fetus's ability to tolerate repeated hypoxic events (**Table 26-7**).

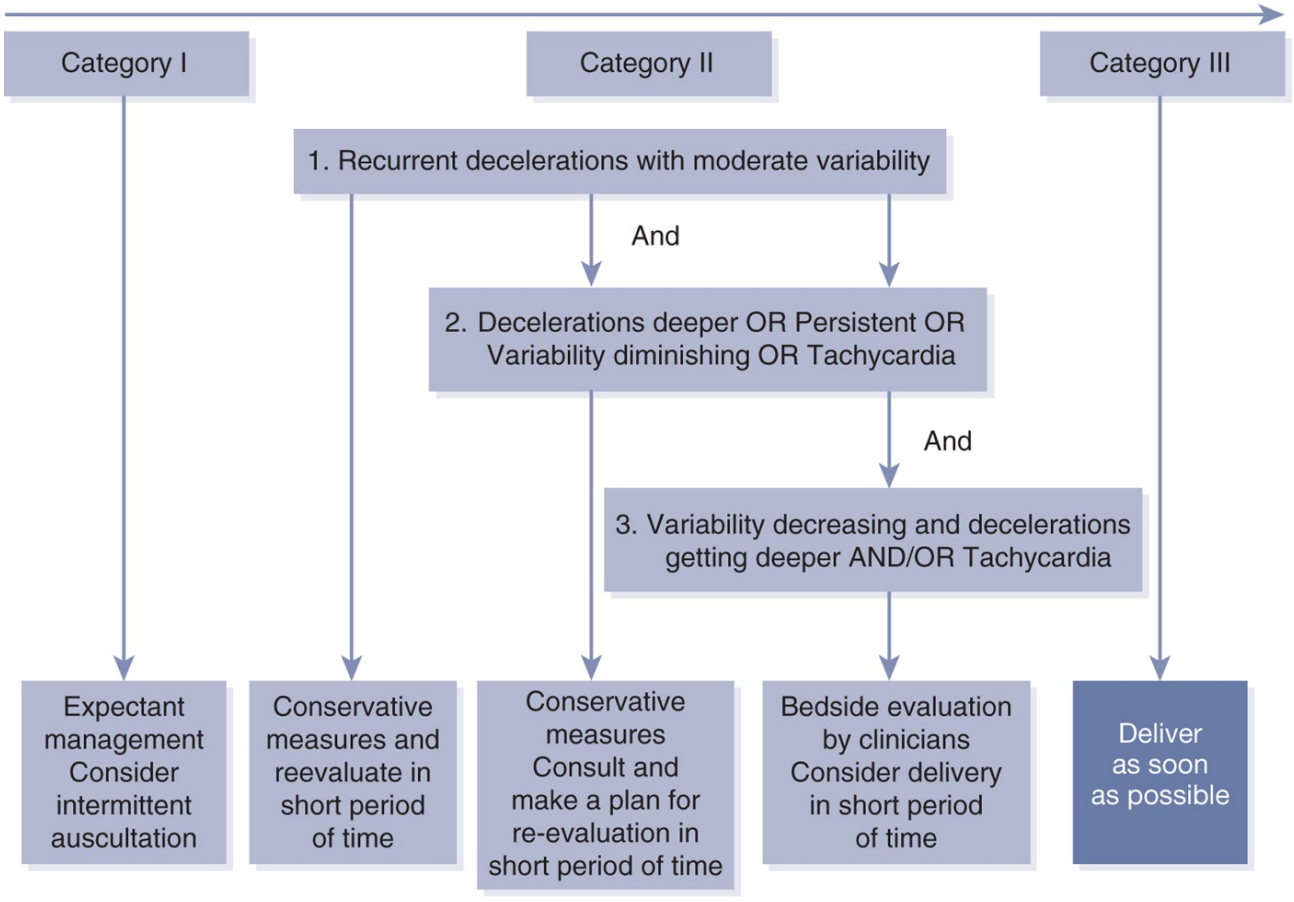


Figure 26-13 Management of FHR pattern evolution during labor.

Table 26-7	Selected Clinical Considerations That Increase the Risk for Fetal Acidemia
Maternal complications that increase the risk for uteroplacental insufficiency (e.g., preeclampsia, hypertension, diabetes, lupus erythematosus)	
Fetal factors that increase the risk for uteroplacental insufficiency (e.g., post dates, fetal growth retardation, decreased amniotic fluid index)	
Presence or absence of tachycardia and/or decreased variability (e.g., chorioamnionitis may increase vulnerability to brain damage that causes cerebral palsy)	
Success or failure of attempts to correct the underlying problem	
Acid–base status of the fetus, determined either through scalp or acoustic stimulation or via direct fetal scalp blood sampling	

Presence or absence of meconium
Gestational age (with the preterm fetus having a lower tolerance for hypoxia than the term infant)
Anticipated time until birth
Availability of consultant physician, emergency operative staff, and facility

Auxiliary Tests: Fetal Scalp Stimulation or Vibroacoustic Stimulation

Fetal scalp stimulation or vibroacoustic stimulation that elicits an acceleration provides reassurance that the fetal pH is higher than 7.2 at that time.[44] Vibroacoustic stimulation is performed using a handheld vibroacoustic stimulator (artificial larynx), which is applied to the maternal abdomen over the region of the fetal head, where it delivers a sound stimulus for 3 to 5 seconds. Approximately 50% of fetuses that do not respond with an acceleration will have a normal pH.[44] Thus, this test is useful only if the scalp stimulation, which is performed between contractions, results in an FHR acceleration. If the acceleration occurs, the clinician can be assured that the fetus is not acidemic. Conversely, when an acceleration does not occur, the presence or absence of fetal acidemia has not been determined and management must be based on other clinical factors.

Fetal scalp stimulation or vibroacoustic stimulation is most useful in the setting of minimal variability and decelerations or minimal variability without decelerations. For example, if a woman has been given opioids, the FHR variability will be iatrogenically decreased. If this woman has an FHR pattern that includes late or variable decelerations, it may be of value to perform a scalp stimulation test.

Corrective Measures for Category II FHR Patterns

Several corrective measures (also referred to as intrauterine resuscitation techniques) are used to relieve umbilical cord compression or improve uteroplacental perfusion (**Table 26-8**).[79-84] With the exception of tocolysis to decrease the frequency of uterine contractions, the evidence supporting the efficacy of these measures is not strong despite common incorporation of these techniques in most FHR management protocols.[83]

Table 26-8 Corrective Measures for Category II Fetal Heart Rate Patterns

Corrective Measure	Underlying Mechanism
Variable Decelerations	
Position change	Alters the relationship between the umbilical cord and the fetus to relieve cord compression.
Amnioinfusion	Increases the amount of amniotic fluid available in the uterus to protect the cord from compression.
Intravenous fluid bolus	Increases the amount of amniotic fluid available in the uterus to protect the cord from compression.
Open glottis pushing	Reduces the frequency and severity of variable decelerations.

Late Decelerations	
Lateral position	Increases uterine blood flow so the intervillous space is adequately perfused.
Reduction of uterine activity by discontinuing oxytocin, tocolysis	Decreases the frequency of uterine contractions so that the fetus has more time to equilibrate acid–base balance between contractions.
Intravenous fluid bolus	Increases uterine blood flow and dilutes plasma oxytocin levels.
Oxygen administration	Oxygen administration to a laboring woman increases fetal oxygenation. The effect, which lasts approximately 30 minutes after supplemental oxygen is stopped, relieves late decelerations. Because of the theoretical risk that supplemental oxygen might produce free oxygen radicals that could harm the fetus, administration of supplemental oxygen should be stopped once the FHR pattern improves.
Prolonged Decelerations	
Intravenous fluid bolus	Increases uterine blood flow and dilutes plasma oxytocin levels.
Reduction of uterine activity by discontinuing oxytocin, tocolysis	Decreases the frequency of uterine contractions so that the fetus has more time to equilibrate acid–base balance between contractions.
Correction of maternal hypotension via intravenous fluids, lateral positioning	If prolonged deceleration occurs secondary to maternal hypotension from supine positioning or following placement of regional analgesia, maternal hypotension will result in a prolonged deceleration from decreased uteroplacental perfusion. If lateral positioning and intravenous fluids do not resolve the deceleration, intravenous ephedrine can be administered.

Fetal Heart Rate Patterns and Uterine Activity

Studies of fetal oxygenation have found that an interval of approximately 60 seconds between contractions is associated with stable fetal cerebral oxygenation, whereas intervals between contractions that are less than 60 seconds are associated with lower umbilical artery pH levels.[85-87] Simpson et al. measured fetal oxygenation ($FSpO_2$) via pulse oximetry among women who were being induced at term.[85] These researchers found that when fewer than 5 contractions occurred in a 10-minute window, fetal saturation was unaffected; when 5 or more contractions occurred in a 10-minute window, fetal $FSpO_2$ dropped 20% in 30 minutes; and when more than 6 contractions occurred in 10 minutes, fetal $FSpO_2$ dropped 29% in 30 minutes.

Thus, amelioration of tachysystole can improve fetal oxygenation and mitigate the development of acidemia. In the Simpson et al. study, discontinuation of oxytocin infusion resulted in resolution of tachysystole in approximately 14.2 minutes.[85] Discontinuing oxytocin and administering an intravenous bolus (at least 500 mL) resulted in resolution in approximately 9.8 minutes. Discontinuing oxytocin, administering an intravenous bolus, and ensuring lateral positioning together resolved tachysystole in approximately 6 minutes.[85]

Administration of a tocolytic such as terbutaline (0.25 mg) will also reliably resolve tachysystole. Finally, maternal pushing with every other contraction to mitigate the effect of uterine contractions may decrease the severity or frequency of variable decelerations during second-stage pushing.

Special Populations

The fetal response to hypoxial stress can be significantly affected by fetal and maternal medical or obstetric disorders, gestational age, and in utero exposures to fetotoxic substances. For example, although the pathophysiologic pathways differ, preterm and post-term fetuses are more vulnerable to asphyxial stress than term fetuses. Furthermore, the fetus responds differently to chronic hypoxia, which is more likely to develop during the prenatal period, than to acute hypoxia, which is more likely to occur in the intrapartum period. Although a detailed discussion of these modifying factors is beyond the scope of this chapter, a brief review of the most common clinical scenarios—that is, preterm gestation, infection, and uteroplacental insufficiency—is presented here.

Preterm Fetus

The preterm fetus has different inherent FHR characteristics as compared to the term fetus. For example, the baseline heart rate is higher in the preterm fetus, averaging 155 bpm at 20 gestational weeks. FHR accelerations in association with fetal movement are a developmental process. By 28 gestational weeks, approximately 80% of fetuses will have accelerations adequate to meet the standard nonstress test criteria. Prior to 32 weeks' gestation, the amplitude of an acceleration may be only 10 bpm with a duration of 10 seconds, which satisfies the requirement for NST reactivity in a preterm fetus. Baseline variability is decreased in the preterm fetus, but no standards have been established in this regard. Short, shallow variable decelerations appear more frequently in FHR tracings of preterm fetuses.

The preterm fetus is also more vulnerable to cerebral injury from a hypoxic event and more likely to be exposed to hypoxial stress via preterm labor or infection. Thus, tachycardia should be evaluated as a possible sign of infection, even though the baseline rate in a preterm fetus may be only slightly elevated above the normal baseline rates seen in term fetuses. Pattern evolution that reflects developing acidemia, characterized by diminishing variability, deeper decelerations, and tachycardia, progresses more rapidly than the expected 1-hour duration that has been reported in term fetuses.

Infection

Infection may reduce the threshold at which hypoxia causes cerebral damage, thereby increasing the fetus's risk for subsequent cerebral palsy. Infection also increases the fetal metabolic rate, such that the fetus requires more oxygen. The cascade of pathophysiologic events linked to infection includes fetal exposure to inflammatory mediators that are introduced into the maternal and fetal circulation in response to the presence of endotoxins. Pro-inflammatory cytokines increase the permeability of the blood-brain barrier, cause endothelial damage, and increase the risk of ischemia, thrombosis, and infarction. In this way, the fetal inflammatory response to infection, in addition to the direct effects of the bacterial toxins, causes dysregulation of cerebral blood flow and subsequent hypoxic–ischemic injury.[88]

Preterm birth is closely linked to this process, as infection also initiates release of

prostaglandins, which cause preterm labor. Indeed, intra-amniotic infection is often the cause of preterm labor. The cardinal FHR characteristic associated with intra-amniotic infection is tachycardia. Treatment is reviewed in the *Complications During Labor and Birth* chapter.

Uteroplacental Insufficiency

Uteroplacental insufficiency (UPI) refers to any condition in which the placenta is not able to supply sufficient oxygen or nutrients to the fetus. It can occur at any time during the course of pregnancy, but is most often associated with preeclampsia, diabetes, maternal vascular disorders, and post dates. In the setting of uteroplacental insufficiency, the fetus can be exposed to chronic hypoxia and must balance reduced oxygen delivery with oxygen consumption. The result is a diminution of metabolic function and growth restriction. The term *intrauterine growth restriction* (IUGR) is applied when a fetus fails to reach the genetic growth potential—that is, when the fetus's estimated weight is less than 10% of the predicted weight based on gestational age.

Baseline FHR variability is generally diminished in fetuses with IUGR compared to the FHR variability commonly seen in fetuses with appropriate growth.[89-91] During labor, fetuses with UPI or IUGR are more likely to develop late decelerations than are fetuses without UPI.

Conclusion

The assessment of fetal well-being is a critical component of midwifery management of the maternal–fetal dyad during labor. Regardless of the use of intermittent auscultation, EFM, or a combination of the two, the focus must be on the care of both the woman and her fetus—not just documentation of electronically generated data.[66] Like many other areas of practice, assessment of the fetus in labor is complex, and information is being gathered continuously through ongoing research.

All midwives can use the methodologies that promote normal labor and birth while screening for and managing deviations from normal. The midwife may be the primary supporter of intermittent auscultation and the educator of other professionals in the birthing environment. Use of intermittent auscultation is a clear opportunity to blend the art and science of midwifery in a way that can make substantive positive change in clinical practice.

Resources

Organization	Description	Webpage
Electronic Fetal Monitoring: Basic and Advanced Study	Online tutorial on basic FHR monitoring	http://www.ob-efm.com
Fetal Diagnostic Center	Electronic fetal heart rate monitoring app	https://itunes.apple.com/us/app/electronic-fetal-heart-rate-monitoring/id593788448?mt=8
EFM Guide	FHR monitoring app that has basic NICHD categories with photos of each FHR characteristic.	https://itunes.apple.com/us/app/efm-guide/id375576665?mt=8

References

1. Gilstrap LC, Leveno KJ, Burris J, Williams ML, Little B. Diagnosis of birth asphyxia on the basis of fetal pH, Apgar score and newborn cerebral dysfunction. *Am J Obstet Gynecol.* 1989;161:835-840.
2. Nelson KB, Dambrosia JM, Ting TY, Grether JK. Uncertain value of electronic fetal monitoring in predicting cerebral pals *N Engl J Med*. 1996;334:613-618.
3. Yeh SY, Diaz F, Paul RH. Ten year experience of intrapartum fetal monitoring in Los Angeles County/University of Southern California Medical Center. *Am J Obstet Gynecol.* 1982;143:496-500.
4. Thacker SB, Stroup DF, Peterson HB. Efficacy and safety of intrapartum electronic fetal monitoring: an update. *Obstet Gynecol.* 1995;86(4):613-620.
5. Vintzileos AM, Nochimson DJ, Guzman ER, Knuppel RA, Lake M, Schifrin BS. Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: a meta-analysis. *Obstet Gynecol.* 1995;85(1):149-155.
6. Alfirevic Z, Devane D, Gyte GML, Cuthbert A. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. *Cochrane Database Syst Rev.* 2017;2:CD006066. doi:10.1002/14651858.CD006066.pub3.
7. MacDonald D, Grant A, Sheridan-Periera M, Chalmers I. The Dublin randomized controlled trial of intrapartum fetal hear rate monitoring. *Am J Obstet Gynecol.* 1985;152:524-539.
8. Grant S, O'Brien N, Joy MT, Hennessy E, MacDonald D. Cerebral palsy among children born during the Dublin randomiz trial of intrapartum monitoring. *Lancet.* 1989;2:1233-1235.
9. Parer JT, King T. Fetal heart rate monitoring: is it salvageable? *Am J Obstet Gynecol.* 2000;182(4):982-987.
10. National Institute of Child Health and Human Development Research Planning Workshop. Electronic fetal heart rate monitoring: research guidelines for interpretation. *Am J Obstet Gynecol.* 1997;17:1385-1390; *JOGNN*. 1997;26:635-640.
11. Macones GA, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development Research Workshop report on electronic fetal heart rate monitoring. *Obstet Gynecol.* 2008;112:661-666; *JOGNN*. 2008;37:510-515.
12. Konje JC, Howarth ES, Kaufmann P, Taylor DJ. Longitudinal quantification of uterine artery blood volume flow changes during gestation in pregnancies complicated by intrauterine growth restriction. *BJOG*. 2003;100:301-305.
13. Flo K, Wolsgaard T, Vartun A, Acharya G. A longitudinal study of the relationship between maternal cardiac output measured by impedance cardiography and uterine artery blood flow in the second half of pregnancy. *BJOG*. 2010;117:837-844.
14. Degner K, Magness RR, Shah DM. Establishment of the human uteroplacental circulation: a historical perspective. *Repro Sci.* 2017;24(5):753-761.
15. Parer JT, King TL, Ikeda T. *Electronic Fetal Heart Rate Monitoring: The 5-Tier System.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2018.
16. Parer JT. Physiological regulation of the fetal heart rate. *JOGNN*. 1976;5:265-295.
17. Fahey J, King TL. Intrauterine asphyxia: clinical implications for providers of intrapartum care. *J Midwifery Womens Health*. 2005;50:498-506.
18. Pearce W. Hypoxic regulation of the fetal cerebral circulation. *J Appl Physiol.* 2006;100(2):731-738.
19. Parer JT. Effects of fetal asphyxia on brain cell structure and function: limits of tolerance. *Comp Biochem Physiol A Molec Integr Physiol.* 1998;119(3):711-716.
20. Hagberg H, Mallard C, Rousset CI, Thornton C. Mitochondria: hub of injury responses in the developing brain. *Lancet Neurol.* 2014;13:217-232.
21. Yli B, Kjellmer I. Pathophysiology of foetal oxygenation and cell damage during labor. *Best Pract Res Clin Obstet Gynecol.* 2016;30:9-21.
22. Inder TE, Volpe JJ. Mechanisms of perinatal brain injury. *Semin Neonatol.* 2000;5(1):3-16.
23. Ugwumadu A. Infection and fetal neurologic injury. *Curr Opin Obstet Gynecol.* 2006;18:106-111.
24. Graham EM, Ruis KA, Hartman AL, Northington FJ, Fox HE. A systematic review of the role of intrapartum hypoxia–ischemia in the causation of neonatal encephalopathy. *Am J Obstet Gynecol.* 2008;199(6):587-595.
25. Shah PS, Beyene J, To T, Ohlsson A, Perlman M. Postasphyxial hypoxic–ischemic encephalopathy in neonates: outcome prediction rule within 4 hours of birth. *Arch Pediatr Adolesc Med*. 2006;160:729-736.
26. American College of Obstetricians and Gynecologists Task Force on Neonatal Encephalopathy. *Neonatal Encephalopathy and Neurologic Outcome*. 2nd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2014.
27. MacLennan A. A template for defining a causal relation between acute intrapartum events and cerebral palsy: internationa consensus statement. *BMJ*. 1999;319:1054-1059.
28. Paine LL, Payton RG, Johnson TRB. Auscultated fetal heart rate accelerations. Part I: accuracy and documentation. *J Nurse-Midwifery.* 1986;31(2):68-72.
29. Paine LL, Johnson TRB, Turner, MH, Payton RG. Auscultated fetal heart rate accelerations. Part II: an alternative to the

nonstress test. *J Nurse-Midwifery.* 1985;31(2):73-77.
30. Ayers-de-Campo D, Nogueira-Reis Z. Technical characteristics of current cardiotocograph monitors. *Best Pract Res Clin Obstet Gynecol.* 2016;30:22-32.
31. Bakker PCAM. Uterine activity monitoring during labor. *J Perinat Med*. 2007;35:468-477.
32. Caldeyro-Barcia R, Sica-Blanco Y, Poseiro JJ, et al. A quantitative study of the action of synthetic oxytocin on the pregnant human uterus. *J Pharmacol Exp Ther.* 1957;121:18-31.
33. Vintzileos AM, Smulian JC. Decelerations, tachycardia, and decreased variability: have we overlooked the significance of longitudinal fetal heart rate changes for detecting intrapartum fetal hypoxia? *Am J Obstet Gynecol*. 2016;215(3):261-264.
34. Liu L, Tuuli MG, Roehl KA, Odibo AO, Macones GA, Cahill AG. Electronic fetal monitoring patterns associated with respiratory morbidity in term neonates. *Am J Obstet Gynecol*. 2015;213(5):681.e1-681.e6.
35. Aina-Mumuney AJ, Althaus JE, Henderson JL, Blakemore MC, Johnson EA, Graham EM. Intrapartum electronic fetal monitoring and the identification of systemic fetal inflammation. *J Reprod Med*. 2007;52(9):762-768.
36. Zoeller BB. Treatment of fetal supraventricular tachycardia. *Curr Treat Options Cardiovasc Med*. 2017;19(1):7.
37. Parer JT, King TL, Flanders S, Fox M, Kilpatrick SJ. Fetal acidemia and electronic fetal heart rate patterns: is there evidence of an association? *J Matern Fetal Neonat Med*. 2006:19:289-294.
38. Fahey JO. The recognition and management of intrapartum fetal heart rate emergencies: beyond definitions and classification. *J Midwifery Womens Health*. 2014;59(6):616-623.
39. Krebs HB, Petres RE, Dunn LJ, Jordan HVF, Segreti A. Intrapartum FHR monitoring. I. Classification and prognosis of fetal heart rate patterns. *Am J Obstet Gynecol.* 1979;135(7):762-772.
40. Nijhuis JG, Prechtl HFR, Martin CB Jr, Bots RSGM. Are there behavioural states in the human fetus? *Early Hum Dev.* 1982;6:177-195.
41. Williams KP, Galerneau F. Intrapartum fetal heart rate patterns in the prediction of neonatal acidemia. *Am J Obstet Gynecol.* 2003;188:820-823.
42. Katsuragi S, Parer JT, Noda S, Onishi J, Kikuchi H, Ikeda T. Mechanism of reduction of newborn metabolic acidemia following application of a rule-based 5-category color coded fetal heart rate management framework. *J Matern Fetal Neonatal Med*. 2015;28(13):1608-1613.
43. Coletta J, Murphy E, Rubeo Z, Gyamfi-Bannerman C. The 5-tier system of assessing fetal heart rate tracings is superior to the 3-tier system in identifying fetal acidemia. *Am J Obstet Gynecol.* 2010;206:226.e1-226.e5.
44. Clark SL, Gimovsky ML, Miller FC. The scalp stimulation test: a clinical alternative to fetal scalp blood sampling. *Am J Obstet Gynecol.* 1984;148:274-277.
45. Sameshima H, Ikenoue T. Predictive value of late decelerations for fetal acidemia in unselective low-risk pregnancies. *Am J Perinatol.* 2005;22:19-23.
46. Sameshima H, Ikenoue T, Ikeda T, Kamitomo M, Ibara S. Unselected low-risk pregnancies and the effect of continuous intrapartum fetal heart rate monitoring on umbilical blood gases and cerebral palsy. *Am J Obstet Gynecol.* 2004;190:118-123.
47. Hader A, Sheiner E, Hallak M, Katz M, Mazor M, Shoham-Vardi I. Abnormal fetal heart rate tracing patterns during the first stage of labor: effect on perinatal outcome. *Am J Obstet Gynecol.* 2001;185:863-868.
48. Hamilton E, Warrick P, O'Keefe D. Variable decelerations: do shape and size matter? *J Matern Fetal Neonat Med.* 2012;25(6):648-653.
49. Spong CY, Collea JV, Engliton GS, Ghidini A. Characteristics and prognostic significance of variable decelerations in the second stage of labor. *Am J Perinatol.* 1998;15(6):369-374.
50. Jaeggi ET, Masalo M. Fetal brady- and tachyarrhythmias: new and accepted diagnostic and treatment methods. *Semin Fetal Neonat Med*. 2005;10:504-511.
51. Donofrio MT, Moon-Grady AJ, Hornberger, et al., for American Heart Association Adults with Congenital Heart Disease Joint Committee of the Council on Cardiovascular Disease in the Young and Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and Council on Cardiovascular and Stroke Nursing. Diagnosis and treatment of fetal cardiac disease: a scientific statement from the American Heart Association. *Circulation*. 2014;129(21):2183-2242.
52. Cahill AG, Roehl KA, Odibo AO, Macones GA. Association and prediction of neonatal acidemia. *Am J Obstet Gynecol.* 2012;207:206.e1-206.e8.
53. Giannubilo SR, Buscicchio G, Gentilucci L, Palla GP, Tranquilli AL. Deceleration area of fetal heart rate trace and fetal acidemia at delivery: a case-control study. *J Matern Fetal Neonatal Med*. 2007;20(2):141-144.
54. Tranquilli AL, Biagini A, Greco P, Di Tommaso M, Giannubilo SR. The correlation between fetal bradycardia area in the second stage of labor and acidemia at birth. *J Matern Fetal Neonatal Med*. 2013;26(14):1425-1429.
55. Leung TY, Chung PW, Rogers MS, Sahota DS, Lao TT, Hung Chung TK. Urgent cesarean delivery for fetal bradycardia. *Obstet Gynecol.* 2009;114(5):1023-1038.
56. Takano Y, Furukawa S, Ohashi M, Michikata K, Sameshima H, Ikenoue T. Fetal heart rate patterns related to neonatal brain damage and neonatal death in placental abruption. *J Obstet Gynecol.* 2013;39(1):61-66.
57. *The Apgar Score*. ACOG Committee Opinion No. 333. Washington, DC: American College of Obstetricians and

Gynecologists; May 2006.
58. Helwig JT, Parer JT, Kilpatrick SJ, Laros RK Jr. Umbilical cord blood acid-base state: what is normal? *Am J Obstet Gynecol*. 1996;174(6):1807-1814.
59. Gilstrap LC, Cunningham G. Umbilical cord blood acid–base analysis. In: *Supplement #4: Williams' Obstetrics*. 19th ed. Raritan, NJ: Ortho Pharmaceutical; 1994:1-24.
60. ACOG Committee on Obstetric Practice. ACOG Committee Opinion No. 348, November 2006: umbilical cord blood gas and acid–base analysis. *Obstet Gynecol*. 2006;108:1319-1322.
61. Valero J, Desantes D, Perales-Puchalt A, Rubio J, Diago Almela VJ, Perales A. Effect of delayed umbilical cord clamping on blood gas analysis. *Eur J Obstet Gynecol Reprod Biol*. 2012;162(1):21-23.
62. De Paco C, Florido J, Garrido MC, Prados S, Navarrete L. Umbilical cord blood acid–base and gas analysis after early versus delayed cord clamping in neonates at term. *Arch Gynecol Obstet*. 2011;283(5):1011-1014.
63. Andersson O, Hellström-Westas L, Andersson D, Clausen J, Domellöf M. Effects of delayed compared with early umbilical cord clamping on maternal postpartum hemorrhage and cord blood gas sampling: a randomized trial. *Acta Obstet Gynecol Scand*. 2013;92(5):567-574.
64. Ross MG, Gala R. Use of umbilical artery base excess: algorithm for the timing of hypoxic injury. *Am J Obstet Gynecol*. 2002;187:1-9.
65. Low JA, Lindsay BG, Derrick EJ. Threshold of metabolic acidosis associated with newborn complications. *Am J Obstet Gynecol*. 1997;177:1391-1394.
66. Malin GL, Morris RK, Khan KS. Strength of association between umbilical cord pH and perinatal and long term outcomes systematic review and meta-analysis. *BMJ*. 2010;340:c1471.
67. Yeh P, Emary K, Impey L. The relationship between umbilical cord arterial pH and serious adverse neonatal outcome: analysis of 51,519 consecutive validated samples. *BJOG*. 2012;119:824.
68. Knutzen L, Svirko E, Impey L. The significance of base deficit in acidemic term neonates. *Am J Obstet Gynecol*. 2015;213:373.e1.
69. Devane D, Lalor JG, Daly S, Fiol V, Pons J, Alonso J. Cardiotocography versus intermittent auscultation of fetal heart on admission to labour ward for assessment of fetal wellbeing. *Cochrane Database Syst Rev*. 2012;2:CD005122.
70. Larma JD, Silva AM, Holcroft CJ, Thompson RE, Donohue PK, Graham EM. Intrapartum electronic fetal heart rate monitoring and the identification of metabolic acidosis and hypoxic–ischemic encephalopathy. *Am J Obstet Gynecol*. 2007;197(3):301.e1-301.e8.
71. Parer JT, Ikeda T. A framework for standardized management of intrapartum fetal heart rate patterns. *Am J Obstet Gynecol*. 2007;197:26.e1-26.e6.
72. Clark SL, Nageotte MP, Garite TJ, et al. Intrapartum management of Category II fetal heart rate tracings: towards standardization of care. *Am J Obstet Gynecol*. 2013;209(2):89-97.
73. Soncini E, Paganelli S, Vezzani C, Gargano G, Giovanni Battista LS. Intrapartum fetal heart rate monitoring: evaluation of a standardized system of interpretation for prediction of metabolic acidosis at delivery and neonatal neurological morbidity. *J Matern Fetal Neonatal Med*. 2014;27(14):1465-1469.
74. Miller DA. Intrapartum fetal heart rate monitoring: a standardized approach to management. *Clin Obstet Gynecol*. 2011;54(1):22-27.
75. Ayres-de-Campos D, Spong CY, Chandraharan E; FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: cardiotocography. *Int J Gynaecol Obstet*. 2015;131(1):13-24.
76. Okai T, Ikeda T, Kawarabayashi T, et al.; Perinatology Committee of the Japan Society of Obstetrics and Gynecology. Intrapartum management guidelines based on fetal heart rate pattern classification. *J Obstet Gynaecol Res*. 2010;36(5):925-928.
77. Katsuragi S, Parer JT, Noda S, Onishi J, Kikuchi H, Ikeda T. Mechanism of reduction of newborn metabolic acidemia following application of a rule-based 5-category color coded fetal heart rate management framework. *J Matern Fetal Neonatal Med*. 2015;28(13):1608-1613.
78. Ikeda S, Okazaki A, Miyazaki K, Kihira K, Furuhashi M. Fetal heart rate pattern interpretation in the second stage of labo using the five-tier classification: impact of the degree and duration on severe fetal acidosis. *J Obstet Gynaecol Res*. 2014;40(5):1274-1280.
79. Simpson KR. Intrauterine resuscitation during labor: should maternal oxygen administration be a first-line measure? *Semin Fetal Neonatal Med*. 2008;13(6):362-367.
80. Simpson KR. Intrauterine resuscitation during labor: review of the current methods and supportive evidence. *J Midwifery Womens Health*. 2007;52: 229-237.
81. Hofmeyr GJ, Lawrie TA. Amnioinfusion for potential or suspected umbilical cord compression in labour. *Cochrane Database Syst Rev*. 2012;1:CD000013. doi:10.1002/14651858.CD000013.pub2.
82. Garite JT, Nageotte MP, Parer JT. Should we really avoid giving oxygen to mothers with concerning fetal heart rate patterns? *Am J Obstet Gynecol*. 2015;212(4):459-460.
83. Bullens LM, van Runnard Heimel PJ, van der Hout-van der Jagt MB, Oei SG. Interventions for intrauterine resuscitation i

suspected fetal distress during term labor: a systematic review. *Obstet Gynecol Surv*. 2015;70(8):524-539.
84. Hamel MS, Hughes BL, Rouse DJ. Whither oxygen for intrauterine resuscitation? *Am J Obstet Gynecol*. 2015;212(4):461-462.
85. Simpson KR, James DC. Effects of oxytocin-induced uterine hyperstimulation during labor on fetal oxygen status and feta heart rate patterns. *Am J Obstet Gynecol*. 2008;199:34.e1-34.e5.
86. Simpson KR. Clinician's guide to the use of oxytocin for labor induction and augmentation. *J Midwifery Womens Health*. 2011;56:214-221.
87. Bakker PCAM, Kurver PHJ, Kuik DJ, Van Geijn HP. Elevated uterine activity increases the risk of fetal acidosis at birth. *Am J Obstet Gynecol*. 2007;196:313.e1-313.e6.
88. Kim CJ, Romero R, Chaemsaithong P, Chaiyasit N, Yoon BH, Kim YM. Acute chorioamnionitis and funisitis: definition, pathologic features, and clinical significance. *Am J Obstet Gynecol*. 2015;213(4 suppl):S29-S52.
89. Stampalija T, Casati D, Monasta L, et al. Brain sparing effect in growth-restricted fetuses is associated with decreased cardiac acceleration and deceleration capacities: a case-control study. *BJOG*. 2015;123(12):1947-1954.
90. Henson G, Dawes GS, Redman CW. Characterization of the reduced heart rate variation in growth-retarded fetuses. *Br J Obstet Gynaecol*. 1984;91(8):751-755.
91. Nijhuis IJ, ten Hof J, Mulder EJ, et al. Fetal heart rate in relation to its variation in normal and growth retarded fetuses. *Eu J Obstet Gynecol Reprod Biol*. 2000;89(1):27-33.

APPENDIX

26A

Intermittent Auscultation During Labor

TEKOA L. KING

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The purpose of intrapartum assessment of the fetal heart rate (FHR) is to monitor fetal well-being over the course of labor and to identify the fetus at risk for clinically significant acidemia. Effective methods of evaluation and accurate interpretation of the FHR during labor have long been the subjects of clinical debate. Continuous electronic fetal heart rate monitoring (EFM) is the most commonly used method of intrapartum fetal surveillance, but has not been found to be more effective than intermittent auscultation (IA).[1] Professional organizations' recommendations for fetal heart rate monitoring during labor are primarily based on protocols used in randomized clinical trials conducted during the 1990s that compared IA to EFM.[1-4] Handheld Doppler or Pinard-type fetoscopes were used for the IA groups in these trials, and those women had continuous one-to-one nursing during labor. Since those studies were performed, knowledge of the relationship between specific FHR patterns and adverse neonatal outcomes has improved, so it cannot be assumed that the IA protocols currently in use reflect current knowledge about the physiology of fetal heart rate characteristics during labor.[5,6]

Intermittent auscultation is typically conducted using a handheld Doppler fetoscope. Pinard-type fetoscopes may be used prenatally, but they have limitations for intrapartum use. A handheld Doppler device can effectively monitor the fetus if the technique is performed correctly. Conversely, intermittent auscultation can miss significant fetal heart rate decelerations if the technique used is not accurate. This appendix presents the technique for intermittent auscultation, interpretation of findings from IA, and management recommendations based on current knowledge of fetal heart rate characteristics.

Intermittent Auscultation Versus Electronic Fetal Heart Rate Monitoring

Both EFM and IA have benefits and limitations. Auscultation allows the woman freedom of movement and permits fetal assessment if the woman is in water. Auscultation also requires one-to-one continuous care, but may not be technically feasible for women who have high body

mass indices (BMIs) or when women are in certain positions such as on hands and knees. EFM will monitor the fetus continuously during labor and provides a record that can be examined by others as needed, often from a central monitoring site. However, EFM limits the woman to a bed or sitting near a bed unless telemetry units are available for her use. Moreover, when a healthcare provider monitors the fetus from a central monitor, the only supportive person for the woman may no longer be in the room, and she may miss the additional advantages of having someone with her continuously throughout her labor.

The randomized controlled trials (RCTs) that compared EFM with IA did not find that either modality was a better option for improving Apgar scores, lowering the incidence of cerebral palsy, or improving perinatal mortality rates.[1] EFM was associated with a significant increase in the rate of cesarean section compared to IA. The value of these findings and limitations of the RCTs are reviewed in this chapter and extensively elsewhere.[7,8] Until another RCT comparing EFM to IA that incorporates current knowledge about which FHR patterns are associated with acidemia is published, it is unlikely that the question of which modality is superior will be answered. Therefore, the focus of this appendix is on safe use of IA based on current knowledge.

Fetal Heart Rate Characteristics Detectable on Intermittent Auscultation

Table 26A-1 lists the FHR characteristics that can be detected via IA and EFM. Auscultation can determine the baseline FHR, changes in the rhythm of the heart rate, and accelerations.[1,9-11] The *Auscultated Acceleration Test* appendix of the *Pregnancy-Related Conditions* chapter describes the methods used to identify and document FHR accelerations in nonlaboring women. Although these methods have not been assessed in a laboring population, it is reasonable to assume that they are valid. Studies assessing reliability of IA use to document accelerations during labor have not been conducted. IA cannot determine FHR variability.[6] Decelerations can be identified as a decrease in the heart rate, but specific timing of the decelerations in relation to uterine contractions cannot be determined with assurance, nor can the degree of decline of the heart rate, such as “sudden” or “gradual,” be confirmed. Thus, decelerations cannot be categorized as early, variable, or late in character. Instead, auscultation of decreases in the FHR can be described as “brief” or “prolonged” based on the auscultated duration and the slowest rate counted or registered on the Doppler device.

Table 26A-1 FHR Characteristics Determined via Fetoscope, Doppler, and Electronic Fetal Heart Rate Monitoring

FHR Characteristic[a]	Fetoscope	Doppler Without Paper Printout	Electronic FHR Monitor
Variability	No	No	Yes
Baseline rate	Yes	Yes	Yes
Accelerations	Detects increases[b]	Detects increases[b]	Yes
Decelerations	Detects decreases in FHR and duration of decrease	Detects decreases in FHR and duration of decrease	Differentiates types of decelerations as early, late or variable
Rhythm[c]	Yes, arrhythmia will be audible	Yes, arrhythmia will be audible	Yes, arrhythmia will be audible and recorded on a paper graph. The type of arrhythmia cannot be determined. May double or halve the count and report an inaccurate FHR if arrhythmia is present.
Differentiation of maternal and fetal heart rate	Yes	May detect maternal heart rate instead of fetal heart rate	May detect and record maternal heart rate instead of fetal heart rate

Abbreviations: FHR, fetal heart rate; NICHD, National Institute of Child Health and Human Development.
[a] Definitions of each FHR characteristic per the NICHD 2008 criteria.[3]
[b] Per method described by Paine et al. in pregnant women not in labor.[9,10]
[c] Determined as regular or irregular. None of these devices can diagnose the type of fetal arrhythmia.
Modified with permission from Intermittent auscultation for intrapartum fetal heart rate surveillance: American College of Nurse-Midwives [erratum, *J Midwifery Womens Health.* 2016;61(1):134]. *J Midwifery Womens Health.* 2015;60(5):626-632.[2] © 2015, with permission from Wiley.

Moderate FHR variability is the most reliable indicator that a fetus is not experiencing clinically significant acidemia. As a consequence, the inability of IA to detect FHR variability or to discriminate between types of FHR decelerations must be addressed. Despite the fact that it cannot detect FHR variability and despite the lack of direct scientific study of this technique, IA is considered a viable option for low-risk women at term for several reasons:

- IA is recommended only for women who do not have an a priori risk (known risk) for developing fetal acidemia during labor. The incidence of clinically significant metabolic acidemia in newborns in a healthy population of women is approximately 3 per 1000 births.[12,13] Of these infants, only a small proportion will have adverse outcomes. A priori risk screening is a critical step in identifying women who are candidates for this method of fetal surveillance. Further evidence that supports risk screening comes from large population-based studies of out-of-hospital birth. Very-low-risk women at term who are monitored with IA during labor have a very low risk for adverse maternal and newborn outcomes during labor and birth.[14-18]
- When significant fetal acidemia does develop during the intrapartum period in a previously healthy fetus, it occurs over a period of time during which the fetus will develop recurrent FHR decelerations that are detectable on IA[19] or as an acute unremitting bradycardia. Although the incidence of acute bradycardia in a rigorously screened low-risk population is quite low, this is an important fact that needs to be included in shared decision making when a woman is choosing a mode of fetal surveillance.
- Randomized controlled trials of IA included one-to-one nursing care, which may have an

independent positive effect on labor outcomes. Thus, use of IA presumes continuous labor support is in effect.

- IA will not detect a sinusoidal FHR pattern if decelerations are not also present. This rare FHR pattern is associated with placental abruption, Rh isoimmunization, and maternal–fetal hemorrhage. A woman with symptoms of one of these conditions is not a candidate for IA.

Professional Guidelines for Intermittent Auscultation

The American College of Nurse-Midwives (ACNM) has published a clinical bulletin that summarizes the technique and interpretation of IA.[2] The method described in this appendix is concordant with the ACNM guidelines. Individual institutions or practices may develop protocols specific to their settings and populations. Protocols for IA should define the method of conducting IA, frequency and duration of IA, indications for IA, and indications for transfer to EFM.

Frequency, Timing, and Technique for Intermittent Auscultation for Women in Labor

ACNM recommends auscultating the FHR every 15 to 30 minutes in the active phase of labor and every 5 minutes in the second stage of labor.[2] The frequency and timing of IA are based on the frequency and timing used in the RCTs comparing EFM with IA. However, the protocols designed for RCTs were not tested for effectiveness and have some important limitations.

Frequency

Strict adherence to a 15- or 30-minute interval fails to account for changes in labor status that could potentially affect the FHR. The frequency of auscultation should be individualized based on labor characteristics such as the frequency of contractions, clinical signs of fetal descent (e.g., spontaneous rupture of membranes, bloody show, urge to push, nonrecurrent decelerations), and institution of hydrotherapy or other interventions that may affect the FHR.

Duration of Auscultation

The original RCTs recommended auscultating between contractions, and for many years professional organizations' guidelines maintained this recommendation. However, listening between contractions can result in missing clinically significant variable decelerations that occur during a contraction and resolve as the contraction ends. For this reason, the current guidelines recommend listening through a contraction and for a period of time after the contraction ends.[1,4] If no FHR decreases are noted, in subsequent assessments the midwife may begin auscultation at the peak of a contraction and continue listening for 30 to 60 seconds after the contraction is over. Listening between contractions is not effective and should not be part

of the technique.

Multicount Strategy

A multicount technique has been shown to be more reliable than counting once throughout the period of assessment.[20] In a study conducted by Shiffrin et al., the multicount strategy identified approximately 93% of FHR decelerations; by comparison, a single-count strategy identified 74% of the FHR decelerations.[15] When using a multicount strategy, the listener counts the FHR for a period of 6, 10, or 15 seconds several times over the course of the listening period. A defined rest period of 5 or 10 seconds occurs between each count. Each count is then multiplied by the appropriate number to obtain an FHR-per-minute rate. The counts can be plotted to diagram the FHR changes over the course of the auscultation period, as shown in **Figure 26A-1**. Some anecdotal evidence suggests that this technique will reveal FHR variability, but this relationship has not been evaluated scientifically.

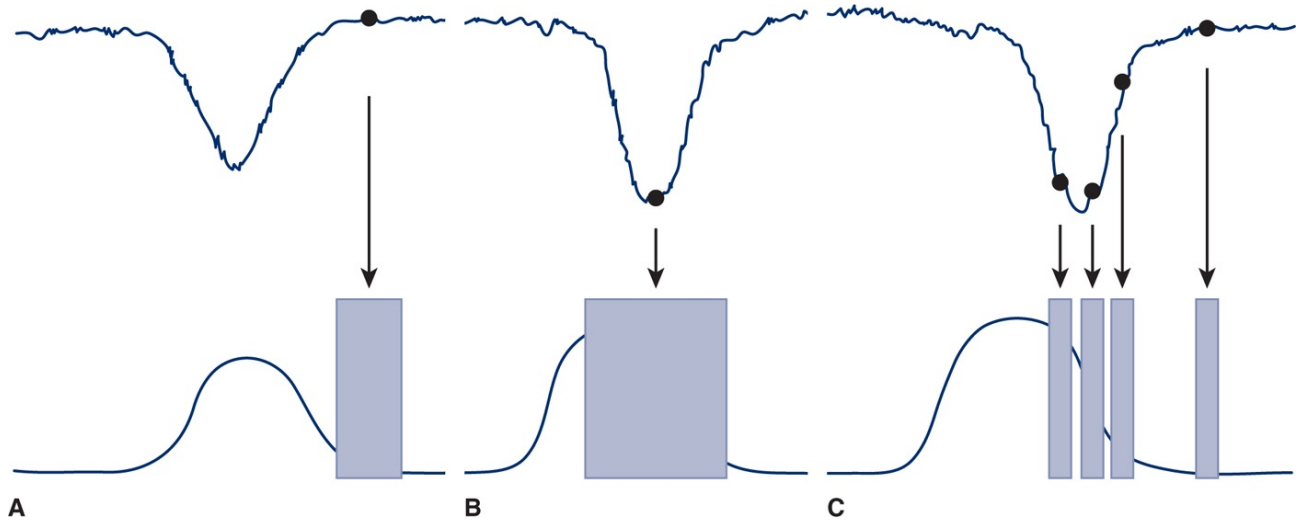


Figure 26A-1 Multicount strategy for assessing the fetal heart rate via intermittent auscultation. **A.** Single count strategy for < 1 minute between contractions may miss a deceleration. **B.** Single count strategy for 30–60 seconds that starts after the peak of the contraction will detect presence of a deceleration qualitatively. **C.** Multicount strategy for 10 seconds with a 5–10 second break between counts allows the clinician to graphically sketch the deceleration.

Interpretation of Intermittent Auscultation Fetal Heart Rate Characteristics

FHR changes identifiable on IA have been evaluated in concert with the 2008 National Institute of Child Health and Human Development (NICHD) interpretative categories for EFM for use in the United States. FHR changes detected on IA are classified as either Category I or Category II, as outlined in **Table 26A-2**.[2] There is no Category III when using IA, because Category III FHR patterns have no variability and cannot be detected via IA.

Table 26A-2	Interpretation of Auscultation Findings Relative to NICHD 3-Tier Categories
Category 1 FHR Characteristics by Auscultation	**Category II FHR Characteristics by Auscultation**
FHR baseline between 110 and 160 bpm with regular rhythm No FHR decelerations from the baseline FHR increases (accelerations) of 15 seconds in duration and amplitude from the baseline may or may not be present, but should be assessed for and documented if present	Tachycardia (baseline > 160 bpm) or bradycardia (baseline < 110 bpm) Audible irregular rhythm Presence of FHR decreases or decelerations from the baseline

Abbreviations: FHR, fetal heart rate; NICHD, National Institute of Child Health and Human Development.

Management of Category II Fetal Heart Rate Changes on Intermittent Auscultation

Category II FHR changes require increased surveillance. A longer listening period may be initiated, the frequency of auscultation increased, or transfer to EFM undertaken. If recurrent decelerations are noted or if the FHR is bradycardic, several commonly performed interventions should be initiated. The woman should be turned to her side and the FHR immediately reevaluated. Oxygen therapy and intravenous fluids may be initiated. If the Category II FHR pattern does not resolve in a short period of time, frequent auscultation and documentation of results should continue while transferring the woman to a setting where EFM and operative delivery are available.

Documentation of Intermittent Auscultation

The type of Doppler technology used and the multicount procedure employed are documented in the health record. The graph for documentation of IA should be simple and straightforward, to facilitate documentation.

Fetoscopes

Several Pinard-type stethoscopes are available, but all rely on bone conduction from the listener's cranium to amplify the sound. The most commonly used alternative is the Allen fetoscope, with either short or long tubing. The DeLee-Hillis fetoscope has a headpiece that fits over the listener's head, which facilitates the bone conduction.

The Pinard-type fetoscopes identify the sounds associated with the opening and closing of ventricular valves and must be placed on the maternal abdomen where the fetal heart is closest, over the fetal scapula or chest (**Figure 26A-2**). If used, the bell must be placed tightly against the maternal abdomen to achieve a good air seal, and the headpiece must be placed against the listener's forehead to take advantage of bone conduction



Figure 26A-2 Pinard-type fetoscopes. **A.** Allen fetoscope with short tubing. **B.** Allen fetoscope with long tubing. **C.** DeLee-Hillis fetoscope. **D.** Pinard fetoscope.

Although some of the RCTs of EFM versus IA used a Pinard-type fetoscope during labor, these devices are not good choices for detecting the FHR during a contraction. Given current knowledge about the role of FHR decelerations as a reflection of developing fetal acidemia, most midwives in developed nations do not use these devices for monitoring the FHR during labor today, and they are not recommended by the authors of this text.

Doppler Fetoscopes

Doppler devices use ultrasound to detect the movement—that is, opening and closing—of the ventricular valves by noting the change in ultrasound wave length that is caused by heart movement. The device converts the wavelength change into a visual digital recording and audible sound.

Doppler devices require use of a gel between the face of the transducer and the maternal skin to ensure transmission of sound without air interference. Some handheld devices will generate a printout record. Most Doppler fetoscopes are waterproof and can be used to record the FHR when a woman is in water.

Technique for Performing Intermittent Auscultation

1. Determine the fetal lie to optimize the quality of the heart sounds. Listening through the fetal back provides the clearest sound.
2. Determine the duration and frequency of uterine contractions.
3. Determine baseline FHR:
   a. If using a Doppler device, check the maternal radial pulse at the same time to be certain the Doppler fetoscope is detecting the FHR. The maternal heart rate may be documented in the woman's record.
   b. Perform the initial auscultation for a minimum of 60 seconds, between contractions, to determine the baseline FHR and to listen for regularity of the rate.
   c. Listen between contractions for several intervals within a 10-minute period to confirm consistency of the FHR and to identify the baseline.

   d. Count for 6 to 10 seconds several times, with a 5- or 10-second pause between counts; then multiply each count by 10 or 6, respectively, to obtain the FHR-per-minute numeric value.
4. Listen for the presence of FHR accelerations. Noting the presence of FHR accelerations can be done qualitatively or quantitatively. Accelerations do not need to be present to identify the FHR as Category I if the FHR rate has a normal baseline and no decelerations are present. However, FHR accelerations are a sign of fetal well-being when present and should be assessed for and documented if auscultated. The *Auscultated Acceleration Test* appendix in the *Pregnancy-Related Conditions* chapter describes the procedure for detecting FHR accelerations quantitatively.
5. Determine the presence of FHR decelerations. Begin listening at the peak of the contraction and continue for at least 30 to 60 seconds after the contraction is over.
   a. If a deceleration is heard, listen through successive contractions or several contractions in a 10-minute window to determine whether they are recurrent or nonrecurrent.
   b. Use a multicount method to document the drop in FHR baseline and depth of the decrease.
6. Document the FHR-per-minute rate for each assessment and inform the laboring woman and her support team of the findings.

References

1. Alfirevic Z, Devane D, Gyte GML, Cuthbert A. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. *Cochrane Database Syst Rev.* 2017;2:CD006066. doi:10.1002/14651858.CD006066.pub3.
2. Intermittent auscultation for intrapartum fetal heart rate surveillance: American College of Nurse-Midwives [erratum, *J Midwifery Womens Health*. 2016;61(1):134]. *J Midwifery Womens Health*. 2015;60(5):626-632.
3. American College of Obstetricians and Gynecologists. Practice Bulletin No. 116: management of intrapartum fetal heart rate tracings. *Obstet Gynecol.* 2010;116:1232-1240.
4. Lewis D, Downe S; FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: Intermittent auscultation. *Int J Gynaecol Obstet.* 2015;131(1):13-24.
5. Parer JT, King TL, Flanders S, Fox M, Kilpatrick SJ. Fetal acidemia and electronic fetal heart rate patterns: is there evidence of an association? *J Matern Fetal Neonatal Med*. 2006:19:289-294.
6. Sholapurkar SL. Intermittent auscultation in labor: could it be missing many pathological (late) fetal heart rate decelerations? Analytical review and rationale for improvement supported by clinical cases. *J Clin Med Res.* 2015;7(12):919-925.
7. Parer JT, King T. Fetal heart rate monitoring: is it salvageable? *Am J Obstet Gynecol.* 2000;182(4):982-987.
8. Resnik R. Electronic fetal monitoring: the debate goes on. . . and on. . . and on. *Obstet Gynecol.* 2013;121(5):917-918.
9. Paine LL, Payton RG, Johnson TRB. Auscultated fetal heart rate accelerations: Part I. Accuracy and documentation. *J Nurse-Midwifery.* 1986;31(2):68-72.
10. Paine LL, Johnson TR, Turner MH, Payton RG. Auscultated fetal heart rate accelerations: Part II. An alternative to the nonstress test. *J Nurse-Midwifery.* 1989;31(2):73-77.
11. Paine LL, Benedict MI, Strobino DM, Gegor CL, Larson EL. A comparison of the auscultated acceleration test and the nonstress test as predictors of perinatal outcomes. *Nurs Res.* 1992;41(2):87-91.
12. Graham EM, Petersen SM, Christo DK, Fox HE. Intrapartum electronic fetal heart rate monitoring and the prevention of perinatal brain injury. *Obstet Gynecol.* 2006;108(3 pt 1):656-666.
13. Kurinczuk JJ, White-Koning M, Badawi N. Epidemiology of neonatal encephalopathy and hypoxic-ischaemic encephalopathy. *Early Hum Develop*. 2010;86(6):329-338.
14. de Jonge A, van der Goes BY, Ravelli ACJ, et al. Perinatal mortality and morbidity in a nationwide cohort of 529,688 low-risk planned home and hospital births. *BJOG*. 2009;116(9):1177-1184.

15. Stapleton SR, Osborne C, Illuzzi J. Outcomes of care in birth centers: demonstration of a durable model. *J Midwifery Womens Health*. 2013;58(1):1-12.
16. Olsen O, Clausen JA. Planned hospital birth versus planned home birth. *Cochrane Database Syst Rev*. 2012;9:CD000352. doi:10.1002/14651858.CD000352.pub2.
17. Cheyney M, Bovbjerg M, Everson C, Gordon W, Hannibal D, Vedam S. Outcomes of care for 16,924 planned home births in the United States: the Midwives Alliance of North America Statistics Project, 2004 to 2009. *J Midwifery Womens Health*. 2014;59(1):17-27.
18. Birthplace in England Collaborative Group, Brocklehurst P, Hardy P, et al. Perinatal and maternal outcomes by planned place of birth for healthy women with low risk pregnancies: the Birthplace in England national prospective cohort study. *BMJ*. 2011;343:d7400.
19. Low JA, Pickersgill H, Killen H, Derrick EJ. The prediction and prevention of intrapartum fetal asphyxia in term pregnancies. *Am J Obstet Gynecol*. 2001;184(4):724-730.
20. Shiffrin BS. The accuracy of auscultatory detection of fetal cardiac decelerations: a computer simulation. *Am J Obstet Gynecol*. 1992;166(2):566-576.

APPENDIX

26B

Fetal Scalp Electrode

TEKOA L. KING

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A fetal scalp electrode (FSE) is a very small, thin, spiral electrode that, when attached to the fetal scalp, enables fetal electrocardiogram (ECG). The FSE is attached to a cord that connects to an electronic fetal monitor, which turns the ECG into an audible and written continuous recording. An FSE requires that membranes are ruptured and the cervix is at least 1 to 2 centimeters dilated.

This device is not recommended for routine use. An FSE is used *only* when a continuous legible recording is considered necessary as a basis of clinical management and an external Doppler fetoscope is *not* able to supply such a record. Although there are no specific indications for use of an FSE in the United States, it is commonly used when an external Doppler device is unable to continuously detect the fetal heart rate (FHR) and the fetus is at risk for metabolic acidemia or perinatal death. Examples of such situations include severe preeclampsia or prior cesarean section birth such that an increased risk for uterine rupture is now present.

First-generation electronic monitors exaggerated FHR variability when the abdominal Doppler device was used, so an FSE would be applied to provide a more accurate rendition of FHR variability. Today, however, electronic fetal monitors have an autocorrelation feature that allows portrayal of FHR variability with an accuracy almost equal to that obtained when an FSE is used.[1] Thus, the need to better assess variability is rarely an indication for use of an FSE if a continuous legible tracing is present. Nevertheless, it is important to note that the external Doppler device can erroneously detect a maternal heart rate or double or halve the signal, which can, in rare situations, confound interpretation of its output.[1,2] In turn, doubt about the Doppler signal's accuracy in portraying a fetal heart rate is another indication for use of an FSE.

The FSE penetrates into the fetal scalp approximately 2 mm. Use of this device is contraindicated in women who have infections that might potentially be vertically transmitted to the fetus, such as active herpes, hepatitis, or human immunodeficiency virus (HIV). An FSE is also contraindicated if the fetus has a known or possible coagulation disorder. Some clinicians consider chorioamnionitis to be a relative contraindication to the use of an FSE.

This type of electrode should not be attached if the fetus is in a face presentation, or if the presentation is unknown. Likewise, it should not be placed on genitalia in breech presentations, or over the fontanelle in a vertex presentation.[1]

Complications following FSE use may include scalp laceration, localized infection, and (rarely) abscess. Some case reports describe facial injury, or insertion in the eye or ear when the FSE is placed without accurate diagnosis of fetal position. An FSE provides a potential pathway for ascending infection. Early studies found a correlation between prolonged use of an FSE (more than 12 hours) and neonatal sepsis, but subsequent retrospective analyses determined that FSE alone is not associated with an increased risk for neonatal sepsis.[3-5] To date, none of the studies conducted have been sufficiently powered to definitely answer this question. When attachment of an FSE is done in a known vertex presentation and the procedure is performed under aseptic conditions, complications are very rare.

Mechanism of Action

The FSE spiral electrode contains two electrodes in close proximity. One penetrates the fetal scalp to the depth of approximately 2 mm, with vaginal secretions providing electrical conduction between the two wires; the difference in voltage between the two electrodes is then measured and recorded. A third reference electrode that helps eliminate electrical interference is placed on the maternal thigh.

Contraindications to Use of Fetal Scalp Electrode

- Active infection such as HIV, herpes, or hepatitis B or C
- Placenta previa
- Unknown fetal presentation or position
- Face presentation

Do not place on genitalia or a fontanel.

Equipment

- Sterile fetal spiral electrode
- Sterile gloves
- Electronic fetal monitor
- Leg plate with Velcro strap or adhesive patches and cable to fetal monitor
- Water-based lubricant

Procedure for Attaching a Fetal Scalp Electrode

1. Explain to the woman the indication for use of an FSE, and review the procedure, along with its benefits and risks. Answer any questions the woman or her support personnel have. Obtain an informed consent prior to placement.
2. Perform hand hygiene.
3. Attach the leg plate to the woman's thigh.
4. Prepare a site, often on the woman's bed, for the material needed.
5. Open the FSE packaging.
6. Put on sterile gloves.
7. Remove the wires from between the drive tube and the guide tube, keeping the materials sterile.
8. Perform a vaginal examination; confirm lie, presentation, and position. Keep the examining hand on the area of the presenting part where the FSE will be applied.
9. Select the FSE with the nonexamining hand. Verify that the spiral end of the electrode is withdrawn so it is completely within the guide tube. This assures that the device will not inadvertently injure any tissue of the woman or the examiner (**Figure 26B-1**).
10. Advance the guide tube between the examining fingers, and position it firmly against the fetal presenting part, avoiding sutures, fontanels, or genitalia. If uncertain about the exact presentation, do not attach the FSE.
11. Advance the drive tube until it touches the fetal presenting part.
12. Maintain pressure on the guide tube so that it remains touching the fetal presenting part, and rotate the drive tube clockwise approximately 1.5 rotations until mild resistance is felt and the drive tube recoils. Rotating causes the tip of the spiral electrode to penetrate the fetal scalp and be attached.[1]
13. Release the end of the electrode wires from the locking device at the end of the drive tube grip.
14. Carefully retract the guide tube a few inches and check the placement of the scalp electrode with the examining hand.
15. Remove the guide tube from the woman's vagina and dispose of it per institutional policy.
16. Remove and discard gloves.
17. Attach the electrode wires to the leg plate and the cable from the leg plate to the fetal monitor.
18. Evaluate the FSE recording of FHR for at least 5 to 10 minutes before leaving the room.
19. Explain the findings to the woman and her support team.

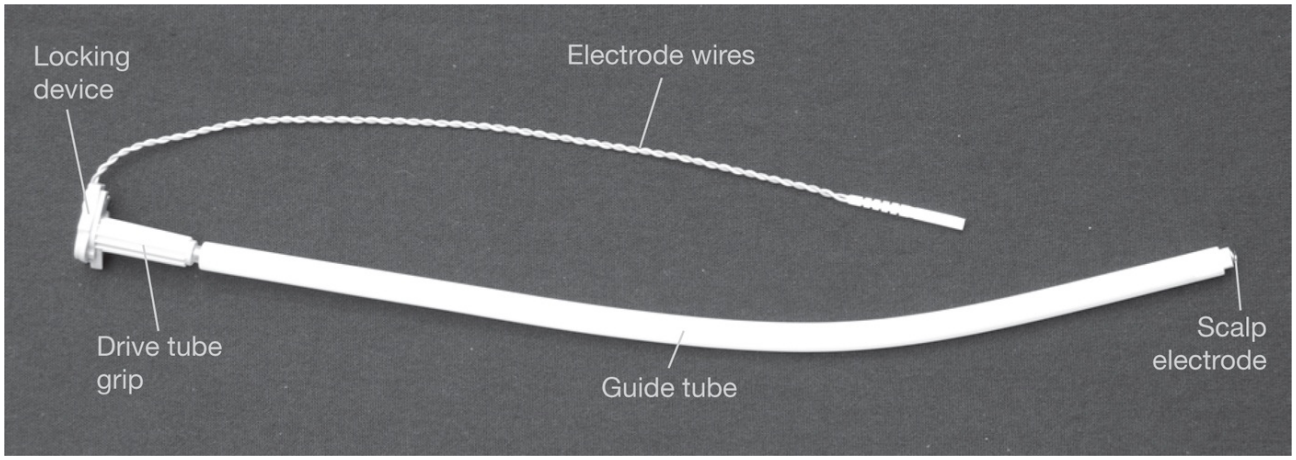


Figure 26B-1 Fetal monitoring spiral electrode assembly.

Procedure for Removing a Fetal Scalp Electrode

1. To detach the electrode, put on sterile gloves.
2. With the examining hand on the electrode, rotate it counterclockwise until it is free of the fetal presenting part.
3. Do not pull the electrode directly, as this could cause a scalp laceration.

References

1. Parer JT, King TL, Ikeda T. *Electronic Fetal Heart Rate Monitoring: The 5-Tier System*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2018.
2. Bernardes J, Ayres-de-Campos D. The persistent challenge of foetal heart rate monitoring. *Curr Opin Obstet Gynecol*. 2010;22(2):104-109.
3. Yancey MK, Duff P, Kubilis P, Clark P, Frentzen BH. Risk factors for neonatal sepsis. *Obstet Gynecol*. 1996;87(2):188-194.
4. Nakatsuka N, Jain V, Aziz K, Verity R, Kumar M. Is there an association between fetal scalp electrode application and early-onset neonatal sepsis in term and late preterm pregnancies? A case-control study. *J Obstet Gynaecol Can*. 2012;34(1):29-33.
5. Harper LM, Shanks AL, Tuuli MG, Roehl KA, Cahill AG. The risks and benefits of internal monitors in laboring patients. *Am J Obstet Gynecol*. 2013;209(1):38.e1-38.e6.

CHAPTER

27

Support for Women During Labor

ELIZABETH NUTTER

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Introduction

Every labor and birth is unique, and a woman's interpretation of labor pain is highly individual. The actions of care providers can have a strong effect in how a woman copes with labor pain and views her experience of labor.[1] A woman's memory of each labor and birth persists for many years, and likely for her lifetime.[2] This chapter divides coverage of support for women during labor into three sections: (1) a review of the physiology of labor pain and factors that influence the experience of labor and birth, (2) nonpharmacologic techniques for supporting women in labor, and (3) pharmacologic techniques used to support women during labor.

Physiology of Pain

Pain is the result of a complex interplay between physical, biological, psychological, and sociocultural factors, each of which contributes to the overall perception of the experience.[3,4] The formal definition of pain used in health care today comes from the International Association for the Study of Pain:

> *Pain is an unpleasant sensory and emotional experience. . . Pain is always subjective. Each individual learns the application of the word through experience related to injury early in life.*[5]

Pain Physiology: Receptors and Pathways

Pain is generally characterized as either acute or chronic, and is further characterized as somatic (deep or superficial) or visceral. Deep somatic pain is dull and aching but poorly localized, whereas superficial somatic pain is sharp and well localized. Visceral pain is caused by ischemia or inflammation in visceral structures (internal organs). Over the course of labor, visceral, deep somatic, and superficial somatic pain can all be experienced.

Pain is caused by stimulation of nociceptors—that is, sensory neurons that detect noxious substances. The pain impulse is transmitted from nociceptors in the periphery or viscera via fast, lightly myelinated A-delta fibers and slower, unmyelinated C fibers (**Figure 27-1**). Pain traveling along the A-delta fibers is perceived within 0.1 second, whereas pain transmitted along the C fibers takes longer—approximately 1 second or more to be perceived by the individual. Numerous types of stimuli, such as mechanical, thermal (temperature related), inflammatory mediators, and chemical agents, can stimulate nociceptors. For example, uterine stretching during labor causes mechanical pain, but lactic acid in uterine muscle can also stimulate uterine pain. Chemical mediators of pain include substance P, glutamate, bradykinin, acetylcholine, serotonin, and histamine. Pain management during labor is aimed at minimizing or allaying the effects of both mechanical and chemical mediators.[6]

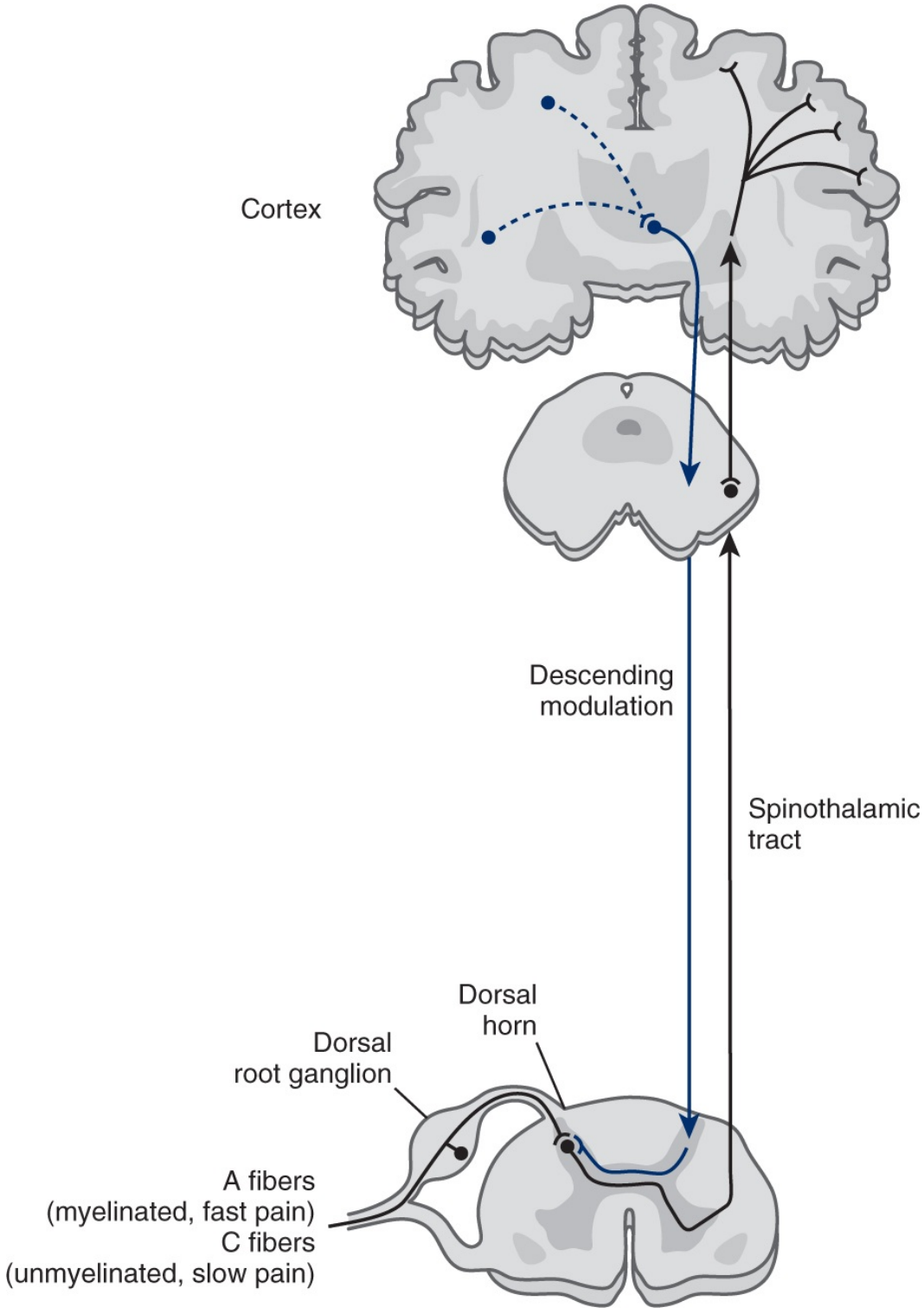


Figure 27-1 Transmission of pain periphery to the central nervous system.

Once pain is transmitted from the nociceptors at the anatomic source of the pain, the impulse is transmitted via A-delta and C fibers to the dorsal column of the spinal cord, where these fibers synapse with other neurons in the cord. During the first stage of labor, the pain fibers from the contracting uterus enter the spinal cord at the T10 to L1 levels, whereas during the second stage the pain fibers enter the spinal cord at the S2 to S4 level.[7] Nerves within the dorsal horn of the spinal cord transfer the impulse to the contralateral spinothalamic tract,

where nerves ascend to the cerebral cortex.

Prior to ascending to the central nervous system (CNS), the pain impulse crosses from one side of the dorsal horn to the other. The impulse can be modulated by several different mechanisms in the dorsal horn. The gate-control theory of pain modulation is the hypothesis that stimulation of A-delta fibers via massage, sterile water injections, or transcutaneous electric nerve stimulation (TENS) results in impulses rapidly traversing to the dorsal horn, where they effectively close a “gate” that blocks impulses from the slower C fibers that transmit deeper pain signals.[3,4] This theory, which was proposed in 1965 by Melzack and Walls, is not exactly correct in its details; notably, the mechanism for how the small and larger fibers interact is more complex.[8] Nonetheless, the gate-control theory of pain correctly identified the general process and set the stage for subsequent research that has greatly expanded knowledge of pain pathways and the factors that influence them.

The pain impulse travels to the brain via two pathways: the neospinothalamic tract and the paleospinothalamic tract. Most of the fibers of the neospinothalamic tract terminate in brain areas such as the brain stem and the thalamus, where the pain signal is transmitted to basal areas of the brain as well as the somatosensory cortex, causing the parturient to “feel” pain. The paleospinothalamic tract fibers terminate in the brain stem, in the areas of the medulla, pons, mesencephalon, and periaqueductal gray region. These lower regions of the CNS also play a role in the perception of pain. Thus, the perception of pain is the result of CNS integration of pain impulses in the thalamus, lower brain areas, and the cortex.

The pathways of pain perception are bidirectional. The CNS also has the capability of modifying the perception of pain. First, inhibitory neurons in the CNS can dampen the pain sensation via activation of an endogenous opiate system. The brain has a fairly elegant analgesic system that varies from person to person, which partially explains some of the individual differences in pain perception across the population. In addition, the periaqueductal gray and the periventricular areas (the areas around the third and fourth ventricles of the brain) possess neurons that transmit anti-nociceptive impulses to the dorsal horns of the spinal cord. The impulses from these fibers block the ascending pain impulse before it is relayed to the brain, via release of neurotransmitters such as enkephalin and serotonin at their terminus in the dorsal horn of the spinal cord. Serotonin promotes release of enkephalin, which is known to inhibit both presynaptic and postsynaptic type C and A-delta pain fibers at the dorsal horns. Several other naturally occurring physiologic opioids, such as beta-endorphin and dynorphin, also play roles similar to that of enkephalin. Most of the opioids now used exogenously to treat pain are derived from opium, a naturally occurring opioid whose structure resembles that of the endogenous opioids found in the brain and dorsal horn of the spinal cord.

Labor Pain

Labor pain is felt in three general physical locations. Virtually all women in labor experience lower abdominal pain, approximately 74% experience contraction-related back pain, and approximately 33% experience continuous low back pain.[9] In Melzack's classic study of 42 nulliparous women and 37 multiparous women who completed hourly pain assessments throughout labor, there was a wide amount of variability between individuals in the spatial distribution of where pain was felt.[10]

In addition to spatial variation, variation in intensity of labor pain is apparent. Generally, nulliparous women report higher average intensity levels of pain than multiparous women on the McGill Pain Questionnaire, especially nulliparous women who have had no preparation for childbirth.[10] However, there is variability in the pain reported at hourly intervals by individuals, just as there is a great deal of variability among different women. Although for most women, the average intensity of pain increases as labor progresses, nulliparous women often report more pain in early labor whereas multiparous women tend to report more pain in active labor. In addition, many women report periods during active labor where pain does not increase or when it actually decreases for a short time.

In summary, despite the general observation that labor pain increases as cervical dilation increases, labor pain can be independent of cervical dilation for both nulliparous and multiparous women, and there is interindividual variability in the location, intensity, and progression of pain during labor.[9-11]

Physiologic Response to Labor Pain

Pain during the first stage of labor is generally believed to be caused by mechanical distension of the lower uterine segment, cervical dilation, and perhaps acidemia in uterine muscles that develops as a result of recurrent uterine contractions.

A number of physiologic systems are affected by pain. One of the first responses is that of the sympathetic nervous system, which releases stress-related hormones such as cortisol, catecholamines (norepinephrine and epinephrine), and cytokines. Concomitantly, the levels of angiotensin II, antidiuretic hormone, growth hormone, and glucagon increase. The effect of these changes is heightened alertness, increased cardiac output, increased heart rate, increased respiratory rate, elevated blood pressure, higher serum glucose levels, and more extracellular fluid in the periphery and lungs. Increased levels of catecholamines also slow gastrointestinal and genitourinary motility and alter coagulation and immune responses.

The relationship between catecholamine release and uterine contractility during labor is poorly understood. It has long been theorized and noted in observational studies of small numbers of laboring women that the uterus does not contract well when catecholamine levels are very high.[12] Epinephrine is a known tocolytic and increases proportionately more than does norepinephrine during labor, although there is a great deal of interindividual variability. Poorly controlled pain can cause anxiety and fear, which in turn further stimulates the hypothalamic–pituitary axis and exacerbates the stress response, leading to the release of even more catecholamines and cytokines. Despite some known physiologic mechanisms, the exact effects

of stress and fear on uterine contractility during labor have not yet been clearly delineated.[12]

Factors That Affect Perception of Labor Pain

To help account for the significant interindividual variability in the perception and response to pain, Melzack expanded the gate-control theory so it evolved into the neuromatrix theory of pain.[3,4,13] According to this theory, the perception of pain by any individual, including the laboring woman, is a dynamic process with physical, psychological, and behavioral components.

The interplay of psychological preparation, expectations, past experience of pain, fear of childbirth, and emotional support during labor are just a few of the factors that affect the degree of pain perceived by a woman during labor. Expectations appear to play a large role in a woman's perception and ability to cope with the pain of labor.[13] In a review of this body of work, Lally et al. found that, in general, there is a significant gap between expectations and experience, with most women reporting more pain than expected and expectations that were not met.[14]

The effects of race, ethnicity, and culture on pain perception are complex.[6] Culture does not appear to influence perception of pain, but does affect behavioral responses to pain.[6] Genetic variability in opioid receptors can affect pain sensation, with specific polymorphisms in opioid receptors occurring more frequently in some racial/ethnic groups.[15,16] Unfortunately, interethnic differences in opioid receptors do not predict pain perception well enough to be of clinical value at this time.[16]

Genetic differences in the microsomal enzymes involved in metabolizing drugs can play a clinically important role in a woman's ability to metabolize the opioids used for pain management during labor.[17] For example, the cytochrome CYP2D6 has several polymorphisms that adversely affect the metabolism of codeine in individuals who have the polymorphism phenotype.[17]

Women's Experience of Labor

Multiple demographic characteristics of women are associated with increased severity of labor pain as well as the experience of labor. For example, women who have severe dysmenorrhea are more likely to have severe labor pain. This outcome may be related to increased prostaglandin synthesis, which results in intense uterine contractions.[6] Pain tends to be worse at night than in the daytime, and unfamiliar settings increase self-reports of pain intensity. Fear of labor and lack of self-efficacy are also associated with severe labor pain. A woman's ability to use mental strategies to cope with labor pain appears to be related to her self-confidence in being able to use those strategies, although this is probably a simplification that does not fully account for the many factors involved.[6] Finally, severe labor pain and a traumatic birth are associated with the development of chronic pain and post-traumatic stress syndrome.[18] Given these factors lead to different experiences of labor, the importance of providing individualized care and support for women during labor cannot be emphasized strongly enough.

Prenatal Factors That Influence Labor Pain

Knowledge of the physiology of labor pain and the ability to assess how a woman is coping with labor pain are important but not enough to ensure a midwife has the ability to support women sufficiently in labor. Multiple antecedents may affect labor pain, and knowledge of these factors can inform the therapeutic approach for an individual woman.

Women with Childbirth Fear or Anxiety

Fear of childbirth can range from mild anxiety to avoidance of childbearing because the fear is so great; the latter extreme is defined as *tokophobia*. Most of the research on fear of childbirth has been conducted in Northern Europe. Although there is a paucity of data on this topic from the United States, significant fear appears to be more common than previously thought.[19,20] Studies that have examined pregnant women's fear of childbirth have found increased fear is associated with nulliparity, young maternal age, preexisting psychological conditions, low-self-esteem, and a history of abuse.[19-21] Fear of childbirth is also associated with increased severity of labor pain.[21,22]

History of Sexual Abuse

In the United States, approximately one in every five women has a history of sexual abuse.[23] The experience of labor and birth can be traumatic for women who have a history of sexual abuse. Although most studies of this topic are small qualitative analyses of women's experiences, findings consistently show that care practices during labor can result in victimization and trauma.[23,24] A safe, trusting relationship with caregivers and trauma-informed support is an essential approach for caring for women who have a history of abuse.

Childbirth Education

Starting with the first edition of Grantly Dick-Read's classic *Childbirth Without Fear* published in the 1930s, various childbirth preparation programs have evolved to assist women in preparing to give birth. By the 1970s, childbirth education classes had become the *de rigueur* means of preparing for labor and birth. The initial focus of these classes was on reducing the fear–pain–tension cycle by providing women with knowledge about the labor process so pain medications could be avoided during labor. Dick-Read postulated that fear generates physical tension, which in turn increases pain; he also suggested that fear could be alleviated with knowledge and skills. Subsequently introduced natural psychoprophylactic methods of childbearing have varied in the emphasis they place on preparatory muscular and breathing exercises, but all educate the woman about the processes of labor and various means of coping with pain, thereby reducing fear.

A Cochrane review of the effects of childbirth preparation classes was not able to identify how childbirth classes affect labor pain, self-efficacy, or knowledge acquisition, with the researchers citing multiple methodological flaws in the trials that made up their systematic review of this topic.[25] A subsequent large randomized trial that involved intention-to-treat analysis found some significant benefits of childbirth education. In this study, women were randomly assigned to a group that received 9 hours of formal childbirth education versus a group that received usual care. The researchers found that women in the usual-care group were less likely to be admitted in active labor (relative risk [RR], 1.45; 95% confidence interval [CI], 1.26–1.65) and less likely to use epidural analgesia for labor (RR, 0.84; 95% CI, 0.73–0.97).[26]

Intrapartum Factors That Influence the Experience of Labor

In 2002, Hodnett performed a systematic review that analyzed results from 137 studies (both qualitative and quantitative) involving more than 14,000 women to determine the major factors that influence birth satisfaction.[13] This analysis identified four major components of birth satisfaction: (1) prenatal expectations, (2) support from caregivers, (3) quality of the caregiver–woman relationship, and (4) involvement in decision making. These results have been validated and replicated in multiple studies since the publication of this seminal analysis. Surprisingly, adequate pain relief is not one of the four factors that affect birth satisfaction. Hodnett concluded:

> *The influences of pain, pain relief, and intrapartum medical interventions on subsequent satisfaction are neither as obvious, as direct, nor as powerful as the influence of the attitude and behaviors of the caregivers.*[13(pS160)]

Coping During Labor

The work of Hodnett and others has greatly expanded our knowledge of women's experiences of labor, including clarifying the point that mitigation or diminution of pain and coping with labor are not the same thing. Research suggests that for the laboring woman, the ability to cope

is more important to birth satisfaction than relief of pain.[27] Yet, adequate pain relief is considered an important patient right by The Joint Commission (formally the Joint Commission on Accreditation of Hospitals). In most acute care settings in the United States, a numerical rating scale (where 0 is no pain and 10 is the worst possible pain) is used to assess pain, plan interventions, and evaluate the effect of those interventions. Such a numeric rating scale of pain does not, however, capture how well a woman in labor is coping with this experience.[27,28]

Fortunately, alternative algorithms have been developed to assess the more important variable of "coping" for the laboring woman. Gulliver et al. developed a Coping with Labor Algorithm that was pioneered at the University of Utah to capture the variable of coping for the laboring woman, while at the same time complying with The Joint Commission standards.[28,29] This algorithm is presented in **Figure 27-2**. Simply asking "How well are you coping on a scale of 1 to 10?" instead of "How bad is your pain on a scale of 1 to 10?" can generate the information that the midwife needs most to help a woman during labor.

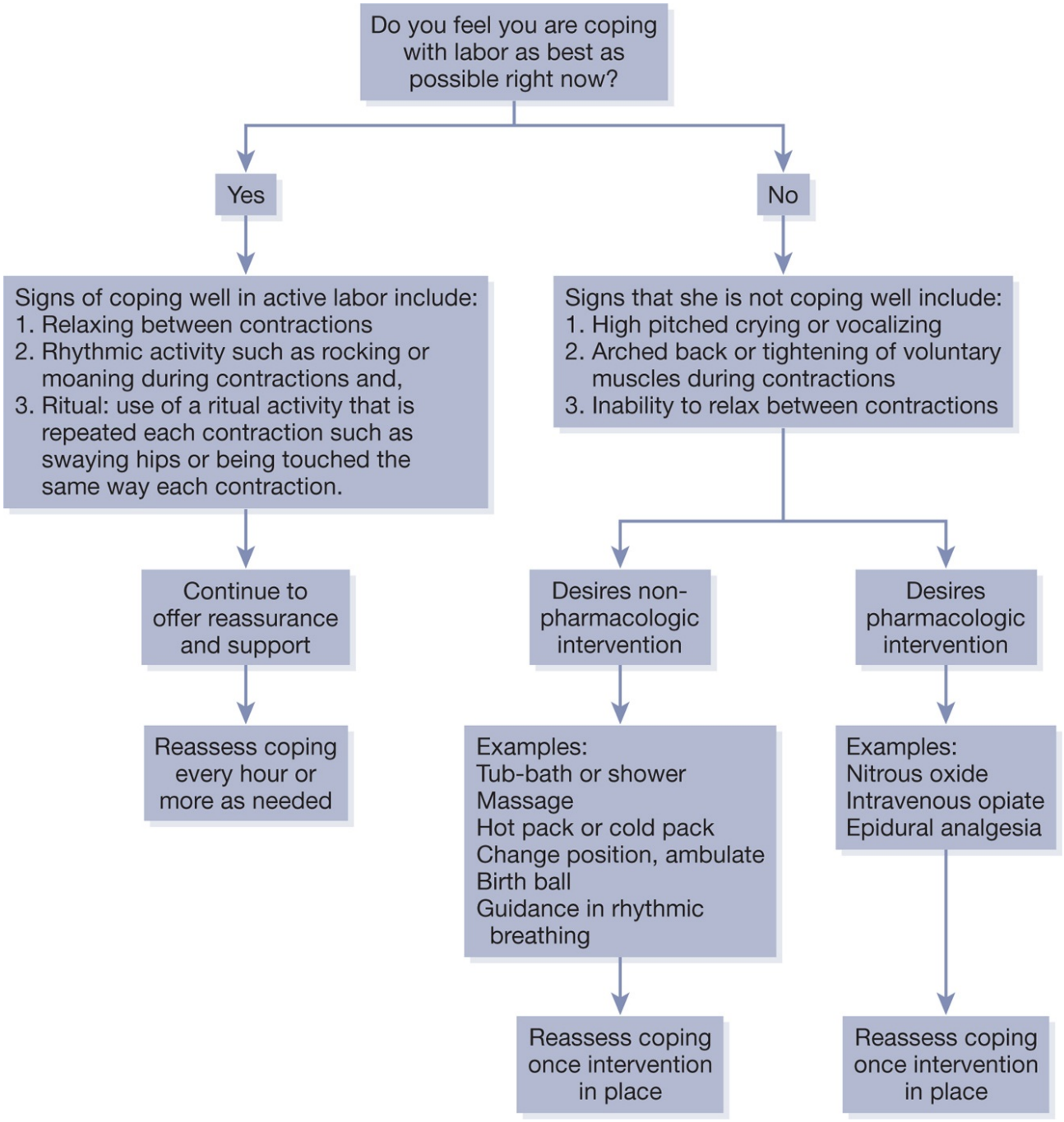


Figure 27-2 Coping with labor algorithm.

Modified with permission from Roberts L, Gulliver B, Fisher J, Cloyes K. The coping with labor algorithm: an alternate pain assessment tool for the laboring woman. *J Midwifery Womens Health*. 2010;55(2):107-116.[29] © 2010, with permission from Wiley.

Simkin analyzed the behaviors of women who were coping well in labor and found three consistent behaviors that serve as cues in addition to what the woman tells her caregivers: (1) relaxation during or between contractions, (2) use of a ritual (repeated use of a meaningful rhythmic activity), and (3) rhythmic activity (breathing, rocking, swaying).[30] If a woman is coping well with the pain of labor, then one can reliably presume she is not suffering or experiencing acute emotional distress. Simkin and Bolding stated:

> *Although pain and suffering often occur together, one may suffer without pain or have pain without suffering. . . one can have pain coexisting with satisfaction, enjoyment and empowerment. Loneliness, ignorance, unkind or insensitive treatment during labor, along with unresolved past psychological or physical distress, increase the chance that the woman will suffer.*[1(p489)]

Signs of not coping well or signs of suffering include crying, rapid shifts in position, and inability to focus. Performing a secondary analysis of data from the National Institutes of Health–funded Second Stage Labor Project, Bergstrom et al. reviewed videotapes of 23 women in the second stage of labor who eventually gave birth vaginally. The researchers observed four broad categories of behavior during the second stage: (1) no pain or distress, (2) low-level pain, (3) focused working, and (4) severe pain.[31] In all but the last category, women were able to maintain their sense of self-control. When pain was severe and women were not able to maintain a sense of self-control, an effective technique identified by the researchers was the "talking down" method. This method has three distinct steps: (1) calling out (getting the woman's attention), (2) positioning very close to the woman's face (en face position), and (3) talking in a low tone of voice so that the woman has to be quiet to hear. The researchers found that when the "talking down" method was used, women went back to a lower level of pain intensity that allowed them to maintain their sense of control.[31]

The Postpartum Effect of Traumatic Birth Experiences

Approximately 1% to 6% of women have symptoms consistent with the diagnosis of post-traumatic stress disorder (PTSD) in the immediate postpartum period following birth and approximately 2% continue to have symptoms of PTSD at 6 months postpartum.[32] More recently, a review of data from the Listening to Mothers II survey found the rate of PTSD after childbirth in women who gave birth in the United States to be approximately 9%.[33] Traumatic births are positively associated with PTSD, depression, and chronic pain. Several prepregnancy factors, such as preexisting anxiety disorders, are independent predictors of both traumatic birth and postpartum PTSD, which makes it difficult to determine the independent effect of labor experiences on these outcomes. Nonetheless, traumatic experiences during labor can have long-lasting negative effects. Consistent predictors of PTSD after birth include obstetric procedures, negative woman–staff interactions, and feelings of loss of control over the birth. This topic is reviewed in more detail in the *Mental Health Conditions* chapter.

General Principles for Assessment of Labor Pain and Provision of Labor Support

Care providers are not very accurate in assessment of a woman's discomfort or pain during labor. Studies that have compared provider assessments to women's assessments have found that the care provider's estimate of a woman's degree of pain is consistent with her estimate only approximately 50% of the time.[34] Approximately 25% of women have their pain overestimated by providers and 25% have their pain underestimated.[34] Multiparous women are more likely to have their pain underestimated by providers, and cultural differences between providers and women also increase the likelihood that pain will be underestimated.[35] In addition, one study found that midwives estimated women's pain fairly well when the woman reported mild or moderate pain but were likely to underestimate her pain when the woman reported experiencing severe pain.[36] Another study found that midwives' personal experiences influenced their estimation of a woman's degree of pain in labor.[37] Although these investigations entail small studies from different countries, they offer a cautionary tale. A woman's report of her experience is the most critical piece of data needed before offering any form of labor support.

The general support and comfort measures listed in **Table 27-1** summarize several general principles that form the foundation of supporting a woman during labor.

Table 27-1 General Support and Comfort Measures to Mitigate Pain During Labor

Basic Principles
Ensure privacy and prevent exposure
Use calm, quiet, purposeful communication
Physical Care Activities
Support relaxation to prevent fatigue
Help the woman maintain an empty bladder
Keep the perineum clean and dry
Offer oral liquids and lip moisteners for oral care
Change positions regularly and support extremities with pillows

Assurance of Privacy and Prevention of Exposure

Assurance of privacy and prevention of exposure are of particular importance in a hospital.[38] Ideas of modesty vary widely, often according to the woman's culture. Some women may not feel the need to be carefully draped to prevent exposure of external genitals, whereas other women will be uncomfortable exposing their body to anyone other than their partner and need as much draping as possible. Walburg et al. found that a limited number of personnel present at the time of exposure of external genitalia, a professional and kind attitude from providers, and

explanation of procedures may minimize feelings of embarrassment.[39]

Prevention of Exhaustion and Provision for Rest

Prevention of exhaustion and provision for rest between contractions are also important general support and comfort measures. Unnecessary exhaustion can be prevented by the following means:

- Have the woman use the most relaxed form of breathing possible. For example, panting is a physically taxing type of breathing, and should be used only when the goal is to help a woman not bear down. Controlled types of breathing, whether Lamaze or abdominal, can also be tiring if used in early, latent-phase labor before they are needed.
- Organize or plan procedures so that as many as possible are done sequentially in the shortest possible time (or number of intervals between contractions).
- Control the environment in accord with what is most restful to the individual woman. This includes controlling lighting, air, external noises, restriction of loud noises, and room arrangement.

Labor Pain Relief Methods Used in the United States

Laboring women can choose many methods of mitigating labor pain. **Table 27-2** presents findings from the second and third national Listening to Mothers surveys (LTM II and LTM III), which were conducted in 2006 and 2012, respectively.[33,40] These surveys interviewed women who gave birth in the previous year and provide a wealth of information about birth practices in the United States. Although epidural anesthesia is consistently rated as very helpful by women who use this technique to mitigate pain, other pharmacologic methods such as opioids are not rated as highly as some nonpharmacologic methods such as immersion in water.[33]

Table 27-2 **Methods of Pain Relief Used by Women in the United States**

Technique	Who Used This Technique 2012 (%)	Who Used This Technique 2006 (%)	It Was Helpful or Somewhat Helpful 2006 (%)	It Was Not Helpful 2006 (%)
Epidural or spinal analgesia	67	76	91	9
Breathing techniques	48	49	77	22
Position changes	40	42	77	24
Mental strategies	21	25	77	23
Opioids	16	22	75	25
Touch or massage	22	20	91	9
Birthing ball	10	7	64	33
Immersion in water	8	6	91	8
Application of hot or cold	12	6	81	17
Changes to environment such as music	2	4	78	22
Shower	10	4	78	19
Nitrous oxide	6	Not assessed		

Based on Declercq ER, Sakala C, Corry MP, Applebaum S. *Listening to Mothers II: Pregnancy and Birth*. New York, NY: Childbirth Connection; 2006. Available at: http://www.nationalpartnership.org/research-library/maternal-health/listening-to-mothers-ii-2006.pdf. Accessed February 20, 2017[33]; Declercq ER III, Sakala C, Corry MP, Applebaum S, Herrlich A. *Listening to Mothers III: Pregnancy and Birth: Report of the Third National U.S. Survey of Women's Childbearing Experiences.* New York, NY: Childbirth Connection; 2013. Available at: http://www.childbirthconnection.org/listeningtomothers. Accessed October 30, 2016.[40]

Nonpharmacologic Methods of Mitigating Pain

Some nonpharmacologic techniques for addressing labor pain are quite simple, whereas others require specialized training to perform. Techniques are reviewed here in the general order of the strength of evidence for their use, although in some cases comparisons are difficult due to different outcome measures, populations, and experimental designs used in the various studies. **Table 27-3** reviews the purported mechanisms of action for each of the following nonpharmacologic methods of labor pain relief.

Table 27-3 Mechanism of Action for Reducing Pain via Selected Nonpharmacologic Methods

Mechanism	Acupuncture/Acupressure	Aromatherapy	Breathing/Focus	Childbirth Education	Cold	Emotional Support	Heat	Hydrotherapy	Hypnosis	Intradermal Water Block	Massage/Touch	Movement and Positioning	Music/Audioanalgesia	Relaxation	TENS
Counter-irritation analgesia					✓					✓					✓
Increases endorphins	✓									✓			✓		✓
Provides stimuli from peripheral sensory receptors to inhibit pain awareness		✓			✓		✓	✓			✓		✓		✓
Increases joint mobility							✓	✓				✓			
Alters pressures within pelvis and on soft tissue								✓			✓	✓			
Improves energy flow along meridians crucial to labor progress and comfort	✓				✓*										
Decreases muscle tension	✓				✓		✓	✓	✓		✓			✓	
Alters nerve conduction velocity (slows pain transmission to central nervous system)					✓										
Decreases anxiety/fear, provides reassurance		✓	✓	✓		✓			✓		✓	✓	✓	✓	
Increases woman's sense of control, reducing pain perception			✓	✓		✓			✓			✓	✓	✓	✓
Distraction of attention from pain		✓	✓		✓	✓	✓	✓	✓		✓	✓	✓	✓	✓
Enhances or changes mood, reducing pain perception		✓				✓			✓		✓		✓	✓	
Cues rhythmic activity and rituals			✓			✓					✓	✓	✓	✓	

Reproduced with permission from Simkin P, Bolding A. Update on nonpharmacologic approaches to relieve labor pain and prevent suffering. *J Midwifery Womens Health.* 2004;49:489-505.[1]
© 2004, with permission from Wiley.

Continuous Labor Support/Doula Care

Multiple randomized controlled trials (RCTs) have shown that continuous labor support improves labor outcomes and has a positive influence on a woman's childbirth experience.[1,41,42] The word "continuous" refers to one support person who provides care to one woman and is physically present throughout labor. Doulas are trained nonmedical persons who provide continuous support for women in labor. The primary role of the doula is to provide continuous nonmedical care, including physical comfort, emotional support (reassurance and encouragement), information (nonmedical advice, anticipatory guidance), and facilitation of communication with healthcare providers. The word *doula* is derived from the Greek word *doule-*, meaning "one who serves." Doulas can provide many of the comfort measures during labor that are detailed in this chapter.

The effects of continuous labor support were evaluated in a systematic review of 27 RCTs that included 15,858 women from high- and middle-income countries.[41] This review revealed that continuous labor support during labor is associated with the following outcomes:

- Increased likelihood of spontaneous vaginal birth (relative risk [RR], 1.08; 95% confidence interval [CI], 1.04 to1.12)
- Lowered risk of cesarean births (RR, 0.75; 95% CI, 0.64 to 0.88)
- Shorter duration of labor (mean duration: –0.69 hour; 95% CI, –1.04 to –0.34)
- Reduced need for pharmacologic pain relief (RR, 0.90; 95% CI, 0.84 to 0.96)
- Less likely to have negative perceptions of childbirth experiences (RR, 0.69; 95% CI, 0.59 to 0.79).
- Reduced risk for low 5-minute Apgar scores (RR, 0.62; 95% CI, 0.46 to 0.85)

No significant differences were found in the use of synthetic oxytocin, newborn admission to a special care nursery, or breastfeeding at 1 to 2 months postpartum.[41]

The randomized trials found that continuous labor support is most effective when provided by doulas rather than by partners, other relatives, nurses, or midwives.[41-43] Nevertheless, smaller nonrandomized studies that employed midwifery students, nursing students, or trained relatives as doulas (following some training) have found positive birth outcomes as well.[44]

Hodnett[45] and Hofmeyr et al.[46] have described two theoretical explanations for the effectiveness of labor support. Both theories posit that labor support increases women's sense of being in control and self-efficacy. Hofmeyr et al. hypothesized that continuous companionship serves as a buffer to the stressors of labor, particularly in a hospital environment. Hodnett suggested labor support enhances fetal passage by promoting mobility and upright positions with improved comfort of the mother. Labor support may also reduce anxiety and fear, thereby decreasing the stress response.[46]

Intrapartum Hydrotherapy

Intrapartum hydrotherapy is the therapeutic use of water for pain relief and relaxation during childbirth. This practice may reduce a laboring woman's perception of pain by transmitting a therapeutic temperature into targeted tissues, changing the state of irritant receptors.[47,48] Intrapartum hydrotherapy can be provided in a shower, bathtub, pool, or any body of water available to a laboring mother.

Intrapartum immersion is the term used to signify hydrotherapy via submersion in a pool/tub that is deep enough for the water to cover the woman's abdomen. More specifically, intrapartum immersion can be divided into water labor and waterbirth. Water labor is defined as use of immersion during some portion of labor, but the actual birth does not take place while the woman is in the tub. Waterbirth is defined as birth of the neonate underwater.[47] Intrapartum immersion causes an increase in the woman's hydrostatic pressure and shift of extracellular fluid into her circulation, which provides a central blood volume bolus, increases cardiac output, and subsequently decreases circulating catecholamine levels. It is postulated that the improved blood flow results in improved uteroplacental perfusion and increased uterine

efficiency.[48,49]

The Cochrane Collaboration completed a systematic review of the effects of intrapartum immersion on labor and birth by examining 12 clinical trials involving more than 3400 women.[50] Water immersion during the first stage of labor was associated with a reduction in the use of epidural/spinal analgesia (RR, 0.90; 95% CI, 0.82–0.99) and a shorter first stage of labor (mean difference [MD]: –32.4 minutes; 95% CI, –58.67 minutes to –6.13 minutes) and no difference in the risk for chorioamnionitis (RR, 0.99; 95% CI, 0.50–1.96). There were no differences in the use of oxytocin, operative vaginal birth, or adverse neonatal outcomes.[50] In addition, intrapartum immersion has been associated with increased mobility, lower episiotomy rates, decreased likelihood of third- and fourth-degree perineal lacerations, and increased patient satisfaction, although the studies yielding these findings were small and the data not yet considered conclusive.[51]

Timing of intrapartum immersion warrants consideration. Immersion is recommended after active labor is established.[52] Immersion in water during latent labor may result in longer labors that can require pharmacologic intervention and oxytocin augmentation.[50,52] The American College of Nurse-Midwives has developed a model template for hydrotherapy in labor that describes eligibility for this therapy, techniques, and recommended policies.[52]

Acupressure/Acupuncture

Traditional Chinese medicine recognizes acupuncture as a means to correct imbalances in vital energy known as Qi (pronounced “chee”). Acupuncture involves the needling of specific points along meridian pathways, thereby promoting the circulation of blood and stimulating the flow of Qi to promote harmony of yin and yang. Acupuncture is thought to stimulate the body to produce endogenous opioids (endorphins), and to secrete neurotransmitters that maintain the normal physiology and provide comfort. Minimizing the stress response may be another mechanism of action underlying acupuncture’s efficacy.[53–55] Acupuncture should be performed only by a qualified practitioner, and most states require a diploma from the National Certification Commission for Acupuncture and Oriental Medicine for licensing to practice acupuncture.

An alternative to acupuncture is acupressure, whereby firm steady pressure is applied to acupoints with a thumb or finger instead of needles (**Figure 27-3**). Although no license is required to administer acupressure, training and education in these techniques are recommended.[55] Commonly cited acupoints utilized to help difficult or slowed labor progress and/or labor pain are Sanyinjiao/spleen 6 (SP6), Hoku or Hegu/large intestine 4 (LI4), bladder 67 (BL67), and gallbladder 21 (GB21).[53] Midwives with appropriate training, in combination with clinical judgment and practice, can incorporate acupressure into care for a laboring woman.

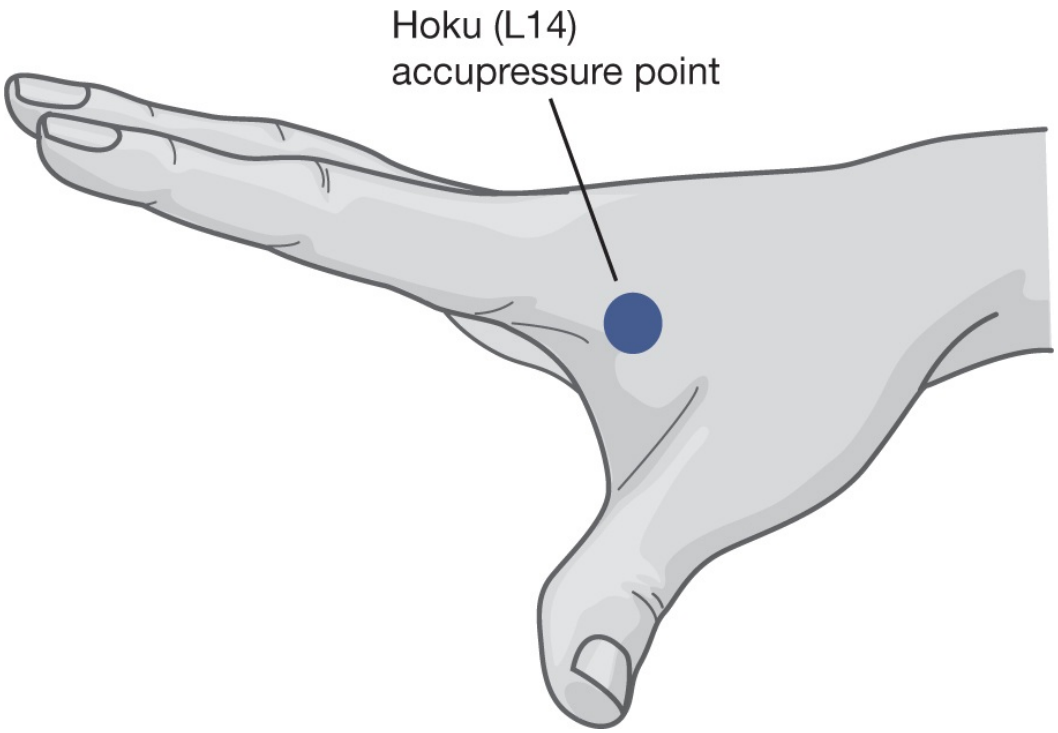


Figure 27-3 Correct positioning of small ice bag for massage stimulation of LI4.

Reproduced with permission from Waters BL, Raisler J. Ice massage for the reduction of labor pain. *J Midwifery Womens Health*. 2003;48:317-321.[55]

Although there is some heterogeneity in the trials of acupuncture or acupressure, results consistently show that these techniques reduce labor pain when compared to no intervention or placebo acupuncture/acupressure.[53-55] A theoretical risk of acupuncture is possible infection if sterile needles are not used, although licensed practitioners would be expected to use sterile needles, usually disposable.

Acupuncture/acupressure has also been evaluated for effectiveness in improving uterine contractility and labor induction. These studies have found a modest increase in cervical ripening (as measured by a Bishop's score) in women exposed to acupuncture, but have not adequately assessed other clinically important variables such as time to onset of labor. Nonetheless, contraindications to use of acupressure during labor may include fetal intolerance of labor, uterine tachysystole, tetanic contractions, preterm gestation, or any other clinical condition in which uterine contractions are contraindicated (e.g., placenta previa).[53]

The most current Cochrane meta-analysis of 14 RCTs of acupressure/acupuncture for pain relief in labor[54] identified the following results:

- Less intense pain with acupuncture compared with no intervention (standardized mean difference [SMD], –1.00; 95% CI, –1.33 to –0.67)
- Increased satisfaction with pain relief compared with placebo control (RR, 2.38; 95% CI, 1.78 to 3.19)
- Reduced use of pharmacologic analgesia when acupuncture is used compared with placebo, and compared with standard care (RR, 0.72; 95% CI, 0.58 to 0.88)
- Fewer operative vaginal births when acupuncture is used compared with standard care

(RR, 0.67; 95% CI, 0.46 to 0.98)
- Reduced pain intensity with acupressure compared with a placebo control (SMD, –0.55; 95% CI, –0.92 to –0.19)

Sterile Water Injections

Sterile water injections, or sterile water papules, involve intradermal injections of sterile water administered in four places in the lower lumbosacral area of the back to form papules. The therapeutic goal is to relieve back pain in labor. Since cutaneous afferent nerves from the lower back converge to the dorsal horns in the same segments, pain may be referred to the lower back. It is speculated that sterile water injections work by the gate-control theory, whereby stimulation of specific areas can relieve this referred pain. The mechanism has been described as counter-irritation, a process by which localized pain felt in one part of the body may be relieved by irritating the skin in an adjacent part of the body. Sterile water injections are thought to cause distension in the skin, which stimulates nociceptors and mechanoreceptors, thereby providing noxious stimulation of nociceptors. This effect results in a subsequent release of endorphins and stimulation of A-delta fibers, which overwhelm the visceral sensations transmitted to the brain by the slower C fibers.[56]

A 25-gauge needle containing 0.6 to 1.0 mL of sterile water, not saline, is used to inject 0.1 to 0.3 mL of sterile water intradermally at each of the four sites adjacent to the Michaelis rhomboid, where referred pain from uterine contractions is usually felt. Women experience an intense stinging sensation for approximately 30 to 90 seconds during (and immediately following) the injection and then relief from their back pain for approximately 45 minutes to 3 hours.[57] **Appendix 27A** reviews the procedure of sterile water papule injection.

Sterile water injections have been found to significantly reduce severe back pain. However, the overall need for additional pain medication is not reduced, presumably because the effect dissipates before the labor is concluded. When Derry et al. performed a meta-analysis of seven RCTs, they concluded that the evidence is insufficient to recommend this practice, due to perceived limitations of the studies involved.[56] In contrast, the meta-analysis of eight RCTs performed by Hutton et al. found sterile water papules were associated with a significant reduction in Visual Analog Scores (VAS) for pain in women and a decreased rate of cesarean births in the sterile water group (4.6%) versus the comparison groups (9.9%) (RR, 0.51; 95% CI, 0.30–0.87).[55]

Position Changes/Ambulation

When women are not confined to bed or acculturated to believe they should be in bed during labor, they frequently change positions throughout the intrapartum course, gradually limiting the number of position changes as labor progresses, and with the incidence of ambulation and sitting diminishing during active labor.[58] Upright positions are likely to improve maternal comfort during labor via several mechanisms. First, pelvic dimensions are wider in upright or squatting positions, and maternal movement is thought to facilitate optimal fetal positioning (**Figure 27-4**).[59,60] Second, optimal feto-pelvic relationships may decrease the pain associated

with malpositions.[1] Third, uterine contractility may be improved with upright positions, which could shorten the duration of labor.[59] Finally, upright positions may enhance a woman's sense of control and self-efficacy, which may make coping with labor easier.

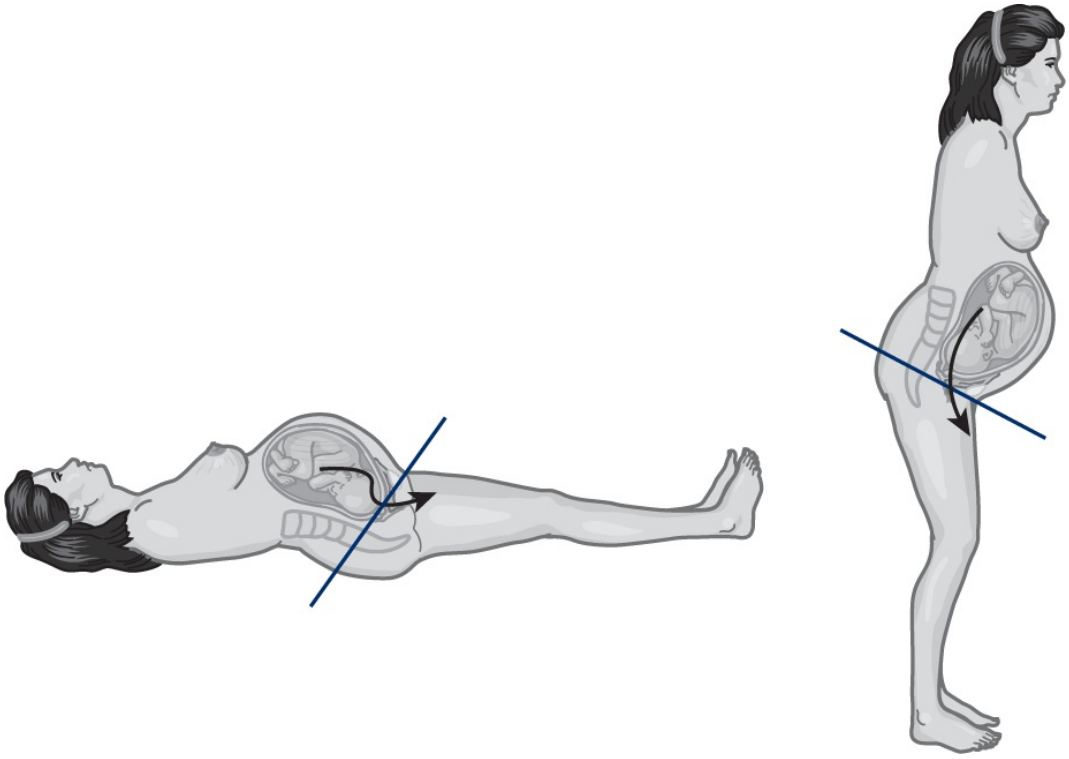


Figure 27-4 When a woman is supine or standing, the plane of the pelvic brim is not perpendicular to the spinal axis. Spinal flexion reduces the lumbar curve, providing the fetus with a straighter path through the birth canal. Note that the S-curve is simplified to a C-curve, allowing pressure on the fundus by the upper trunk.

Reproduced with permission from Fenwick L, Simkin P. Maternal positioning to prevent or alleviate dystocia in labor. *Clin Obstet Gynecol.* 1987;30(1):83-89.[59]

Atwood's 1976 article remains the key reference defining the different positions women may assume during labor or birth.[60] Atwood described maternal positions for labor or birth as either standing or neutral. The four standing positions are standing, sitting, kneeling, and squatting. Each of these positions places the maternal pelvis perpendicular to the spine and vertical plane of the uterus. Neutral positions include supine, lithotomy, lateral, prone, Sims, and semi-recumbent. In each of these positions, the woman's weight is primarily supported by her back, but by changing the position of her legs, she can alter the feto-pelvic relationship. The reader is referred to the articles listed in the Resources section at the end of this chapter for detailed descriptions of maternal positions.

Lawrence et al. conducted a Cochrane meta-analysis (n = 5218; 25 RCTs) of studies involving women in the first stage of labor who labored in an upright position as opposed to recumbent position. Upright positions were associated with a shorter labor by approximately 1 hour and 22 minutes (average MD, –1.36; 95% CI, –2.22 to –0.51).[61] Women who were upright were more likely to experience a vaginal birth, and were less likely to have a cesarean birth, epidural analgesia, or admission of their neonate to the neonatal intensive care unit

(NICU). There were no significant differences between groups in the duration of the second stage of labor, nor in other outcomes related to the well-being of women and newborns between the groups who were upright versus those who were recumbent during the first stage of labor.[61]

A Cochrane meta-analysis of studies involving women who did not have epidural analgesia and who assumed upright positions in the second stage of labor (n = 9015; 32 RCTs) revealed fewer operative vaginal deliveries, fewer abnormal fetal heart rate patterns, fewer episiotomies but no change in cesarean section rates, and an increased risk of an estimated blood loss of more than 500 mL.[62] The difference in estimated blood loss may be related to differences in estimating rather than differences in actual blood lost. By comparison, another Cochrane meta-analysis assessed studies involving women who utilized epidural analgesia in the second stage of labor (n = 879; 5 RCTs) and revealed no statistically significant difference between upright and recumbent positions on mode of birth, duration of the second stage of labor, trauma to the birth canal requiring suturing, operative birth for fetal distress, low cord pH, or admission to a NICU.[63] Although this body of work is limited by heterogeneity in study designs and small numbers of participants, the findings suggest that upright positions may benefit women who do not receive an epidural, but that preserving the perineum may be technically more difficult at the time of birth.

Contraindications to ambulation and movement are rare but may include ruptured membranes with high fetal station with the presenting part not well applied to the cervix, or any other condition for which bed rest is necessary.

Birthing Balls

A physiotherapy ball is a 65-centimeter ball that can be adjusted for individual “fit” by adding or letting out air so that the ball is firm but gives to the touch, and rolls easily, with the woman’s legs being bent at a 90-degree angle when sitting on the ball. Women sit on the birthing ball with their feet approximately 2 feet apart and flat on the floor. This puts the woman in an upright position from which she can rotate her hips in ways that relieve back pain and encourage fetal descent. A woman can also sit on the ball and lean forward, or can use the birthing ball to lean against either in a kneeling (ball on floor) or standing (ball on a bed or table) position (**Figure 27-5**). These positions support the woman’s body and provide a position in which she can rest. They also align the long axis of the uterus and the fetus with the mother’s pelvis and facilitate occiput anterior positions. The hypothesized mechanism of action with regard to mitigating pain is similar to the purported mechanisms for upright positions.



Figure 27-5 Positions for laboring women using a birthing ball.

Research on the effects of birthing balls on pain is limited to a few RCTs with a small number of subjects. A meta-analysis (n = 220; 4 RCTs) found that birthing balls provided statistically significant improvements in labor pain when compared to women who did not use such a ball during labor (pooled MD,–0.92; 95% CI, –1.25 to –0.56; p > 0.0000005).[64]

Another type of birthing ball is known as the "peanut ball." As the name implies, it is shaped like a peanut shell, with the middle circumference being smaller than the circumference at the ends. The peanut ball can be placed between a woman's legs or under her legs while in the bed. This positioning widens the diameter of the pelvic outlet, thereby allowing more room for fetal descent or repositioning of the fetus.

One RCT evaluated the impact of the peanut ball on duration of the first and second stages of labor in women who were scheduled for elective induction of labor at 39 weeks' gestation or more and who were planning epidural pain management. The researchers found that use of a peanut ball reduced the duration of the first stage of labor for primiparous women (P = 0.018), but did not lead to any significant difference (reduction) in the length of first-stage labor for multiparous women (P = 0.057). Because the authors did not report actual differences in time, it is not clear how clinically significant these findings are. Use of the peanut ball did not alter the duration of second-stage labor in either group in women undergoing induction of labor.[65]

A second RCT evaluated use of the peanut ball in women who received epidural analgesia, compared to standard care with epidural analgesia and no peanut ball. The researchers found that the peanut ball decreased the duration of labor (first stage, 29 minutes; P =.053 and second stage, 11 minutes; P <.001), and decreased the incidence of cesarean deliveries (10.3% versus 21.1%, respectively, P =.011).[66]

Although these studies suggest that both the birthing ball and the peanut ball have positive impacts on labor outcomes, more research is needed to determine the evidence of effectiveness of these techniques.

Touch and Massage

Massage, which refers to purposeful manipulation of soft tissues for therapeutic purposes, is

widely utilized during labor. Massage is applied to a specific body part, such as the hands, feet, scalp, shoulders, or back. Touch can be firm or light and repeated in a specific pattern (e.g., effleurage), or it can consist of still pressure or kneading. A specific form of touch often used to aid women with back pain is firm, steady counterpressure applied to the center of the sacroiliac portion of the back during a contraction. With counterpressure, the amount of pressure increases during the contraction and decreases as the contraction wanes. Massage can be performed with use of the hands or with massage devices, and with or without lotions, oils, and powders.[67]

Touching and massage will be effective only if the woman is comfortable being touched, as cultural views of touch appropriateness vary. This technique's probable mechanism of action in reducing pain is an increase in the endogenous release of endorphins and oxytocin. A meta-analysis (n = 225, 6 RCTs) evaluating massage and touch demonstrated a statistically significant reduction in pain intensity during the first stage of labor (SMD, –0.82; 95% CI: –1.17 to –0.47).[68] A subsequent trial that compared a specific form of massage/counterpressure over the sacrum and lower back, applied for 30 minutes at three different times during labor, found a significant decrease in reported lower back pain and increased satisfaction with the birth experience among the women in the massage group.[69]

While these studies demonstrate that massage may have a positive impact on laboring women, research into massage or touch has examined many different methods. Thus, it is not clear which massage or touch techniques are the most helpful.

Hypnosis

Hypnosis employs specific mind–body techniques that are used to induce profound relaxation. The goal of using hypnosis during labor is to help the woman achieve a state of deep relaxation within a state of focused attention, thereby reducing perception of external stimuli and increasing receptivity to specific suggestions by the hypnotist. The ability to experience hypnosis varies, but hypnotic susceptibility has generally been found to increase during pregnancy.[70] Hypnosis can involve a hypnotist or can be self-administered as self-hypnosis, whereby women can be trained to guide themselves through a hypnotic state.

Studies evaluating hypnosis during childbirth have yielded conflicting findings to date. Women who practice self-hypnotic techniques during the prenatal period may reduce their need for analgesia during labor and may have shorter labors when compared to women who use other childbirth preparation techniques.[71-73] The beneficial effects of introducing hypnosis for the first time to women in labor, however, have not been confirmed.[73] A Cochrane meta-analysis (n = 2954; 9 RCTs) reported no difference between women in the hypnosis group and those in the control groups in regard to sense of coping with labor, mode of birth, or satisfaction with pain relief.[72] Further research is needed to assess the value of hypnosis as a pain management technique during labor and birth. Notably, this technique is contraindicated for women with a history of psychosis or severe mental health condition.

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) units are handheld, battery-operated devices that use low-voltage electric current to reduce pain in labor. TENS has been used in childbirth to help reduce labor pain since the 1970s. With this technique, stimulation electrode pads are placed on the skin over the low back on the paraspinal muscles, on either side of the spine, at the vertebral positions of T10 and S2, which correspond to the nerve pathways through which the pain signal from the uterus enters the spinal cord. With the pads in place, the TENS unit emits low-voltage impulses. The frequency and intensity of the nerve stimulation can be controlled by the woman. The low-voltage sensation is not painful, but rather is often experienced as a tingling or buzzing feeling at the site of the electrodes.[74]

The mechanism of action of TENS is based on the gate-control theory of pain, as well as a theory that painful stimulation results in the release of neurotransmitters. TENS units are believed to induce the release of endorphins. Endorphins' primary function is to inhibit the transmission of pain signs, similar to the effect produced by opioids.

The effectiveness of TENS for treating labor pain remains unclear. A Cochrane meta-analysis (n = 1466; 17 trials) of women who used TENS units in labor was unable to find consistent evidence that TENS units have any impact on interventions or labor outcomes.[74]

Application of Heat and Cold

Heat and cold therapies are simple, safe, and inexpensive nonpharmacologic pain relief methods. Application of cold for minimizing pain is based on gate-control theory; cold therapy effectively blocks neural transmission via sensory fibers, thereby elevating the pain threshold. Cold therapy can also lead to a reduction in pain by inducing numbness, decreasing muscle tension, and offering distraction. Heat therapy, in addition to the same mechanisms of action related to application of cold, stimulates heat receptors in the derma and deeper tissues, which purportedly leads to closure of the gate, thereby subsequently impeding neural impulses from reaching the brain. Topical application of heat increases circulation to the area, thereby combating pain from tissue anoxia.

Most studies of heat or cold therapy published to date have been small and hindered by differing methodologies. In general, though, the studies published have found some benefits to heat or cold therapy with regard to decreasing labor pain.[75,76]

Caution is warranted when using heat or cold packs, as burns can be caused by using too high a temperature or by putting heat on an area to which creams, oils, or ointments have already been applied. Using a microwave to heat hot packs can be particularly dangerous, as microwaves heat elements at different temperatures; thus, the hot pack can be differentially heated in various places within the pack. It is recommended that at least one layer of cloth be placed between the woman's skin and hot or cold pack, and that the pack be tested on an arm or other portion of skin by someone other than the laboring woman before being placed against her skin. Use of heat or cold should also be avoided in the dermatomes anesthetized by an epidural due to concerns that women may not be able to perceive if the temperature is too hot or cold.

Fever is a contraindication to using heat. Contraindications to use of cold include sickle cell anemia, cold hypersensitivities, cryoglobulinemia, Raynaud's phenomenon, and cultural

proscriptions.[1]

Breathing and Relaxation Exercises

Breathing and relaxation exercises can help a woman in labor by decreasing her anxiety and fear, and by increasing her sense of control or self-efficacy.[1] Use of these techniques is directly related to childbirth satisfaction, and they are essential components of most childbirth education programs.[1] Approximately 48% of women in the United States use breathing and relaxation methods during labor, and 77% of those women report that these techniques were either somewhat helpful or very helpful.[33] Practice may play a large role in how well these techniques work. Specific breathing techniques work best in early labor but do not help women cope with labor if introduced for the first time during active labor.

Breathing techniques that focus on progressive relaxation may be more effective than those that focus on specific types of breathing patterns. Progressive relaxation can be practiced prenatally, so that the woman can quickly relax between contractions during labor. A knowledgeable caregiver can also teach some relaxation techniques during labor.

Audioanalgesia

Audioanalgesia was originally defined as a technique whereby auditory stimulation (e.g., music, white noise, and/or environmental sounds) is provided to create distraction and decrease perception of pain by activating regions of the brain that are responsible for mediating reward and anxiolytic effects. These areas of the central nervous system overlap with regions of the brain involved with analgesia.[77]

Audioanalgesia is common in dental practice, but research on its effectiveness during labor has yielded mixed results. Some small studies have found audioanalgesia may be beneficial during latent and active labor and may positively affect pain perception via elevation of mood and an increase in pain tolerance.[78,79] However, Cochrane systematic reviews of these studies have found insufficient evidence to support the effectiveness of audioanalgesia for labor pain management.[1,80] The wide variation in personal choices may account for the lack of sufficient evidence to make recommendations for the use of music for labor pain management.

Aromatherapy

Aromatherapy refers to the medicinal or therapeutic use of essential oils absorbed through the skin or olfactory system. Essential oils are derived from plants and are utilized to enhance physical and psychological well-being. Clinical aromatherapy is the controlled use of pure essential oils to achieve a desired measurable outcome based on the chemical constituents of an oil. Every essential oil comprises a variety of chemical constituents (e.g., esters, alcohols, phenols, aldehydes, ketones, terpenes, sesquiterpenes) that may have therapeutic effects.

Research into the effectiveness of aromatherapy in reducing pain during childbirth has yielded inconsistent results. A Cochrane review of two RCTs (n = 535 women) found no difference between groups in terms of pain intensity, assisted vaginal birth, cesarean section,

use of pharmacologic pain relief, spontaneous vaginal birth, duration of labor, or augmentation in women using clary sage, chamomile, lavender, ginger oil, or lemongrass essential oils compared to women receiving standard care.[81] In contrast, a combined systematic review and meta-analysis identified a significant positive effect of aromatherapy (compared to placebo or usual treatment controls) in reducing pain reported on a visual analogue scale. This study found that aromatherapy was effective in treating nociceptive, acute, postoperative, and obstetric and gynecologic pain ($P < 0.0001$).[82]

Essential oils are generally considered safe for use in childbirth. Midwives wanting to integrate essential oils into their clinical practice should seek additional training on the clinical application of essential oils during labor and birth prior to their utilization in practice. Midwives using essential oils in clinical practice need to understand relevant essential oil physiology, chemistry, and pharmacology, and must know how to blend and administer the oils safely and appropriately.

Pharmacologic Methods of Mitigating Labor Pain

Women can be knowledgeable and prepared; want active participation in the natural, normal process of childbearing; and yet still desire to use medication to mitigate labor pain. Medications may be used during labor to relieve pain, decrease anxiety and apprehension, provide sedation, or control nausea and vomiting. All medications used by laboring women should be evaluated for (1) effectiveness, (2) safety for the laboring woman, (3) safety for the fetus and newborn, and (4) effects on labor progress. Prior to administering any medication during labor, a careful assessment of the woman's vital signs, fetal heart rate pattern, other medications being used, and stage of labor is made and documented in the health record. The choice of medication will be tailored to the degree of pain relief the woman wants to attain and what is safe for her stage of labor.

Two types of pain relief drugs exist: analgesics and anesthetics. Analgesia is the relief of pain without total loss of feeling or muscle movement. Analgesics do not always stop pain completely, but rather tend to lessen pain. Anesthesia refers to blockage of all sensation, including pain. In most cases, analgesia is offered to women in labor or after surgery or birth, whereas anesthesia is used during cesarean birth. Pharmacologic management can also be classified as either systemic or neuraxial (e.g., epidural, spinal). Drugs placed in the epidural space (neuraxial) can induce *epidural analgesia* (reduces but does not obliterate all sensation) or *epidural anesthesia* (all sensation and motor ability is inhibited). Epidural analgesia is used to treat labor pain and epidural anesthesia is used for surgery when all sensation needs to be blocked.

Systemic Analgesic Medications

Systemic medications were the mainstay agents used to assist laboring women for most of the first half of the twentieth century. These medications are utilized by women who prefer less physically invasive pain management techniques; they can also be administered to women for whom regional neuraxial techniques are contraindicated or unavailable. Systemic medications are given by intravenous, intramuscular, or inhalation routes. The most popular of these agents are opioids, mixed opioid agonist–antagonists, sedatives, and hypnotics. Systemic analgesics act on the entire nervous system, rather than a specified area. The primary drawback of these drugs is that they adversely affect the fetus to varying degrees.

Sedatives and Hypnotics

Various sedatives and hypnotic agents are used during childbirth to induce sedation, decrease anxiety, and decrease pain. Non-opioid agents commonly utilized in labor are listed in **Table 27-4**. These drugs are usually administered in combination with opioids to decrease nausea and vomiting, both of which are often side effects of opioids. In clinical practice, they are typically used to induce sedation and stop contractions for women who are experiencing a prolonged latent phase of labor.

Table 27-4 **Sedatives and Hypnotics for Management of Pain During Latent Labor**

Generic (Brand)	Drug Class	Dose/Route	Onset	Duration	Comments
Promethazine (Phenergan)	Phenothiazine	25–75 mg IV/IM	10–20 minutes	3–4 hours	Commonly used with opioids to mitigate nausea and vomiting; induces sedation; may produce hypotension
Hydroxyzine (Vistaril)	Antihistamine	25–50 mg IM	30 minutes	4 hours	Commonly used with opioids to mitigate nausea and vomiting; induces sedation; not to be used intravenously; painful for IM injection
Diphenhydramine (Benadryl)	Anticholinergic antiemetic	10–50 mg IV	5–10 minutes	6–8 hours	Commonly used with opioids to mitigate nausea and vomiting; induces sedation; maximum daily dose 400 mg
Prochlorperazine (Compazine)	Dopamine receptor antagonist	5–10 mg IV/IM	5–10 minutes	3–4 hours	Commonly used with opioids to mitigate nausea and vomiting; induces sedation

Abbreviations: IM, intramuscular; IV, intravenous.

Sedatives such as promethazine (Phenergan) and hydroxyzine (Vistaril) do not induce sufficient analgesia when used alone, but are often used in combination with opioids in an attempt to potentiate the opioid effect.[83] Controversy exists regarding the effect of sedatives on labor pain. Promethazine, for example, can produce significant sedation, which results in the woman appearing as though she is in less pain but may actually just prevent her from communicating her level of pain. Thus, although anecdotally the combination of an opiate with a sedative results in pain relief potentiation, a potentiation effect has not been definitely proven.

Opioids

Women desiring pain relief during labor may be offered opioids when neuraxial analgesia is contraindicated or unavailable. The opioids most commonly used to treat labor pain in the United States are listed in **Table 27-5**.[83] These drugs are readily available, easy to administer, fast acting, and less invasive than neuraxial analgesia.

Table 27-5 **Pharmacokinetic Profiles of Selected Opioids**

Drug (Brand)	Usual Dose	Onset/Peak Effect	Duration of Action/Elimination Half-Life	Clinical Implications
Butorphanol (Stadol)	IV: 1–2 mg every 3–4 hr IM: 1–2 mg every 3–4 hr	IV: 2–3/5–10 min IM: 10–20/30–60 min	IV or IM: 4–6 hr Half-life: 2.5–3.5 hr in adults	Kappa agonist and sigma agonist; may precipitate acute withdrawal syndrome in an opiate-dependent mother and baby Maternal ceiling effect on respiratory depression and analgesia Fetal transient pseudo-sinusoidal FHR pattern
Fentanyl (Sublimaze)	IV: 50–100 mcg/hr IM: 50–100 mcg/hr	IV: 1 min/5 min IM: 7–15 min/ 10–20 min PCA: 10–25 mg with a lockout of 3–12 min and a set maximum 1-hr dose	IV: 30–60 min IM: 1–2 hr Half-life: Adults: 3–4 hr Neonates: 75–440 min	Pure agonist at mu receptors,[a] kappa receptors,[b] sigma receptors,[c] and delta receptors[d] Short-acting; less effective than morphine or Demerol, but very few side effects noted With higher doses or prolonged infusions, fentanyl becomes longer acting Transient decreased FHR variability or pseudo-sinusoidal pattern Maximum cumulative labor dose is usually 500–600 mcg when given IV or IM
Meperidine or pethidine (Demerol)	IV: 25–50 mg every 1–2 hr IM: 50–100 mg every 2–4 hr	IV: 5 min/5 min IM: 10–20 min/ 30–60 min	IV: 2–4 hr IM: 2–5 hr Half-life: Maternal: 3–7 hr Neonates: 18–23 hr	Fetal exposure is highest 1–4 hr after maternal administration with associated neonatal respiratory depression; highest 2–3 hr after administration May temporarily decrease FHR variability Normeperidine is an active metabolite of meperidine that has a half-life of 21 hours in the woman and 63 hours in the neonate; normeperidine accumulates in maternal plasma after multiple doses and adversely affects newborn neuroadaptive scores and breastfeeding behaviors

Morphine	IV: 2–5 mg every 4 hr IM:10 mg every 4 hr	IV: 5 min/0.5–1 min IM: 10–20 min/20 min	IV: 1–3 hr IM: 3–5 hr Half-life: Maternal: 43 min Neonates: 6.5 ± 2.8 hr	Pure agonist at mu[a] and kappa[b] receptors Faster absorption when administered in deltoid muscle compared to gluteal muscle Use lower doses for women with impaired ventilation or asthma Side effects include pruritus, nausea and vomiting, and urinary retention; has an active metabolite that can cause respiratory depression as the dose accumulates[e] The active metabolite of morphine is morphine-6-glucuronide; this metabolite has a half life of 2–4 hours in the woman and 13.9 hours in the neonate, and produces greater analgesic effect than morphine; can accumulate with repeated doses
Nalbuphine (Nubain)	IV or IM: 10 mg every 3 hr	IV: 2–3 min/30 min IM: <15 min/30–60 min	IV: 2–4 hr IM: 4–6 hr Half-life: 2–5 hr in adults	Mu antagonist and kappa partial agonist Maternal ceiling effect on respiratory depression and analgesia at 30 mg May precipitate acute withdrawal syndrome in an opiate-dependent mother and baby
Remifentanil (Ultiva)	PCA administration with varying dosing	1–1.5 min/2 min	20 min Half-life: 9 min in adults	Potent maternal respiratory depressant; this agent's use in clinical practice is controversial Dose at beginning of uterine contraction to maximize peak effect during contraction

Abbreviations: FHR, fetal heart rate; IM, intramuscular; IV, intravenous; PCA, patient-controlled analgesia.

[a] Mu-receptor stimulation causes slowed gastrointestinal motility, nausea and vomiting, respiratory depression, spinal and supraspinal analgesia, sedation, urinary retention, pruritus, and euphoria.
[b] Kappa-receptor stimulation causes sedation, spinal analgesia, and miosis.
[c] Sigma-receptor stimulation causes dysphoria, hallucination, and mydriasis.
[d] Delta-receptor stimulation causes spinal and supraspinal analgesia.
[e] Morphine causes more pruritus and urinary retention when administered via epidural or intrathecally. Maternal respiratory depression can be a late effect in epidurally administered morphine sulfate secondary to slow uptake into the central nervous system.

Based on Anderson D. A review of systemic opioids commonly used for labor pain relief [erratum, *J Midwifery Womens Health*. 2011;56(4):411-418]. *J Midwifery Womens Health*. 2011;56(3):222-239.[83]

Opioids can be administered by the intramuscular, intravenous, or subcutaneous routes, or via a patient-controlled analgesia (PCA) pump. The intramuscular and subcutaneous routes involve potentially painful injections, delayed onset of action, and variable absorption, which can lead to unpredictable plasma levels. Intravenous injections have a faster onset, with less variability of peak concentrations. PCA pumps allow women to self-administer a programmed dose of the opioid intravenously with minimum intervals between doses. PCA pumps provide rapid onset of action, better control of pain relative to side effects compared to parenteral

opioid injection, and a sense of control for the woman.[84] In general, PCA administration of fentanyl in labor may provide the best combination of effective pain relief with low risk of side effects or neonatal effects. A fentanyl PCA regimen of 10–25 mcg every 5–10 minutes has been found to provide effective analgesia with fewer side effects than morphine, meperidine, or alfentanil PCA regimens.[84-87]

Historically, meperidine (Demerol) was the most frequently used opioid for treating labor pain worldwide; now, however, it has largely fallen out of favor in the United States. Meperidine does not provide strong pain relief and when women who have used it are asked, more than half report that it was ineffective.[88] Meperidine has a long half-life and active metabolites, so its use is associated with a high risk of neonatal respiratory depression.

Morphine and fentanyl (Sublimaze) are the two mu-opioid receptor agonists most frequently used for labor pain in the United States. Morphine is most often used to treat prolonged latent labor, as it decreases uterine activity prior to active labor but does not inhibit active labor contractions.[89] A common combination for treating prolonged latent-phase labor is 10 mg of morphine sulfate and 50 mg of promethazine (Phenergan) given intramuscularly. Because morphine has active metabolites that can cause maternal or newborn depression, it is generally contraindicated for use in the later stages of labor and is replaced by fentanyl if an opioid is considered for a woman in active labor.

Fentanyl has a short half-life, so it has not been associated with neonatal respiratory depression. Due to its short duration of action it is well suited for administration toward the end of the first stage of labor or for intravenous PCA administration when neuraxial analgesia is contraindicated or unavailable.

Remifentanil is the shortest-acting opioid available. It is used only in PCA infusions, and primarily for women who cannot use neuraxial analgesia for some reason. Remifentanil does not have active metabolites, and is associated with fewer side effects or neonatal effects when compared to other opioids. Because it can cause significant maternal respiratory depression, its use in laboring women remains controversial, and research on this indication continues.[90]

Butorphanol (Stadol) and nalbuphine (Nubain) are kappa-receptor agonists and mu-receptor antagonists. They have a longer half-life and, anecdotally, have been reported to provide better pain relief than fentanyl. Both are contraindicated for use by women who have a history of drug abuse or addiction because the mu-receptor antagonist effect can elicit withdrawal symptoms.

One of the first systematic reviews of parenteral opioids found that this therapy provides limited pain relief and leads to moderate satisfaction with analgesia, with two-thirds of women reporting moderate or severe pain and/or poor or moderate pain relief one to two hours after parenteral opioid administration.[88] Furthermore, comparative effectiveness studies have not found one opioid to be better than another in mitigating labor pain. Research has also demonstrated there is no apparent effect on labor progress if an opioid is administered during the active phase of labor. Some women feel an unwelcome loss of control after being given an opiate, although this experience is not common. The more common side effects of such therapy include euphoria and sedation. Potential side effects include nausea and vomiting and urinary retention. Adverse effects include respiratory depression, but this outcome is rare with the doses and methods of administration typically used to treat labor pain. All of the opioids have

some drug–drug interactions that can be clinically significant in some populations.[83]

All opioids are lipid soluble, so they readily cross the placenta and concentrate in the fetus. Fetal effects include decreased fetal heart rate variability and neonatal respiratory depression; in addition, there is controversy about whether opioids adversely affect early neurobehavioral scores in the newborn.[91] Naloxone (Narcan) is the opiate/opioid antagonist given to the newborn to reverse the respiratory depression effect of opiates administered to the woman during labor. Naloxone must be available whenever opiate and opioid medications are used. The dose is 0.01 mg/kg of body weight, and the effect becomes evident 2 to 5 minutes after an intramuscular injection and 1 to 2 minutes after an intravenous injection. The peak effect occurs in 5 to 15 minutes.[91]

Inhaled Analgesia: 50:50 Nitrous Oxide/Oxygen

Nitrous oxide inhalation analgesia for labor pain management consists of 50% nitrous oxide and 50% oxygen (N_2O/O_2). This therapy has only recently been reintroduced in the United States, but it has been in widespread use in Northern Europe and Canada for decades. This gaseous agent is widely considered both safe and effective.[86,92-101] Nitrous oxide is a weak anesthetic when used in high doses and has analgesic and anxiolytic effects at low doses.[92] N_2O/O_2 can be used as the sole analgesic, or it can be administered in combination with opioids or opioid agonist–antagonists. The gas mixture is blended in a machine attached to nitrous oxide and oxygen tanks or attached to piped nitrous oxide and oxygen if the hospital has these gases available in labor rooms (**Figure 27-6**). It is then self-administered by the woman via a handheld facemask over her nose and mouth or via a mouthpiece (**Figure 27-7**). A demand valve opens with inhalation and closes with exhalation. Self-administration is not only empowering for the woman, but also acts as a safety mechanism because it is almost impossible to overdose with N_2O/O_2 when it is self-administered. Guidelines for use of this agent are listed in **Table 27-6**.[92]



Figure 27-6 A nitrous oxide machine.

Courtesy of Porter Instrument Division, Parker Hannifin Corp.



Figure 27-7 A woman resting on a birthing ball, using 50:50 nitrous oxide/oxygen for pain relief during labor.

Courtesy of Judith Bishop, CNM, MPH, MSN.

Table 27-6 **Clinical Application of 50% N_2O/O_2 for Labor Analgesia**

Steps	Description
Precautions	1. Determine that there are no contraindications, such as inability to hold the face mask, impaired

	oxygenation, or hemodynamic instability. 2. Use with caution if other sedating drugs are administered concomitantly.
Preparation	1. Inform the woman of the potential side effects of nausea, vomiting, and/or dizziness. 2. Instruct her on how to hold the mask so it creates a seal; instruct her on timing of inhaation. 3. Review with the woman and any labor support persons that only the woman can hold the mask. a. Sedation from N_2O/O_2 is possible only if the woman continues to inhale the gas after she can no longer hold the mask tightly to her face. N_2O/O_2 dissipates rapidly with exhalation and the effects resolve very quickly. b. Self-administration gives the woman personal control of management of pain, and this control may potentiate the analgesic effect.
Procedure	1. The woman holds the mask over her mouth and nose, creating a seal that allows activation of a second-stage regulator. 2. She begins breathing the gas 30 seconds before a contraction starts; the onset of action is approximately 30 seconds, and the full analgesic effect occurs in 50 seconds. It often takes three to four contractions to learn the correct technique. 3. The woman exhales into the mask to facilitate scavenging.
Intermittent versus continuous inhalation	1. Intermittent inhalation 30 seconds prior to the onset of contraction maximizes the peak effect of N_2O/O_2 so the peak analgesic effect occurs during the peak of the uterine contraction. 2. Continuous inhalation is easier to initiate, but in this scenario the woman is exposed to the effects of the gas between contractions when the central nervous system effects of dizziness or dysphoria could be bothersome.

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Women have generally reported high degrees of satisfaction with the use of N_2O/O_2 for pain relief in labor even when they report lower or moderate degrees of pain relief. This finding may relate to maternal control of the agent, or possibly some anxiolytic effect of the agent, or a mixture of analgesia, anxiolytic effect, and increased self-efficacy.[86,98] The analgesic effect of N_2O/O_2 is relatively mild with the doses used for labor, but in combination with the anxiolytic effect, it effectively reduces the perception of pain.[94] Approximately 40% to 60% of women who start using N_2O/O_2 convert to neuraxial analgesia later in labor.[99,100] N_2O/O_2 can also provide effective analgesia for placement of a Foley bulb for labor induction, postpartum suturing, or manual removal of the placenta, as well as gynecologic procedures such as a difficult insertion of an intrauterine contraceptive device.

Potential side effects of N_2O/O_2 include nausea in 5% to 40% of women and vomiting in as many as 15%, dizziness, and dysphoria, although the occurrence of these side effects is rare.[96] Administration of N_2O/O_2 must occur in settings where scavenging of the exhaled gases occurs so it does not exit out into the air. No fetal heart rate abnormalities have been attributed to its use, and this agent does not affect uterine contractility.[86,93-96] The half-life in the neonate is

approximately 3 minutes, and no neonatal adverse outcomes have been found following maternal use during labor.[86] Adverse effects on the fetus or newborn have not been seen in the extensive history of this agent as a labor analgesic, although some theoretical risks are associated with higher doses than those used for labor analgesia that have not been well studied. Likewise, there are no risks to healthcare personnel who are in a room where N_2O/O_2 is being used.[94]

In the body, nitrous oxide oxidizes an active form of cobalamin (vitamin B_{12}). Cobalamin is used in the enzymatic reaction of methionine synthase that converts homocysteine into methionine. Methionine uses folate to synthesize myelin, DNA, and RNA. Acting through this pathway, prolonged use of nitrous oxide has resulted in neurodegeneration in animal studies. Thus, despite a long record of safety in the concentrations and total amounts used by laboring women, there is some controversy about use of N_2O/O_2 by pregnant women.[94] Contraindications to use of N_2O/O_2 are rare but include persons with extreme vitamin B_{12} deficiency, or other conditions that might lead to vitamin B_{12} deficiency. Other contraindications include inability of the woman to hold the mask, impaired oxygenation, or hemodynamic instability.[94]

Neuraxial Analgesia

Neuraxial analgesia encompasses both epidural and spinal analgesic techniques. Neuraxial analgesia must block T10 to L1 during the first stage of labor and extend to S2 to S4 during the late first stage and second stage of labor if it is to block sensory input from the uterus and perineum. Neuraxial analgesia is performed by anesthesia specialists (nurse anesthetists and anesthesiologists), and the specific technique depends on the needs of the laboring woman as well as the availability and choice of the anesthetist. Regional analgesic techniques include spinal (intrathecal injection), epidural, and combined spinal/epidural injections. The effect depends on where the medication is placed, which medications are used, and which dose is administered. Absolute contraindications to neuraxial analgesia are rare but include coagulopathy, infection of the lower back, and increased intracranial pressure.

Spinal Analgesia

To induce spinal anesthesia, a single injection of opioid is administered in the intrathecal (subarachnoid) space that contains cerebrospinal fluid, spinal nerves, and blood vessels, all of which are enclosed by the membrane of the dura mater. The onset of action with this therapy is rapid, but its duration is limited to the duration of the specific opioid used.

Spinal analgesia has no effect on motor ability, so women can ambulate or assume any position they want. This therapy is often called “the walking epidural” despite the fact that this technique does not deliver analgesia into the epidural space. Spinal analgesia is associated with transient fetal heart rate abnormalities such as prolonged decelerations and a transient increase in uterine contractions and uterine tone.[102] As a consequence, close observation of the fetal heart rate is necessary for a period of time after the injection. The major drawback to spinal analgesia is that it requires a dural puncture, which is not safe to do more than once.

Therefore, a spinal can be performed only once. Because it is not easy to predict when during labor a woman will need analgesia the most, spinal analgesia is not used as often as is epidural analgesia. Spinal analgesia is most appropriate for women who are having a rapidly progressing course of labor without the need for an extended block (epidural).

Side effects of intrathecal analgesia may include pruritus or nausea and vomiting, which occur secondary to the effect of the opioid rather than the technique itself. Pruritus (skin itching) is the most common side effect, occurring in varying degrees of severity in 50% to 90% of women.[103] Diphenhydramine (Benadryl 25 mg given intravenously) can be of value in this situation, or in severe cases naloxone (Narcan) can be administered with caution. If naloxone is needed, the anesthetic effect of the spinal analgesia will also be reversed. Urinary retention and respiratory depression are less common side effects.

Spinal analgesia is more often used today as part of a combination spinal/epidural, wherein the spinal technique is used to inject a small dose of opioid and, as the needle is withdrawn, a catheter is placed in the epidural space. When the spinal analgesia wears off, medication is injected to initiate epidural analgesia.

Epidural Analgesia

Epidural analgesia provides the most effective relief of labor pain compared to opioids and inhalation anesthetics. Women using epidural analgesia have lower pain scores and are more satisfied with their analgesia than women using parenteral opioids when the experience of pain is evaluated.[104] The most recent study that compared the effectiveness of epidural analgesia to that of N_2O/O_2 found that women who relied on N_2O/O_2 alone (without converting to neuraxial analgesia) reported higher scores of satisfaction compared to women who used epidural analgesia, despite lower scores of actual pain diminution.[99] This finding confirms the observation that direct analgesia is not the only factor associated with birth satisfaction. Over the past three decades, the use of epidural analgesia/anesthesia for managing labor pain has increased dramatically, reflecting improvements in the administration techniques, greater access to the procedure, and consumer demand.

Mechanism of Action

The epidural space is located exterior to the dura mater, bounded posteriorly by the ligamentum flavum (**Figure 27-8**). It contains nerve roots, fat, lymphatics, and blood vessels. When an anesthetic drug is placed in this space, impulses traveling in the sensory and motor nerves in contact with the drug are blocked. The effect of epidural analgesia depends greatly on the drug, the dose placed in the epidural space, and the administration technique (i.e., intermittent versus continuous infusion).

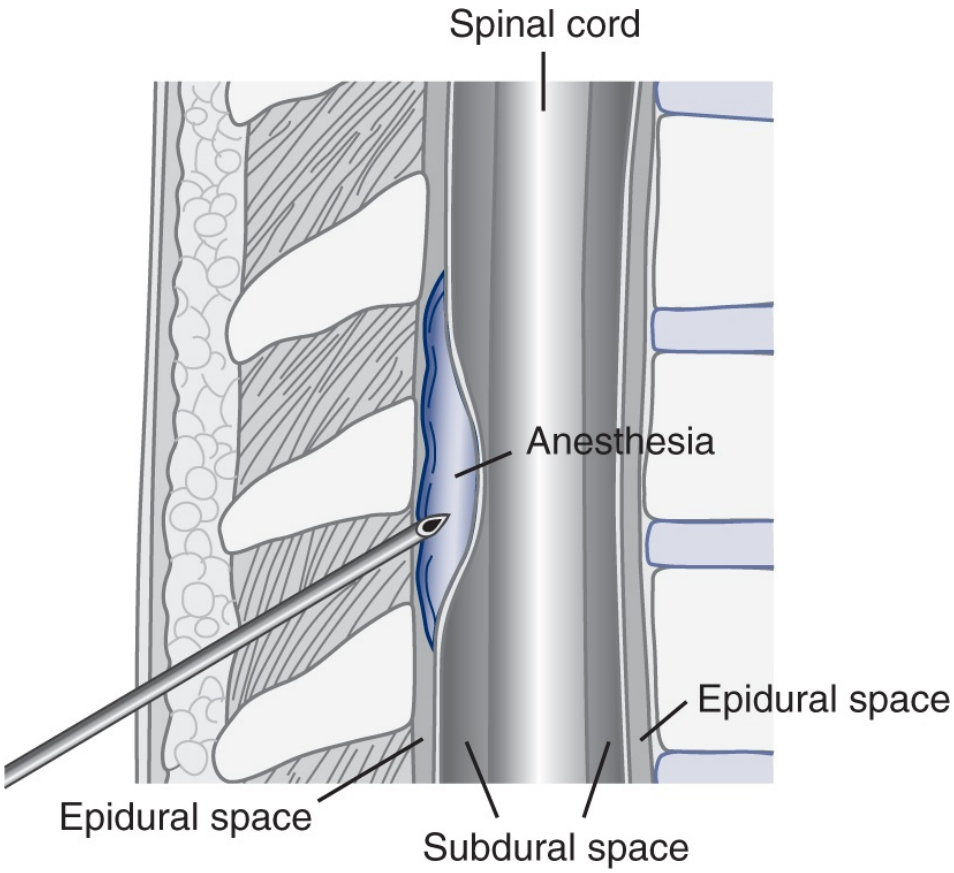


Figure 27-8 Epidural space.

The anesthetic agents most commonly used in epidural analgesia are bupivacaine (Marcaine) and ropivacaine (Naropin). Fentanyl or morphine can be added to potentiate the analgesic effect and allow smaller doses of the anesthetic drug. Often epinephrine is added to the local anesthetic solution because it prolongs the duration of action. Bupivacaine, an amide anesthetic with a long duration of action, provides an excellent sensory block, while preserving some motor function when used at low doses. A concentration of 0.25% or 0.5% is often used for labor analgesia. Fentanyl and remifentanil are synthetic opioids with potency 100 times greater than that of morphine; one of these opioids is frequently added to the anesthetic to augment the analgesic effect.

Some of the individual differences noted in the quality of neuraxial blocks, despite use of consistent technique, may reflect individual anatomic variations that occur in the epidural space. For example, differences in nerve root size or the presence of membranes in the epidural space that disallow equal spread of the medication to all nerves passing through the epidural space may lead to variable effects in individual women.

Indications and Contraindications

Indications for the use of epidural analgesia/anesthesia include relief of pain at any time during labor following maternal request, operative vaginal birth, cesarean section, and need to diminish maternal expulsive efforts (e.g., maternal cardiac or ophthalmologic disease). If a woman’s medical or obstetric condition places her at increased risk for needing an emergent

cesarean section, placement of an epidural catheter may be recommended to avoid loss of time if the emergency does, in fact, occur. This “double setup” is often done, for example, if membranes are to be ruptured when the fetal presenting part is not in the pelvis, for a vaginal breech birth, or for a woman with a multifetal gestation.

Effective epidural analgesia may be particularly helpful for some women who have a history of sexual abuse. For some of these women, labor can induce anxiety about painful stimuli in the vaginal/rectal area or flashbacks of abusive events. Thus the “numbing” effect of epidural analgesia can mitigate some aspects of the post-traumatic stress caused by labor and birth.

Absolute contraindications to epidural or spinal anesthesia include refusal, inability to cooperate, skin or soft-tissue infection at the site of needle placement, frank coagulopathy, untreated sepsis, and maternal hemodynamic instability. Relative contraindications include heparin therapy or thrombocytopenia, and neurologic disease of the spinal cord. In addition, women with the conditions listed in **Table 27-7** should be offered consultation with an anesthesia provider prior to labor.[105] Contrary to popular belief, a tattoo is not a contraindication to epidural analgesia.

Table 27-7 Indications for Anesthesiology Consultation

The following are some of the most common indications for consultation with an anesthesiologist during the antenatal period or peripartum period. In many cases, a phone consultation may be sufficient; in other cases, a face-to-face consultation will be appropriate.
Cardiac Disease
Congenital and acquired disorders such as repaired tetralogy of Fallot and transposition of the great vessels
Cardiomyopathy
Valvular disease such as aortic and mitral stenosis, tricuspid regurgitation, and pulmonary stenosis
Pulmonary hypertension and Eisenmenger syndrome
Rhythm abnormalities such as supraventricular tachycardia and Wolff–Parkinson–White syndrome
Presence of an implanted pacemaker or defibrillator
Hematologic Abnormalities or Risk Factors
Immune and gestational thrombocytopenia
Coagulation abnormalities such as von Willebrand disease
Current use of anticoagulant medications
Jehovah’s Witness
Spinal, Muscular, and Neurologic Disease
Structural vertebral abnormalities and prior surgeries such as vertebral fusion and rod placement
Prior spinal cord injury
Central nervous system problems such as known arterial–venous malformation, aneurysm, Chiari malformation, or ventriculoperitoneal shunt
Major Hepatic or Renal Disease
Chronic renal insufficiency
Hepatitis or cirrhosis with significantly abnormal liver function tests or coagulopathy
History of or Risk Factors for Anesthetic Complications
Anticipated difficult airway

Obstructive sleep apnea
Previous difficult or failed neuraxial block
Malignant hyperthermia
Allergy to local anesthetics
Obstetric Complications That May Affect Anesthesia Management
Placenta accreta
Nonobstetric surgery during pregnancy
Planned cesarean delivery with concurrent major abdominal procedure
Miscellaneous Medical Conditions That May Influence Anesthesia Management
Body mass index of 50 or greater
History of solid organ transplantation
Myasthenia gravis
Dwarfism
Sickle cell anemia
Neurofibromatosis

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Side Effects and Adverse Effects of Epidural Analgesia

The anesthesia provider will review the risks, benefits, side effects, adverse effects, and alternative therapies prior to initiating the procedure. Midwives who care for women who are administered epidural analgesia need to know the effects of this procedure and be prepared to manage complications. Often the anesthesia provider focuses on maternal risks associated with the procedure or the block, while the maternity care provider addresses the effect of neuraxial analgesia on labor progress and the fetus or newborn.

The primary side effect related to performance of this procedure is postdural puncture headache following accidental dural puncture. If the epidural needle inadvertently pierces the dura and cerebrospinal fluid leaks into the epidural space, the drop in intrathecal pressure can cause a positional headache, which increases in severity when the woman is in the sitting or standing position. Resolution of the headache occurs spontaneously within 4 to 5 days. If not severe, this headache may be treated with oral hydration, caffeine, and simple analgesics such as acetaminophen (Tylenol) or ibuprofen (Motrin, Advil). If the headache is severe or prolonged, it can be treated with a blood patch, which is an epidural injection of the woman's blood that seals the leaking membrane.

Back pain is a common side effect of labor and birth, with or without an epidural. When lower back pain occurs following regional analgesia, the back pain may be attributed to the epidural. However, it is not known if the etiology of back pain following use of epidural analgesia for labor and birth is related to physiologic changes of pregnancy, effects of labor itself, or the epidural and resulting regional block.[106,107]

Adverse effects related to performance of the epidural analgesia procedure are rare, but include epidural abscess (fewer than 1 in 60,000), epidural hematoma (1 in 250,000), and

serious neurological injury (1 in 36,000).[106] Inadvertent intrathecal (subarachnoid) or intravascular placement can result in toxicity (**Table 27-8)**. Total spinal anesthesia occurs in 0.03% of women when the level of the anesthesia rises dangerously high, resulting in paralysis of the respiratory muscles including the diaphragm (1 in 4000).[106] When this effect occurs, the woman will experience numbness of the hands and fingers, as well as dyspnea. Treatment is accomplished with assisted respiration until the anesthesia resolves.

Table 27-8	Signs of Intrathecal or Systemic Toxicity Following Epidural Injection
Placement of Anesthetic	**Signs and Symptoms of Toxicity**
Intrathecal	Rapid cephalad spread of anesthesia
	Profound hypotension
	Respiratory depression
	Loss of consciousness
Intravascular	Tinnitus
	Dizziness
	Metallic taste in the mouth
	Disorientation
	Seizures
	Cardiac dysrhythmias
	Cardiac arrest

Side effects of local anesthetic placed in the epidural space include hypotension, urinary retention, and leg numbness and weakness. Local anesthetics cause a sympathetic blockade that leads to decreased vascular tone, peripheral vasodilation, and subsequent drop in blood pressure. Hypotension (defined as a systolic blood pressure less than 100 mm Hg) is noted in approximately 10% of women following administration of epidural analgesia.[108] Significant complications are uncommon, but blood flow to the uterus diminishes with hypotension, and fetal heart rate decelerations (e.g., prolonged deceleration or late decelerations) frequently appear for a short time 10 to 20 minutes after the epidural is emplaced. These fetal heart rate patterns are associated with moderate fetal heart rate variability, usually resolve spontaneously or following a bolus of intravenous fluids, and are not associated with adverse newborn outcomes. In rare cases, ephedrine is required to elevate maternal blood pressure and restore blood flow to the uterus. This is most commonly accomplished with incremental doses (5–10 mg administered intravenously) of ephedrine, a mixed catecholamine agonist that increases blood pressure and heart rate without constricting uterine blood vessels.

Bladder distension can occur when the woman experiences loss of the sensation to void, relaxation of overall bladder tone, and loss of control of the urinary sphincter. Intermittent catheterization during labor and/or at the time of birth is common. If many hours will elapse between initiation of an epidural and birth, and the woman is unable to void on her own, an indwelling catheter or intermittent catheterization may be indicated. The catheter should be discontinued during the second stage of labor.

An increase in the incidence of maternal fever, defined as a body temperature equal to or

higher than 38°C (100.4°F), has been demonstrated in women who have epidural analgesia during labor.[109] Women who develop temperature elevations tend to have increases that start immediately after the epidural is initiated and average 0.18°C (0.33°F) per hour. The occurrence of fever equal to or higher than 38°C (100.4°F) is apparent after approximately 4 hours.[110,111] Most likely, epidural-related fever is attributable to thermoregulatory alterations such as a decrease in heat dissipation secondary to sympathetic block of sweating. Some women who develop a fever after epidural initiation, however, may have subclinical chorioamnionitis.[110,111] Unfortunately, it is not possible to distinguish fever of benign versus infectious origin, so all women who develop a fever in labor are treated for chorioamnionitis. Newborns of women who develop a fever during labor are more likely to be subjected to evaluations for sepsis in the first few days of life.[111]

A secondary analysis of data from a large study that followed women in labor to determine fetal position confirmed the increased incidence of a rise in temperature in women who have an epidural.[112] This analysis found that a rise in temperature was positively associated with increased incidence of occiput posterior position at birth despite the groups having a nonsignificant difference in the incidence of occiput posterior position at the onset of labor.[112] Furthermore, the incidence of occiput posterior position at birth and cesarean section increased proportionately as the maternal temperature increased (17.2% vs 9.2% in women with and without fever respectively; odds ratio [OR], 2.0; 95% CI, 1.5–2.8).[112] This finding remained significant after controlling for confounding factors such as body mass index, gestational age, birth weight, and length of labor (OR, 1.5; 95% CI, 1.1–2.1).[112]

Effect of Neuraxial Analgesia on Labor Progress

During the many years over which epidural analgesia has been in use, there has been a great deal of controversy regarding the potential effects of this therapy on the course and outcomes of labor.[107,113] The effect of epidural analgesia on duration of labor is difficult to determine due to many confounding variables. Retrospective studies have found the use of epidural analgesia to be associated with longer first and second stages of labor. The largest and most recent of these studies, which was conducted by Zhang et al. (n = 62,400), found the 95th percentile was approximately 50 minutes longer for the second stage in nulliparous women.[113] This extended duration is presumed to occur secondary to the lack of sensation for bearing down and perhaps the relaxation of the levator ani muscles that make up the pelvic floor. The meta-analyses of RCTs that compared labor outcomes in women with and without epidural analgesia have not found epidural analgesia to be associated with a prolongation of the second stage of labor in the aggregate (MD, 13.66 minutes; 95% CI, 6.67–20.66; 11 trials; 2981 women).[109] In addition, it appears that lower concentrations of anesthetic are less likely to cause longer durations of the second stage of labor.[114]

However, the aggregate mean values for duration of labor do not tell the full story. In general, epidural analgesia is often associated with a slow rate of cervical dilation in the first stage of labor, especially if such analgesia is initiated during the latent phase of labor. Women who need epidural analgesia in the latent phase of labor for pain relief, however, may have an

underlying uterine dysfunction that results in a longer labor. Thus, it is not clear if the analgesia slows the first stage of labor or if women who need epidural analgesia have inherent differences that result in slower labors.

Conversely, some women experience an accelerated course of labor after epidural analgesia is initiated. This effect may be due to improved uterine contractility after previous inhibition from markedly elevated catecholamine levels in women who had severe pain. Alternatively, the higher level of pain that initiated the request for epidural analgesia might have reflected rapid dilation that occurred spontaneously. Thus, the combination of some women whose labors are slowed by epidural analgesia with some women whose labors are faster after epidural analgesia is initiated results in an aggregate mean duration of the first stage that is no different than the duration of the first stage in women who do not use epidural analgesia. Given these offsetting groups, it is difficult to draw clinically meaningful conclusions from these data.

Epidural analgesia is not associated with an increase in cesarean births, but women who have epidural analgesia are more likely to have an operative vaginal birth (RR, 1.42; 95% CI, 1.28–1.57).[109] The incidence of instrumental deliveries can be reduced with the use of a more dilute solution of local anesthetic.[115] Some practitioners have advocated cessation of epidural analgesia during second-stage labor to enable the woman to experience the urge to push and to be able to actively participate in expulsive efforts. For a few women, this is a successful approach; for many others, the reoccurrence of severe pain and anxiety may interfere with their ability to push. Discontinuing the epidural infusion is not recommended.

Fetal and Neonatal Side Effects and Adverse Effects of Neuraxial Analgesia

Both anesthetic drugs and opioids are highly lipid soluble and can cross into the fetal circulation in small amounts, depending on the quantity found in the maternal circulation. Nevertheless, neonatal outcomes such as poor 5-minute Apgar scores and low umbilical artery pH have not been shown to be significantly different between women using epidural analgesia and those using intravenous opioids or no medications.[113] Neurologic studies of epidural analgesia-exposed infants, opioid-exposed infants, and nonmedicated infants have not identified significant differences in performance between any of these groups.

The effect of epidural analgesia on breastfeeding outcomes has also been controversial. Women cannot be randomized prospectively to epidural analgesia or no-epidural analgesia groups, and because women who have epidural analgesia tend to have longer second stages, more fevers, and a higher rate of operative birth, it is not clear if lower rates of breastfeeding initiation occur secondary to the epidural analgesia or to the labor course. Overall, rates of breastfeeding initiation and continuation through 6 weeks postpartum appear to be unchanged regardless of the method of analgesia.[107]

Pudendal Block

A pudendal block is used to alleviate pain stemming from the perineum and vagina during the second stage of labor.[116] Before the widespread adoption of epidural analgesia for mitigating labor pain, pudendal blocks were the preferred analgesic technique for birth. A pudendal block

can be an effective form of analgesia if the woman has a contraindication to neuraxial anesthesia and an anesthetic is needed for a low vaginal or perineal procedure. The lower vagina, perineum, and vulva obtain most of their sensory and motor innervation from sacral nerve roots 2, 3, and 4 via the pudendal nerve. Infiltration of a local anesthetic around the pudendal nerve at the level of the ischial spine results in analgesia of these areas. Pudendal nerve blocks do not decrease the pain associated with uterine contractions or cervical dilation. The appendix in the *Second Stage of Labor and Birth* chapter describes the procedure for performing a pudendal block in detail.[116]

Pudendal blocks are simple to perform and provide satisfactory analgesia in many cases. However, 10% to 50% of bilateral pudendal blocks are ineffective on one or both sides. Pudendal blocks do not interfere with uterine contractions, so they have no adverse effects on labor progress or maternal or fetal well-being. Neonatal local anesthetic toxicity after maternal pudendal block is extremely rare. A case report described three cases of neonatal lidocaine intoxication, which manifested as transient hypotonia, apnea, seizures, mydriasis, cyanosis, and need for mechanical ventilation; all of the neonates recovered completely.[117] Potential complications are rare, but may include post-block fetal bradycardia, systematic toxicity after intravascular administration, lower-extremity paresthesia, vaginal hematoma, and infection.[116-117]

General Anesthesia

General anesthesia refers to the use of an inhaled anesthetic agent to induce sleep for obstetric applications, primarily for emergency cesarean birth. Induction of anesthesia is accomplished with short-acting agents that render the woman unconscious, such as thiopental or propofol. After the induction agent is administered, a muscle relaxant such as succinylcholine is typically administered to facilitate intubation.

Intubation generally proceeds without complications; however, in pregnant women, intubation is several times more complicated than in the nongravid person. For this reason, general anesthesia is avoided when possible, due to maternal and fetal side effects that are undesirable and possibly life threatening. General anesthesia complications due to inability to intubate and maternal aspiration remain leading causes of anesthesia-related maternal mortality.[118] Even so, true emergencies such as cord prolapse and acute placental abruption may necessitate the use of general anesthesia if neuraxial anesthesia is not already in place. For many clinical situations where a surgical birth becomes necessary, there is adequate time for placement of spinal anesthesia, which has less potential for maternal–fetal complications.

Conclusion

Unmedicated or medicated, easy labor and birth or complicated labor and birth, the midwife sets the tone for all involved in the care of the laboring woman. Speaking in a low voice, dimming bright lights, and making all involved as comfortable as possible help women feel cared for and supported. The midwife always has a special opportunity to make each birth experience one that shows respect for birth, even if the birth takes place in the midst of a high-technology environment. Providing support to the woman in labor and facilitating her choices for coping with the pain of labor are key responsibilities for the midwife, regardless of birth setting.

Resources

Articles and Textbooks
Acupressure and Acupuncture
Schlaeger JM, Gabzdyl EM, Bussell JL, et al. Acupuncture and acupressure in labor. *J Midwifery Womens Health*. 2016: 62(1):12-28.
Hydrotherapy in Labor
Kane Low L, Nutter EM. A model practice template for hydrotherapy in labor and birth. *J Midwifery Womens Health*. 2017;63(1):120-126.
Maternal Positions
Cohen SR, Thomas CR. Rebozo technique for fetal malposition in labor. *J Midwifery Womens Health*. 2015;60(4):445-451.
Simkin P. Supportive care during labor: a guide for busy nurses. *JOGNN.* 2002;31:721-722.
This article has several well-done figures that show different maternal positions and includes discussion of how each position affects the feto-pelvic relationship.
Nonpharmacologic Support Techniques
Simkin P, Bolding A. Update on nonpharmacologic approaches to relieve labor pain and prevent suffering. *J Midwifery Womens Health.* 2004;49(6):489-505.
This article also has figures showing techniques for mitigating back pain.
Simkin P, Ancheta R, Hanson L. *The Labor Progress Handbook: Early Interventions to Prevent and Treat Dystocia.* 3rd ed. Hoboken, NJ: Wiley-Blackwell; 2011.

Organization	Description	Webpage
Childbirth Connection	Organization that promotes evidence-based maternity care. Its website has resources for midwives and women.	http://www.childbirthconnection.org/resources/
Doula Organization of North America (DONA) International	The DONA website contains training and education for doulas as well as resources for labor support.	https://www.dona.org
Penny Simkin, PT, childbirth educator	Ms. Simkin is a renowned childbirth educator who has developed innovative techniques for supporting women in labor. Her website contains videos, articles, and other resources.	https://www.pennysimkin.com/articles-resources/

References

1. Simkin P, Bolding A. Update on nonpharmacologic approaches to relieve labor pain and prevent suffering. *J Midwifery Womens Health*. 2004;49(6):489-505.
2. Simkin P. Just another day in a woman's life? Part II. Nature and consistency of women's long-term memories of their firs birth experiences. *Birth*. 1992;19(2):64-81.
3. Melzack R. From the gate to the neuromatrix. *Pain*. 1999;S121-S126.
4. Trout, KK. The neuromatrix theory of pain: implications for selected nonpharmacologic methods of pain relief for labor. *J Midwifery Womens Health*. 2004;49(6):482-488.
5. Merskey H, Bogduk N, eds. Part III: pain terms, a current list with definitions and notes on usage. In: IASP Task Force or Taxonomy. *Classification of Chronic Pain*. 2nd ed. Seattle, WA: IASP Press; 1994:209-214.
6. Lowe NK. The nature of labor pain. *Am J Obstet Gynecol*. 2002;186:S16-S24.
7. Eltzchig HK, Lieberman ES, Camann WR. Regional anesthesia and analgesia for labor and delivery. *N Engl J Med*. 2003;348:419-432.
8. Mendell LM. Constructing and deconstructing the gate theory of pain. *Pain*. 2014;155(2):210-216.
9. Melzack R. Low-back pain during labor. *Am J Obstet Gynecol*. 1987;156:901-905.
10. Melzack R. The myth of painless childbirth: the John J. Bonica lecture. *Pain*. 1984;(19):321-327.
11. Melzack R, Kinch R, Dobkin P, Lebrun M, Taenzer P. Severity of labor pain: influence of physical as well as psychological variables. *Can Med Assn J*. 1984;30(5):579-584.
12. Lowe NK, Corwin EJ. Proposed biological linkages between obesity, stress, and inefficient uterine contractility in humans. *Med Hypotheses*. 2011;76:755-760.
13. Hodnett ED. Pain and women's satisfaction with the experience of childbirth: a systematic review. *Am J Obstet Gynecol*. 2002;186:S160-S173.
14. Lally JE, Murtagh MJ, Macphail S, Thomson R. More in hope than expectation: a systematic review of women's expectations and experiences with pain relief in labour. *BMC Med*. 2008;6:7.
15. Mogil S. Pain genetics: past, present and future *Trends Genet*. 2012;28(6):258-266.
16. Landau R. Genetic contributions to labor pain and progress. *Clin Perinatol*. 2013;40(3):575-587.
17. Landau R, Smiley R. Pharmacogenetics in obstetric anesthesia. *Best Pract Res Clin Anaesthesiol*. 2017;31(1):23-34.
18. Eisenach JC, Pan PH, Smiley R, Lavand'homme P, Landau R, Houle TT. Severity of acute pain after childbirth, but not type of delivery, predicts persistent pain and postpartum depression. *Pain* 2008;140(1):87-94.
19. Roosevelt L, Low LK. Exploring fear of childbirth in the United States through a qualitative assessment of the Wijma Delivery Expectancy Questionnaire. *J Obstet Gynecol Neonatal Nurs*. 2016;45(1):28-38.
20. Lowe NK. Self-efficacy for labor and childbirth fears in nulliparous pregnant women. *J Psychosom Obstet Gynaecol*. 2000;21(4):219-224.
21. Laursen M, Johansen C, Hedegaard M. Fear of childbirth and risk for birth complications in nulliparous women in the Danish National Birth Cohort. *BJOG*. 2009;116(10):1350-1355.
22. Haines HM, Rubertsson C, Pallant JF, Hildingsson I. The influence of women's fear, attitudes and beliefs of childbirth on mode and experience of birth. *BMC Pregn Childbirth*. 2012;12:55. doi:10.1186/1471-2393-12-55.
23. LoGiudice JA, Beck CT. The lived experience of childbearing from survivors of sexual abuse: "it was the best of times, it was the worst of times." *J Midwifery Womens Health*. 2016;61(4):474-481.
24. Montgomery E. Feeling safe: a metasynthesis of the maternity care needs of women who were sexually abused in childhood. *Birth*. 2013;40(2):88-95.
25. Gagnon AJ, Sandall J. Individual or group antenatal education for childbirth or parenthood, or both. *Cochrane Database Syst Rev*. 2007;3:CD002869. doi:10.1002/14651858.CD002869.pub2.
26. Maimburg RD, Vaeth M, Durr J, Hvidman L, Olsen J. Randomised trial of antenatal training sessions to improve birth process. *BJOG*. 2010;117:921-928.
27. Lowe NK. Context and process of informed consent for pharmacologic strategies in labor pain care. *J Midwifery Women Health*. 2004;49:250-260.
28. Gulliver BG, Fisher J, Roberts L. A new way to assess pain in laboring women: replacing the rating scale with a "coping" algorithm. *Nurs Womens Health*. 2008;12(5):405-408.
29. Roberts L, Gulliver B, Fisher J, Cloyes K. The coping with labor algorithm: an alternate pain assessment tool for the laboring woman. *J Midwifery Womens Health*. 2010;55(2):107-116.
30. Simkin P. The three R's in childbirth preparation: relaxation, rhythm and ritual. Available at: https://www.pennysimkin.com/the-3-rs-in-childbirth-preparation-relaxation-rhythm-and-ritual/. Accessed October 17, 2017.
31. Bergstrom L, Richards L, Morse JM, Roberts J. How caregivers manage pain and distress in second stage labor. *J Midwifery Womens Health*. 2010;55(1);38-40.
32. Beck CT, Gable RK, Sakala C, Declercq ER. Posttraumatic stress disorder in new mothers: results from a two-stage U.S

national survey. *Birth*. 2011;38(3):216-227.
33. Declercq ER, Sakala C, Corry MP, Applebaum S. *Listening to Mothers II: Pregnancy and Birth*. New York, NY: Childbirth Connection; 2006. Available at: http://www.nationalpartnership.org/research-library/maternal-health/listening-to-mothers-ii-2006.pdf. Accessed February 20, 2017.
34. Sheiner E, Sheiner EK, Hershkovitz R, Maxor M, Katz M, Shoham-Vardi I. Overestimation and underestimation of labor pain. *Eur J Obstet Gynecol Reprod Biol*. 2000;91:37-40.
35. Sheiner EK, Sheiner E, Shoham-Vardi I, Mazor M, Katz M. Ethnic differences influence care giver's estimates of pain during labour. *Pain*. 1999;81(3):299-305.
36. Baker A, Ferguson SA, Roach GD, Dawson D. Perceptions of labour pain by mothers and their attending midwives. *J Advanced Nurs*. 2000;32(2):171-179.
37. Williams AC, Morris J, Stevens K, Gessler S, Cella M, Baxter J. What influences midwives in estimating labour pain? *Eur J Pain*. 2013;17(1):86-93.
38. Hodnett E, Stremler R, Weston J, McKeever P. Re-conceptualizing the hospital labor room: the PLACE (Pregnant and Laboring in an Ambient Clinical Environment) pilot trial. *Birth*. 2009;36(2):159-166.
39. Walburg V, Friederich F, Callahan S. Embarrassment and modesty feelings during pregnancy, childbirth and follow-up care a qualitative approach. *J Repro Infant Psychol*. 2014;32(2):126-136.
40. Declercq ER III, Sakala C, Corry MP, Applebaum S, Herrlich A. *Listening to Mothers III: Pregnancy and Birth: Report of the Third National U.S. Survey of Women's Childbearing Experiences*. New York, NY: Childbirth Connection; 2013. Available at: http://www.childbirthconnection.org/listeningtomothers. Accessed October 30, 2016.
41. Bohren MA, Hofmeyr GJ, Sakala C, Fukuzawa RK, Cuthbert A. Continuous support for women during childbirth. *Cochrane Database Syst Rev*. 2017;7:CD003766. doi:10.1002/14651858.CD003766.pub6.
42. Fortier JH, Godwin M. Doula support compared with standard care: meta-analysis of the effects on the rate of medical interventions during labour for low-risk women delivering at term. *Can Fam Physician*. 2015;61(6):e284-e292.
43. Hodnett ED, Lowe NK, Hannah ME, et al. Effectiveness of nurses as providers of birth in North American hospitals: a randomized controlled trial. *JAMA*. 2002;288:1373-1381.
44. Paterno MT, Van Zandt SE, Murphy J, Jordan ET. Evaluation of a student-nurse doula program: an analysis of doula interventions and their impact on labor analgesia and cesarean birth. *J Midwifery Womens Health*. 2012;57(1):28-34.
45. Hodnett ED. Caregiver support for women during childbirth. *Cochrane Database Syst Rev*. 2002;1:CD000199.
46. Hofmeyr GJ, Nikodem VC, Wolman WL, Chalmers BE, Kramer T. Companionship to modify the clinical birth environmer effects on progress and perceptions of labour, and breastfeeding. *Br J Obstet Gynaecol*. 1991;98:756-764.
47. Nutter E, Meyer S, Shaw-Battista J, Marowitz A. Waterbirth: an integrative analysis of peer-reviewed literature. *J Midwifery Womens Health*. 2014;59(3):286-319.
48. Benfield RD, Hortobagyi T, Tanner CJ, Swanson J, Heltkemper MM, Newton ER. The effects of hydrotherapy on anxiety pain, neuroendocrine response, and contraction dynamics during labor. *Bio Res Nurs*. 2010;12(1):28-36.
49. Benfield RD. Hydrotherapy in labor. *J Nurs Schol*. 2002;34(4):347-352.
50. Cluett ER, Burns E. Immersion in water in labour and birth. *Cochrane Database Syst Rev*. 2009;2:CD000111. doi:10.1002/14651858.CD000111.pub3.
51. Kane Low L, Nutter EM. A model practice template for hydrotherapy in labor and birth. *J Midwifery Womens Health*. 2017;63(1):120-126.
52. Eriksson M, Mattson LA, Ladfors L. Early or late bath during the first stage of labor: a randomized study of 200 women. *Midwifery*. 1997;13(3):146-148.
53. Schlaeger JM, Gabzdyl EM, Bussell JL, et al. Acupuncture and acupressure in labor. *J Midwifery Womens Health*. 2016; 62(1):12-28.
54. Smith CA, Collins CT, Crowther CA, Levett KM. Acupuncture or acupressure for pain management in labour. *Cochrane Database Syst Rev*. 2011;7:CD009232. doi:10.1002/14651858.CD009232.
55. Waters BL, Raisler J. Ice massage for the reduction of labor pain. *J Midwifery Womens Health*. 2003;48:317-321.
56. Derry S, Straube S, Moore RA, Hancock H, Collins SL. Intracutaneous or subcutaneous sterile water injection compared with blinded controls for pain management in labour. *Cochrane Database Syst Rev*. 2012;1:CD009107. doi:10.1002/14651858.CD009107.pub2.
57. Hutton EK, Kasperink M, Rutten M, Reitsman A, Wainman B. Sterile water injection for labor pain: a systematic review and meta-analysis of randomized controlled trials. *BJOG*. 2009;116(9):1158-1166.
58. Carlson JM, Diehl JA, Sachtleben-Murray M, McRae, M, Fenwich L, Friedman A. Maternal position during parturition in normal labor. *Obstet Gynecol*. 1986;68:443-447.
59. Fenwick L, Simkin P. Maternal positioning to prevent or alleviate dystocia in labor. *Clin Obstet Gynecol*. 1987;30(1):83-89
60. Atwood RJ. Parturitional posture and related birth behavior. *Acta Obstet Gynecol Scand*. 1976;55:3-25.
61. Lawrence A, Lewis L, Hofmeyr GJ, Styles C. Maternal positions and mobility during first stage labour. *Cochrane Database Syst Rev*. 2013;10:CD003934. doi:10.1002/14651858.CD003934.pub4.
62. Gupta JK, Sood A, Hofmeyr GJ, Vogel JP. Position in the second stage of labour for women without epidural anaesthesia.

Cochrane Database Syst Rev. 2017;5:CD002006. doi:10.1002/14651858.CD002006.pub4.
63. Kibuka M, Thornton JG. Position in the second stage of labour for women with epidural anaesthesia. *Cochrane Database Syst Rev*. 2017;2:CD008070. doi:10.1002/14651858.CD008070.pub3.
64. Makvandi S, Latifnejad Roudsari R, Sadeghi R, Karimi L. Effect of birth ball on labor pain relief: a systematic review and meta-analysis. *J Obstet Gynaecol Res*. 2015;41(11);1679-1686.
65. Roth C, Dent SA, Parfitt SE, Hering SL, Bay RC. Randomized controlled trial of use of the peanut ball during labor. *MCN Am J Matern Child Nurs*. 2016;41(3):140-146.
66. Tussey CM, Botsios E, Gerkin R, Kelly L, Gamez J, Mensik J. Reducing length of labor and cesarean surgery rate using a peanut ball for women laboring with an epidural. *J Perinat Educ*. 2015;24(1):16-24.
67. Simkin P, Ancheta R, Hanson L. *The Labor Progress Handbook: Early Interventions to Prevent and Treat Dystocia*. 3rd ed. Hoboken, NJ: Wiley-Blackwell; 2011.
68. Smith CA, Levett KM, Collins CT, Jones L. Massage, reflexology and other manual methods for pain management in labour. *Cochrane Database Syst Rev*. 2012;2:CD009290. doi:10.1002/14651858.CD009290.pub2.
69. Unalmis Erdogan S, Yanikkerem E, Goker A. Effects of low back massage on perceived birth pain and satisfaction. *Complement Ther Clin Pract*. 2017;28:169-175.
70. Alexander B, Turnbull D, Cyna A. The effect of pregnancy on hypnotizability. *Am J Clin Hypn*. 2009;52(1):13-22.
71. Werner A, Uldbjerg N, Zachariae R, Wu CS, Nohr E. Antenatal hypnosis training and childbirth experience: a randomized controlled trial. *Birth*. 2013;40(4):272-280.
72. Madden K, Middleton P, Cyna AM, Matthewson M, Jones L. Hypnosis for pain management during labour and childbirth. *Cochrane Database Syst Rev*. 2012;11:CD009356. doi:10.1002/14651858.CD009356.pub2.
73. Landold AD, Millin LS. The efficacy of hypnosis as an intervention for labor and delivery pain: a comprehensive methodological review. *Clin Psychol Rev*. 2011;31:1022-1031.
74. Dowswell T, Bedwell C, Lavender T, Neilson JP. Transcutaneous electrical nerve stimulation (TENS) for pain relief in labour. *Cochrane Database Syst Rev*. 2009;2:CD007214. doi:10.1002/14651858.CD007214.pub2.
75. Taavoni S, Abdolahian S, Haghani H. Effect of sacrum–perineum heat therapy on active phase labor pain and client satisfaction: a randomized, controlled trial study. *Pain Med*. 2013;14(9):1301-1306.
76. Shirvani MA, Ganji Z. The influence of cold pack on labour pain relief and birth outcomes: a randomized controlled trial. *J Clin Nurs*. 2014;23(17-18):2473-2479.
77. Hsieh C, Kong J, Kirsch I, Edwards R, Jensen K, Kaptchuk T, Gollub R. Well-loved music robustly relieves pain: a randomized controlled trial. *PLoS One*. 2014;11(9):e107390.
78. Phumdoung S, Good M. Music reduces sensation and distress of labor pain. *Pain Manage Nurs*. 2003;4:54.
79. Dehcheshmeh FS, Rafiei H. Complementary and alternative therapies to relieve labor pain: a comparative study between music therapy and Hoku point ice massage. *Complement Ther Clin Pract*. 2015;21(4):229-232. Available at: http://www.ncbi.nlm.nih.gov/pubmed/26573448. Accessed December 15, 2016.
80. Smith CA, Collins CT, Cyna AM, Crowther CA. Complementary and alternative therapies for pain management in labour. *Cochrane Database Syst Rev*. 2006;4:CD003521. doi:10.1002/14651858.CD003521.pub2.
81. Smith CA, Collins CT, Crowther CA. Aromatherapy for pain management in labour. *Cochrane Database Syst Rev*. 2011;7:CD009215. doi:10.1002/14651858.CD009215.
82. Lakhan S, Sheafer H, Tepper D. The effectiveness of aromatherapy in reducing pain: a systematic review and meta-analysis. *Pain Res Treat*. 2016;1-13. http://dx.doi.org/10.1155/2016/8158693
83. Anderson D. A review of systemic opioids commonly used for labor pain relief [erratum, *J Midwifery Womens Health*. 2011;56(4):411-418]. *J Midwifery Womens Health*. 2011;56(3):222-239.
84. Phillips SN, Fernando R, Girard T. Parenteral opioid analgesia: does it still have a role? *Best Pract Res Clin Anaesthesiol*. 2017;31(1):3-14.
85. Miyakoshi K, Tanaka M, Morisaki H, et al. Perinatal outcomes: intravenous patient-controlled fentanyl versus no analgesia in labor. *J Obstet Gynaecol* Res 2013;39:783-789.
86. Markley JC, Rollins MD. Non-neuraxial labor analgesia: options. *Clin Obstet Gynecol*. 2017;60(2):350-364.
87. Bricker L, Lavender T. Parenteral opioids for labor pain: a systematic review. *Am J Obstet Gynecol*. 2002;186:S94-S109.
88. Ullman R, Smith LA, Burns E, Mori R, Dowswell T. Parenteral opioids for maternal pain relief in labour. *Cochrane Database Syst Rev*. 2010;(9):CD007396. doi:10.1002/14651858.CD007396.pub2.
89. Van de Velde M. Remifentanil patient-controlled intravenous analgesia for labor pain relief: is it really an option to consider *Anesth Analg*. 2017;124(4):1029-1031.
90. Mattingly JE, D'Alessio J, Ramanathan J. Effects of obstetric analgesics and anesthetics on the neonate: a review. *Paediatr Drugs*. 2003;5(9):615-627.
91. Guinsburg R, Wyckoff MH. Naloxone during neonatal resuscitation: acknowledging the unknown. *Clin Perinatol*. 2006;22:121-132.
92. Rooks JP. Safety and risks of nitrous oxide labor analgesia: a review. *J Midwifery Womens Health*. 2011;56:557-565.
93. Rosen MA. Nitrous oxide for relief of labor pain: a systematic review. *Am J Obstet Gynecol*. 2002;186:S110-S126.

94. Klomp T, van Poppel M, Jones L, Lazet J, Di Nisio M, Lagro-Janssen AL. Inhaled analgesia for pain management in labour. *Cochrane Database Syst Rev*. 2012; 9:CD009351. doi:10.1002/14651858.CD009351.pub2.
95. Likis FE, Andrews JC, Collins MR, et al. Nitrous oxide for the management of labor pain: a systematic review. *Anesth Analg*. 2014;118(1):153-167.
96. Collins MR, Starr SA, Bishop JT, Baysinger CL. Nitrous oxide for labor analgesia: expanding analgesic options for women in the United States. *Rev Obstet Gynecol* 2012; 5:e126-e131.
97. Migliaccio L, Lawton R, Leeman L, Holbrook A. Initiating intrapartum nitrous oxide in an academic hospital: consideration and challenges. *J Midwifery Womens Health*. 2017;62(3):358-362.
98. Richardson MG, Lopez BM, Baysinger CL, Shotwell MS, Chestnut DH. Nitrous oxide during labor: maternal satisfaction does not depend exclusively on analgesic effectiveness. *Anesth Analg*. 2017;124(2):548-553.
99. Sutton CD, Butwick AJ, Riley ET, Carvalho B. Nitrous oxide for labor analgesia: utilization and predictors of conversion to neuraxial analgesia. *J Clin Anesth*. 2017;40:40-45.
100. American College of Nurse-Midwives. Nitrous oxide for labor analgesia. *J Midwifery Womens Health*. 2010;55:292-296.
101. Mardirosoff C, Dumont L, Boulvain M, Tramèr MR. Fetal bradycardia due to intrathecal opioids for labour analgesia: a systematic review. *BJOG*. 2002;109:274-281.
102. Gehling M, Tryba M. Risk and side effects of intrathecal morphine combined with spinal anaesthesia: A meta-analysis. *Anaesthesia*. 2009;64(6):643-651.
103. Schrock SD, Harraway-Smith C. Labor analgesia. *Am Fam Physician*. 2012;85(5):447-454.
104. Lyons GR, Kocarev MG, Wilson RC, Columb MO. A comparison of minimum local anesthetic volumes and doses of epidural bupivacaine (0.125% w/v and 0.25% w/v) for analgesia in labor. *Anesth Analg*. 2007;104:412-415.
105. American College of Obstetricians and Gynecologists. Practice Bulletin No. 177: obstetric analgesia and anesthesia. *Obstet Gynecol*. 2017;129:373-389.
106. Leighton BL, Halpern SH. The effects of epidural analgesia on labor, maternal, and neonatal outcomes: a systematic review. *Am J Obstet Gynecol*. 2002;186:S69-S77.
107. Simmons SW, Taghizadeh N, Dennis AT, Hughes D, Cyna AM. Combined spinal–epidural versus epidural analgesia in labour. *Cochrane Database Syst Rev*. 2012;10:CD003401. doi:10.1002/14651858.CD003401.pub3.
108. Anim-Somuah M, Smyth R, Jones L. Epidural vs non-epidural or no analgesia in labor. *Cochrane Database Syst Rev*. 2011;12:CD000331.
109. Goetzl L. Epidural analgesia and maternal fever: a clinical and research update. *Curr Opin Anaesthesiol*. 2012;25(3):292-299.
110. Shatken S, Greenough K, McPherson C. Epidural fever and its implications for mothers and neonates: taking the heat. *J Midwifery Womens Health*. 2012;57(1):82-85.
111. Osborne C, Ecker JL, Gauvreau K, Davidson KM, Lieberman E. Maternal temperature elevation and occiput posterior position at birth among low-risk women receiving epidural analgesia. *J Midwifery Womens Health*. 2011;56(5):446-451.
112. Lieberman E, O'Donoghue C. Unintended effects of epidural analgesia during labor: a systematic review. *Am J Obstet Gynecol*. 2002;186(5 pt. 2):S31-S68.
113. Zhang J, Landy HJ, Branch DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcomes. *Obstet Gynecol* 2010;116:1281-1287.
114. Wang TT, Sun S, Huang SQ. Effects of epidural labor analgesia with low concentrations of local anesthetics on obstetric outcomes: a systematic review and meta-analysis of randomized controlled trials. *Anesth Analg*. 2017;124:1571-1580.
115. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK. Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomized controlled trial. *Lancet*. 2001;7;358(9275):19-23.
116. Anderson D. Pudendal nerve block for vaginal birth. *J Midwifery Womens Health*. 2014;59(6):651-659.
117. Pagès H, de la Gastine B, Quedru-Aboane J, Guillemin MG, Lelong-Boulouard V, Guillois B. Lidocaine intoxication in newborn following maternal pudendal anesthesia: report of three cases. *J Gynecol Obstet Biol Reprod*. 2008;37(4):415-418.
118. Kinsella SM, Winton AL, Muschambi MC, et al. Failed tracheal intubation during obstetric general anesthesia: a literature review. *Int J Obstet Anesth*. 2015;24:356-374.

APPENDIX

27A

Injection of Intradermal Sterile Water Papules for Relief of Low Back Pain in Labor

TEKOA L. KING

The editors acknowledge Saraswathi Vedam and Carolyn L. Gegor for contributions to this appendix in the previous edition.

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Approximately 30% of women experience severe continuous low back pain in labor.[1] Back pain in labor, especially when caused by an occiput posterior position, is one of the most challenging conditions for the midwife to treat effectively. The tension caused by this pain has been associated with prolonged labor, increased need for pain medication, and prevention of the spontaneous rotation of the vertex to an anterior position. Several techniques have been reported to be helpful, including massage, counterpressure, applied heat or ice, transcutaneous electrical nerve stimulation (TENS), and hydrotherapy. However, most of these measures offer only partial relief with constant application by another person.

Several randomized controlled trials (RCTs)[1-6] and two meta-analyses[7,8] have shown that intradermal sterile water papule injections have greater analgesic effects than massage, TENS, or counterpressure. The pain relief has been shown to last approximately 1 to 3 hours in 85% to 90% of women,[2–6] and intradermal injections appear to reduce pain by approximately 60%.[7,8] This technique offers the advantages of needing minimal, readily available equipment; moreover, it is adaptable to any birth setting and simple to administer. There are no systemic maternal or fetal side effects. Laboring women are able to ambulate and move freely. There is no need for additional fetal heart rate monitoring or other supplemental interventions. The main disadvantage is the sensation of intense burning or stinging at time of application—an effect that lasts 30 to 90 seconds. Uneven or inadequate relief of pain is also an undesirable side effect.[7-9]

The mechanism of action seems to be primarily counter-irritation, which is thought to inhibit

multireceptive neurons to the brain.[10] The gate control mechanism of pain control may also be involved in the effectiveness of this procedure.[7,11,12]

The onset of relief occurs within 2 minutes of injection and usually lasts 2 to 3 hours. The technique may be repeated up to three times, but some women will decline reapplication because of the initial burning. Some women will experience no relief or only partial relief. The exact location of the injected papules can influence the effectiveness of this technique. Hence, prior to injecting the sterile water, the midwife assesses both the fetal position and the woman's preferred focal points for pressure.

Method to Inject Sterile Water Papules

1. Prior to beginning the procedure:
   a. Prepare a 1-cc syringe with 0.6 mL of sterile water (do not use normal saline) and a 25-gauge needle. Use two syringes if two midwives will do the injections simultaneously.
   b. Gather alcohol swabs, pen(s) for marking the skin, and sterile gloves to avoid introduction of pathogens and to protect the provider from exposure to the woman's blood.
2. Explain the procedure to the woman and her support persons.
   a. Benefits include relief of back pain, lack of systemic maternal or fetal side effects, and continued ability to ambulate or move as desired.
   b. Risks include intense burning pain at the injection sites for 30 to 90 seconds and less effective pain relief than desired.
   c. Relief occurs by 2 minutes of completion of injections. Prepare the woman and her support persons with methods of maintaining a stable position and coping with discomfort during the procedure.
3. Place the woman in a sitting, kneeling, or standing position, stabilizing herself with her arms and hands as she leans forward slightly. The injection sites in the sacral area will be more visible when the woman leans forward and flattens her back.
4. Expose the woman's lower back enough to visualize the sacral dimples, which correspond to the dorsal sacral foramina. Place and press the pads of the thumbs on the superior iliac spines. Mark these landmarks with a pen. Place the index finger at these marks and move the thumbs about 3 to 4 cm down and 1 to 2 cm inward into the sacral dimples so that the fingertips touch the corners of a trapezoid (**Figure 27A-1**); mark these points as well.[12] Put pressure on these four points, moving them very slightly laterally and vertically until the woman indicates the points of greatest relief.[8]

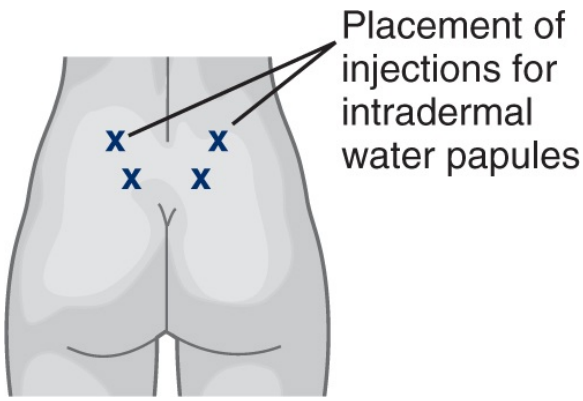


Figure 27A-1 Injection sites for sterile water papules.

5. To mark the points of greatest relief most accurately, ask an assistant to place an X or draw a half-circle around the provider's fingertips while they remain in place.
6. Clean the sites with an alcohol swab, taking care not to erase the circular marks around the area to be injected. Put on sterile gloves. Remind the woman that she will feel temporary burning and stinging as the papules are placed.
7. Plan to do the injections quickly during a contraction unless the woman prefers having the injections done between contractions.[7]
   a. Inject 0.1 to 0.15 mL intradermally in each of the four points delineated (within the half-circle mark) until the distended skin forms a blanched 1-cm bleb or papule.
   b. If possible, have another clinician inject two papules simultaneously to reduce the burning episodes. Do not touch or rub the injection sites when complete.[7]
8. Onset of relief should occur within 2 minutes.
9. If the relief is uneven, the area of less analgesia is often consistent with a bleb that is noted to absorb too quickly, is misplaced, or was injected too deeply. Replacing the papule slightly laterally or vertically often achieves the desired effect.
10. Reevaluate the areas with finger pressure as initially applied, and then repeat the injection.
11. Once the effects have worn off (in 1 to 3 hours, depending on the woman and the fetal position and station), the papules may be replaced with the woman's consent. More than three applications may not be advisable.
12. Document the woman's consent for the procedure. Also document the time of injections and patient pain scores both before and after the procedure.

References

1. Bahasadri S, Ahmadi-Abhari S, Dehghani-Nik M, Habibi GR. Subcutaneous sterile water injection for labour pain: a randomised controlled trial. *Aust N Z J Obstet Gynaecol.* 2006;46(2):102-106.
2. Trolle B, Moller M, Kronborg H, Thomsen S. The effect of sterile water block on low back labor pain. *Am J Obstet Gynecol.* 1991;5(1):1277-1281.
3. Ader L, Hansson B, Wallin G. Parturition pain treated by intracutaneous injections of sterile water. *Pain.* 1990;41(2):133-138.
4. Kushtagi P, Bhanu BT. Effectiveness of subcutaneous injection of sterile water to the lower back for pain relief in labor. *Acta Obstet Gynecol Scand.* 2009;88(2): 231-233.

5. Reynolds JL. Intracutaneous sterile water for back pain in labour. *Can Fam Physician*. 1994;40:1785-1788, 1791-1792.
6. Martenson L, Wallin G. Sterile water: labor pain treated with cutaneous injections of sterile water: a randomized controlled trial. *Br J Obstet Gynaecol*. 1999;106:633-637.
7. Fogarty V. Intradermal sterile water injections for the relief of low back pain in labour: a systematic review of the literature. *Women Birth*. 2008;21;157-163.
8. Mårtensson L, McSwiggin M, Mercer JS. US midwives' knowledge and use of sterile water injections for labor pain. *J Midwifery Womens Health*. 2008;53:115-122.
9. Jaynes AC, Scott KE. Intrapartum care the midwifery way: a review. *Prim Care*. 2012;39(1):189-206.
10. Martensson L, Wallin G. Sterile water injections as treatment for low-back pain during labour: a review. *Aust N Z J Obstet Gynecol*. 2008;48(4):369-374.
11. Huntley AL, Coon JT, Ernst E. Complementary and alternative medicine for labor pain: a systematic review. *Am J Obstet Gynecol*. 2004;191(1):36-44.
12. Simkin P, Bolding A. Update on nonpharmacologic approaches to relieve labor pain and prevent suffering. *J Midwifery Womens Health*. 2004;49:489-505.

CHAPTER

28

Second Stage of Labor and Birth

LISA KANE LOW AND KATHRYN OSBORNE

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Introduction

Midwives create a circle of support and safety during labor and birth that encourages, rather than disrupts, the normal processes that facilitate healthy outcomes for families. In this capacity, the role of the midwife has best been described as watchful waiting or "the art of doing 'nothing' well."[1] The midwife anticipates needs and provides vigilance in the assessments conducted for women and their support persons during labor and birth, while remaining cognizant of the wide range of what constitutes normal and where the boundaries of abnormal begin. All persons involved participate in shared decision making, with the midwife being responsive, rather than reactive, and supportive, rather than directive. This chapter reviews midwifery management during the second stage of labor and through the event of birth.

Definition of Second-Stage Labor

Anatomically, second-stage labor is defined as beginning with complete dilation of the cervix (10 cm) and ending with expulsion of the fetus. However, the clinical and physiologic demarcation between the final dynamics of first-stage labor and the beginning of second-stage labor is less distinct. Subtle changes indicative of the second stage may appear near the end of the active phase of the first stage of labor, or there may be a lull in the frequency and intensity of contractions at the end of first-stage labor prior to the onset of active second-stage labor.[2] Although the anatomic definition is the one most commonly used in clinical practice, from a physiologic perspective second-stage labor may be defined as the onset of the urge to bear down until the birth of the infant. Using this definition, second-stage labor is initiated when the vertex or presenting part of the fetus has descended to a station wherein an involuntary maternal urge to bear down occurs. The physiologic urge to push typically occurs when the fetal presenting part reaches +1 station.

Normal Course of the Second Stage of Labor

Observations of women in labor without epidural analgesia who are not coached to push, regardless of whether they have an urge to do so, have revealed that most women experience two or three distinct phases of the second stage of labor.[2,3] If the fetus is above 0 station when the cervix becomes completely dilated, the first phase may consist of a short lull or pause during which uterine contractions are less frequent and there is no urge to push. This is followed by an expulsive phase or phase of active pushing, during which the urge to push and bearing-down efforts become progressively stronger to accomplish the necessary fetal rotation and descent. The final perineal phase occurs when the fetal head is visible at the introitus. During this last phase, a strong sense of pressure combined with a sensation of stretching often results in pushing efforts even without contractions.[2]

Signs and Symptoms of Second-Stage Labor

The most common symptom that heralds the onset of the second stage is an urge to push or bear down. Women who are not using epidural analgesia usually have an irresistible need to push or bear down, but this is not universally true.[4] Many women interpret this feeling as needing to have a bowel movement. Signs of second-stage labor also include finding the fetal heart rate (FHR) in progressively lower locations on the maternal abdomen, rectal bulging, perineal bulging, and progressive visibility of the fetal head at the vaginal introitus. In addition, there may be an increase in bloody show, and the FHR pattern may demonstrate variable decelerations secondary to the vertex descending with maternal bearing-down efforts. Interestingly, an almost infallible signal of imminent birth is the woman saying, “The baby’s coming!”—a verbal expression that occurs in most cultures and languages.

Fetal Descent During Second-Stage Labor

During the second stage of labor, the fetus descends. Internal rotation often takes place during the late first stage or in the second stage. Birth of the head occurs first with flexion of the fetal neck, followed by extension and external rotation. This process can occur very quickly, especially for women who have given birth before, or it can occur over a period of time, which is more common when a woman is experiencing her first birth. A detailed description of the cardinal movements that describe fetal passage through the birth canal during labor and birth is found in the *Anatomy and Physiology During Labor and Birth* chapter.

Duration of Second-Stage Labor

For several decades, the norms used to define duration of second-stage labor were based on investigations conducted in the 1950s and early 1960s by Emanuel A. Friedman.[3] During that era, labor management strategies were quite different from contemporary practice: Approximately 55% of the women in Friedman’s observations had their labors terminated by application of low or mid-forceps. As a result, Friedman’s analysis of second-stage duration

was more an observation of practice patterns at the time than an investigation of the natural course of the second stage. According to Freidman, the average length of the second stage was 46 minutes for a woman having a first vaginal birth and 14 minutes for women having subsequent births.[3]

The parameters used to define the duration of second-stage labor have changed, in part due to the widespread use of epidural analgesia for pain management and the effect of epidural analgesia on maternal bearing-down efforts. Women who enter the second stage of labor with effective epidural analgesia have diminished or absent pelvic and rectal sensation and, therefore, experience lessened or no urge to push, often resulting in a second stage that is longer than the parameters established by Friedman. Studies examining the maternal and fetal effects of a longer second stage and delayed pushing for women with epidural analgesia began to appear in the literature more than 30 years ago. Recent retrospective and prospective study results suggest that the normal duration of second stage labor is at least 1 to 2 hours longer than the duration described by Friedman.[5-7] The findings of research examining the relationship between second-stage labor duration and perinatal outcomes have been conflicting, and are reviewed later in this chapter.

The factors that influence the duration of the second stage of labor are listed in **Table 28-1**. For women who have no medical or obstetric complications at the onset of labor, the three factors that have the strongest effect on the duration of the second stage are parity, fetal position, and the presence or absence of regional analgesia.

Table 28-1	Factors That Influence the Duration of the Second Stage of Labor
Parity	
Fetal position	
Epidural analgesia	
Fetal station at complete cervical dilation	
First-stage labor dystocia	
Uterine contraction activity: strength and frequency of contractions	
Presence or absence of infection	
Effectiveness of maternal pushing efforts	
Pelvic architecture	
Birth weight	
Maternal weight or high body mass index	
Induction of labor	
Maternal fear or anxiety	
Maternal pain or discomfort	
Maternal history of sexual abuse or trauma	

Second-stage labor abnormalities have traditionally been categorized as prolonged duration or abnormalities in the process of descent and/or rotation. Abnormalities in the process of descent can be further subdivided into protracted descent, failure of descent, or arrest of descent.

Prolonged Duration of Second-Stage Labor

There are three clinically significant questions related to prolonged duration of the second stage: (1) What is the time frame during which the woman's chance of successful vaginal birth declines? (2) What are the maternal outcomes? (3) What are the fetal outcomes?

Using Friedman's criteria, prolonged second-stage labor has been defined as second-stage labor lasting longer than 2 hours for a primigravida and longer than 1 hour for a multigravida.[3] In 1989, Kilpatrick and Laros published the first analysis of the duration of labor in women (*n* = 6991), both with and without epidural anesthesia, who had a spontaneous vaginal birth at term without the use of oxytocin.[8] They concluded that longer durations should be used to define prolonged second-stage labor when an epidural is in place. Their work became the "1-2-3 Rule," which established the limits of normal for second-stage labor as 1 hour for multiparous women without an epidural, 2 hours for multiparous women with an epidural and for nulliparous women without an epidural, and 3 hours for nulliparous women with an epidural.[8] The Kilpatrick and Laros study was conducted prior to the introduction of "laboring down"—that is, the use of passive descent (without maternal bearing-down efforts) as a strategy for second-stage labor management in the presence of an epidural. Later studies have provided evidence to support even longer durations of the second stage, particularly when passive descent has been used as a management strategy.[9-11]

In a secondary analysis of a randomized controlled trial (RCT) in which 94% of the participants had an epidural, Rouse et al. found that 85% of women who pushed for less than 1 hour had a spontaneous vaginal birth. Among women who pushed between 1 and 2 hours, 78.7% had a spontaneous vaginal birth; among women who pushed between 2 and 3 hours, the rate was 59.1%; and among women who pushed between 3 and 4 hours, the rate was 24.7%. When women pushed 5 hours or more, the vaginal birth rate was 8.7%.[12] Thus, it appears there is a positive correlation between duration of the second stage and spontaneous vaginal birth rates. Furthermore, there is also a second-stage labor duration at which the chance of vaginal birth diminishes significantly. The authors concluded that duration of second-stage labor could be safely extended for some women, but that decisions to terminate the second stage should not be made based on duration alone.[12]

To date, no prospective randomized trial has been conducted to address the question of "how long is too long" for the duration of second-stage labor; retrospective cohort studies and secondary analyses have been conducted that support longer durations.[13] The debate is ongoing in the literature, with some investigators advocating caution in regard to extending the duration beyond 2 hours for multiparous women and 3 hours for nulliparous women because of concerns about increased maternal and neonatal morbidity.[14] Other investigators have identified that the parameters for second-stage labor duration can be safely extended without an increase in adverse maternal or fetal outcomes and recommend a reexamination of the definition of prolonged second-stage labor.[11]

Maternal Complications Associated with Prolonged Second-Stage Labor

A prolonged duration of second-stage labor is associated with an increased risk of maternal

morbidity. Specifically, actively pushing for more than 2 to 3 hours has been associated with an increased risk for intra-amniotic infection postpartum hemorrhage, operative birth, and third- and fourth-degree lacerations.[12,14,15] Prolonged pushing may also contribute to long-term adverse outcomes in the pelvic floor such as levator ani muscle tears, which are associated with pelvic floor disorders later in life.[16-19] In multiparous women, those with a prolonged second stage had a higher risk of shoulder dystocia. It is not clear if prolonged duration of the second stage results in intra-amniotic infection or if intra-amniotic infection adversely affects uterine contractility sufficiently to cause a prolonged duration. Intra-amniotic infection may be both a cause and effect of prolonged labor.

Fetal Complications Associated with Prolonged Second-Stage Labor

Most of the research on second-stage labor has not found a significant relationship between second-stage duration and perinatal mortality, 5-minute Apgar scores of less than 7, neonatal seizures, or admission to the neonatal intensive care unit (NICU) as long as the FHR pattern does not indicate an increasing risk for fetal acidemia.[14,15,19] A large epidemiologic analysis of women who gave birth between 2002 and 2008 (n = 59,605) shed more light on these relationships. Using guidelines that defined prolonged second-stage labor as longer than 3 hours for nulliparous women with an epidural, longer than 2 hours for nulliparous women without an epidural and multiparous women with an epidural, and longer than 1 hour for multiparous women without an epidural, Laughon et al. found a positive association between a prolonged second stage and neonatal morbidity with regard to several outcome measures.[14] In particular, prolonged second-stage labor was associated with a significant increased risk for 5-minute Apgar score less than 4, neonatal sepsis, admission to the NICU, and perinatal mortality. The findings were stratified by parity and epidural use. The incidence of neonatal sepsis was 2.6% versus 1.2% (adjusted odds ratio [aOR], 20.08; 95% confidence interval [CI], 1.60–2.70) and incidence of 5-minute Apgar scores less than 4 was 0.5% versus 0.2% (aOR, 2.71; 95% CI, 1.20–1.60) in nulliparous women with an epidural who had a prolonged second stage versus those in the same cohort who did not have a prolonged second stage. Nulliparous women without an epidural and multiparous women with and without an epidural had similar findings with regard to neonatal outcomes, although adverse newborn outcomes were less common in multiparous women. Although these are statistically significant increased odds, the absolute risk of adverse neonatal outcomes was quite low for all the newborn outcomes. Nonetheless, as second-stage labor progresses beyond the recommended guidelines, ongoing fetal assessment should be part of the consideration in balancing the risks versus the benefits of a spontaneous vaginal birth.[14]

In summary, the criteria for a diagnosis of prolonged second-stage labor has evolved from the strict limitations described by Freidman to the latest recommendations listed in **Table 28-2**,[7] which offers greater latitude in evaluation of the ongoing progress of rotation and descent and identification of prolonged second-stage labor. In brief, in the absence of maternal exhaustion, other maternal complications, or signs that the fetus is developing acidemia, there are no clear reasons for rigidly adhering to any preset time limit for duration of second-stage labor. Instead, emphasis should be placed on evidence of progress in rotation and descent of

the presenting part in response to maternal pushing efforts, maternal stamina, and fetal well being.

Table 28-2 Definition of Prolonged Second-Stage Labor

No progress in descent or rotation for:

- ≥ 4 hours in nulliparous women with an epidural
- ≥ 3 hours in nulliparous women without an epidural
- ≥ 3 hours in multiparous women with an epidural
- ≥ 2 hours in multiparous women without an epidural

Based on Spong CY, Berghella V, Wenstrom KD, Mercer BM, Saade GR. Preventing the first cesarean delivery: summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal–Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. *Obstet Gynecol.* 2012;120(5):1181-1193.[7]

Ongoing assessment during second-stage labor includes continuous evaluation and documentation of fetal descent, rotation, other aspects of fetal position such as asynclitism, and the degree to which the fetus and the woman in labor are tolerating the vigorous work of the second stage. Most women will progress through second-stage labor by following their physiologic urges and sensations without the need for intervention. The point in time at which the midwife first consults the physician for suspected (or anticipated) prolonged second-stage labor varies from practice to practice and is influenced by the circumstances under which the midwife works. For example, a midwife practicing in a rural hospital with the physician colleague 25 minutes away would likely need to consult the physician sooner than a midwife whose physician colleague is available in the hospital at all times. The prudent midwife–physician team has preestablished parameters, relative to prolonged second stage and consultation, that meet the unique needs of the women served by that practice.

In general, the midwife stays with the woman and continues to offer support when physician consultation becomes necessary. During the consultation, the midwife and the physician establish a plan of care that often involves co-management, such that the midwife provides ongoing care and assessment of the woman, and gives periodic updates to the physician. Critical elements of a second-stage labor consultation note are presented in **Table 28-3**.

Table 28-3 Critical Elements of a Second-Stage Labor Consultation Note

Element	Description
Subjective Data	Describe what the woman says about second-stage labor. This description generally includes comments about how she is tolerating pushing and the sensations she feels.
Objective Data	Describe status of the labor (frequency, duration, and intensity of contractions; time of complete cervical dilation; time that the woman began active pushing); status of the fetus (fetal heart tones, particularly the presence or absence of any changes in baseline or decelerations); fetal position (station and orientation of the fetal head); and maternal status (maternal vital signs and other objective findings).
Assessment	Describe the midwife's assessment of maternal and fetal status and progress (descent and rotation)

	made during second-stage labor; document total time in second-stage labor and total time actively pushing.
Plan	Document the time of the physician consultation and the plan established—specifically, the plan for continued pushing or a period of rest and the specific plan for reevaluation (who will conduct the assessment and when).

Descent Abnormalities in Second-Stage Labor

Descent abnormalities in second-stage labor can occur secondary to malpositions such as persistent occiput posterior or asynclitism, uterine hypocontractility, cephalopelvic disproportion, or maternal fatigue. Failure of descent is present if the fetal presenting part makes no descent after 1 to 2 hours of adequate contractions and maternal pushing efforts. Relative cephalopelvic disproportion may also manifest as protracted descent and prolonged second stage. Protracted descent is more common than failure of descent or arrest of descent, and malpositions are a common cause of protracted descent. The process of rotation and descent should be regularly assessed to determine whether progress is ongoing or has stopped. Assessment of station, fetal position, and presentation at the onset of second-stage labor is critical in determining the amount of progress that has been made over time, particularly if concerns about duration arise. From a clinical standpoint, it is important not to confuse molding or caput with true descent. Physician consultation should be obtained when second stage progress is protracted.

Protracted Descent and Persistent Occiput Posterior Position

One of the most common reasons for protracted fetal descent is persistent occiput posterior (OP) position. When the fetus remains in a persistent OP position during descent in the second stage of labor, the cardinal movements of labor are slightly different as described in the *Anatomy and Physiology During Labor and Birth* chapter. Flexion of the vertex (fetal chin touching fetal chest) may be incomplete, so that the larger occipitofrontal diameter is the anterior-posterior diameter that descends through the pelvis (**Figure 28-1**). When the fetus is in an occiput anterior (OA) position, the occiput descends and flexes so the chin is against the chest as the occiput moves down the 5-cm anterior–posterior diameter of the posterior inferior portion of the symphysis pubis, and then, once the occiput is out from under the symphysis, the head is born by extension of the neck. In contrast, when the fetus is in an OP position, the occiput descends along the 10-cm distance between the top of the sacrum to the bottom of the coccyx and the final few centimeters along the floor of the birth canal before the fetal head is born via flexion, with the occiput presenting in the lower portion of the introitus, then extension on the fetal head occurring only as the occiput fully exits the vagina.

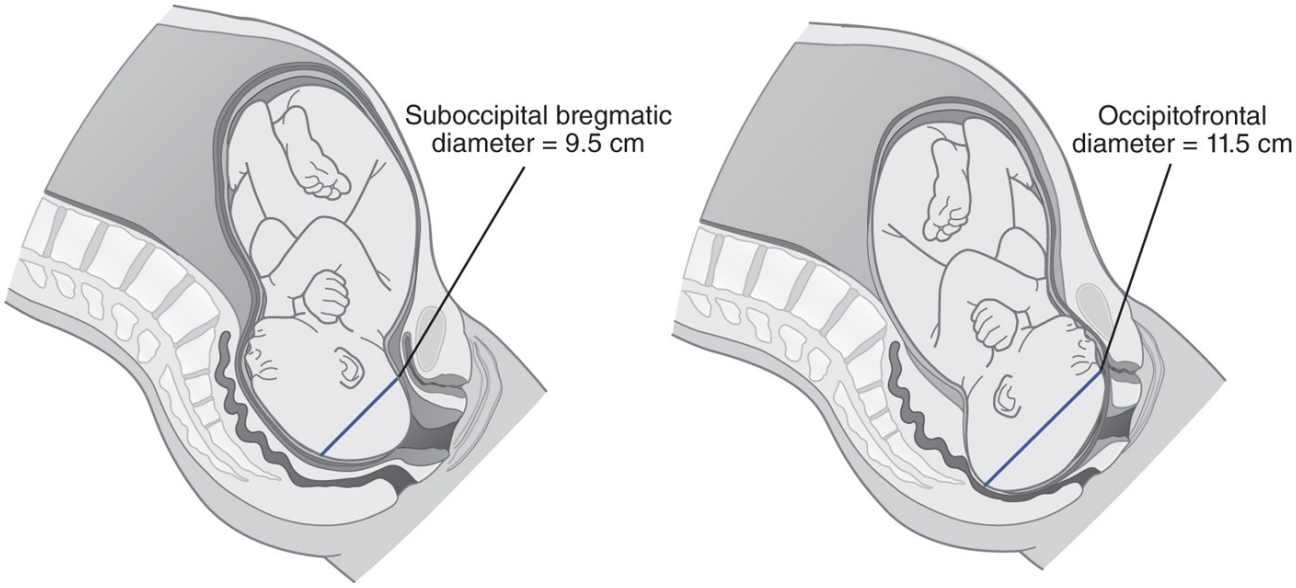


Figure 28-1 Presenting anterior posterior fetal skull diameters in occiput anterior and occiput posterior positions.

Approximately 15% to 25% of fetuses are in an OP position prior to labor, yet only 5% to 7% of fetuses are born in this position.[7,20] Most fetuses in an OP position during the first stage of labor rotate to OA position during the second stage. Anecdotally, it has been reported that women experience more intense back pain when the fetus is in an OP position, although the veracity of this supposition has been neither proven nor disproven.[21]

The occiput posterior position is more likely in women who have an epidural in place. It is not conclusively known if this association occurs secondary to relaxation of pelvic musculature caused by the epidural, or if women with a fetus in an OP position have more painful labors and, therefore, are more likely to choose epidural analgesia. A few studies have examined the relationship between OP position at the time of epidural placement and outcomes.[22,23] Lieberman et al. examined fetal position at the time of epidural placement and position just prior to birth (n = 1562) and found that there were no differences in fetal position at the time of epidural placement, but that more fetuses were in the OP position after the epidural was administered (12.9% with an epidural versus 3.3% with no epidural; P = 0.002).[22] Thus, it appears that epidural analgesia does have an independent effect in increasing the incidence of OP position.

Persistent occiput posterior position is associated with several adverse outcomes, including prolonged first-stage labor, arrest disorders in second-stage labor, a twofold increase in the risk for operative birth, and an increased incidence of perineal lacerations.[7,21,23] Newborn outcomes include higher rates of acidemic umbilical cord blood gases, meconium-stained amniotic fluid, and NICU admission. Some of these outcomes are influenced by the increased incidence of caesarean and operative modes of vaginal birth associated with a persistent OP position.[23]

Diagnosis of an OP position can be made via digital examination or with a combination of transvaginal and transabdominal ultrasound.[21,24] There is a high degree of discordance between digital and ultrasound determinations, however. Although ultrasound is more accurate

than digital examination, approximately 6% to 10% of ultrasounds are not able to determine the fetal position, particularly when the fetal head is deep into the pelvis.[20,21] Diagnostic quandaries aside, the real clinical question is treatment of the OP position once it is diagnosed.

Management of Persistent Occiput Posterior Position

Practices purported to assist internal rotation of an OP fetus to the OA position include having the woman labor on her hands and knees for a period of time, digital rotation with the examiner's fingers, and manual rotation with the examiner's hands. Studies have been conducted that assessed the effect of placing the woman in an asymmetric lateral decubitus position and hands-and-knees positions. Although the number of studies is small and methodologies cannot be compared easily, the asymmetric lateral decubitus position did not increase the incidence of internal position change from OP to OA in one RCT.[25] In another RCT of hands-and-knees positioning during labor, 16% of the OP fetuses rotated to OA position in the hands-and-knees group versus 7% in the control group, although these results were not statistically significant (relative risk [RR], 2.4; 95% CI, 0.88–6.62).[26] In this study, the women who assumed a hands-and-knees position reported less back pain and high satisfaction with laboring in this position. Other maternal maneuvers—for example, lunging, asymmetric kneeling, walking, or side-lying—have not been rigorously studied. Although study findings to date are inconclusive, there is a logical basis in maternal anatomy and physiology for the use of these strategies, and theoretical positive effects on pelvic diameter that may aid the fetus in finding the "best fit."[25] Frequent changes of position such as occur during use of the traditional Mexican rebozo technique may be more useful than any single specific position.[27] It is not known if frequent maternal position changes have an effect on fetal position in the first stage of labor compared to the second stage of labor.

Both digital and manual rotation reduce the incidence of OP birth.[28] The techniques for digital and manual rotation are described in **Table 28-4**. Although there is a theoretical risk of umbilical cord prolapse or trauma to the fetal neck with use of these techniques, these complications appear to be rare and the procedures are considered safe. Manual rotation should not be performed prior to the second stage of labor or if the fetus is at a high station. However, some evidence suggests this technique is more successful if performed prophylactically before arrest of descent occurs.

Table 28-4	Technique for Digital and Manual Rotation of Occiput Posterior Position
For Both Techniques	
Using a process of shared decision making review the risks and benefits of the procedure and obtain informed consent. Make certain the bladder is empty.	
Digital Rotation	**Manual Rotation**
1. Place the tips of the gloved index and middle fingers in the anterior portion of the lambdoidal suture near	1. Place four gloved fingers behind the posterior parietal bones with the palm and fingers resting on the

the posterior fontanelle. 2. Place the thumb of the same hand over the parietal bone. 3. Move the fetal head up toward the pelvic brim slightly and flex it with upward pressure from the two fingers. 4. Rotate the fetal head to the occiput anterior position via the shortest arc during a contraction and when the woman is pushing (i.e., if the fetus was right OP, then rotate to right OA). 5. If the fetal head does not rotate with the above mechanism, rotation of the fetal head can be attempted when the woman is not pushing, again following the short arc.	occipital bone. 2. Place the thumb of the same hand over the anterior parietal bone. 3. Flex and rotate the fetal head anteriorly during a contraction and when the woman is pushing. Mild upward pressure of the fetal head toward the pelvic brim may facilitate rotation.

Abbreviations: OA, occiput anterior; OP, occiput posterior.

Assessment of Maternal and Fetal Well-Being in Second-Stage Labor

Assessment during the second stage of labor includes evaluation of maternal well-being, fetal well-being, and progress of labor, as described in **Table 28-5**.

Table 28-5	Midwifery Assessment During Second-Stage Labor
Maternal vital signs and well-being	Blood pressure: Taken between contractions every 15 minutes, unless more frequent evaluation is indicated. Temperature, pulse, and respirations: Check hourly, unless more frequent evaluations are indicated. Maternal pulse normally increases during contractions. Assess bladder for distension periodically during the second stage. Intermittent catheterization may be needed if the woman is unable to void spontaneously. If an indwelling catheter is in place at the onset of the second stage, it should be removed when the woman is pushing to minimize trauma to the urethra when the fetal head compresses it. Assess hydration and nutrition. Dehydration can contribute to ineffectual uterine activity. Frequent intake of small amounts of oral fluids is recommended. Assess pain, comfort, and coping. Does there appear to be any fear or anxiety about giving birth? Are the comfort measures being used effective? Evaluate coping versus suffering behaviors. Assess level of fatigue. Maternal pushing efforts will become less effective if the woman becomes too fatigued after pushing for a prolonged period of time.
Fetal well-being[a]	Assess fetal heart rate every 5–15 minutes. Increase frequency as the presenting part descends, unless more frequent evaluation is indicated. Document findings in the medical record.
Labor progress	Assess contraction pattern (frequency, duration, intensity). Assess progress with a sterile digital cervical examination when the findings will inform a change in management. Assess each of the following: Fetal position Fetal station Fetal descent or rotation in response to maternal pushing effort Fetal adaptation to the pelvis including: Synclitism or asynclitism Molding or caput succedaneum Rotation, malposition

[a] A detailed discussion of fetal assessment can be found in the *Fetal Assessment During Labor* chapter.

Vital Signs

Maternal blood pressure is taken between contractions and is, on average, 10 mm Hg higher in the second stage than values obtained during the first stage of labor if the woman has been pushing. Similarly, the maternal pulse increases during contractions.

Bladder

The use of epidural analgesia results in reduced ability to void and leads to increased risk for urinary retention and distension. In women who have had an epidural, the bladder often contains larger volumes of residual urine compared to women who have not had an epidural; this retention may occur secondary to larger urine volume that follows use of intravenous fluids to maintain systemic blood pressure following the sympathetic block induced by the epidural.[29,30] If an indwelling Foley catheter is in place when the woman starts bearing down, the catheter should be removed to avoid undue stress on the urethra.

If the woman has a distended bladder and is unable to void, intermittent catheterization can be performed. If catheterization must be performed during second-stage labor and the fetal head is in the true pelvis, the direction of catheter insertion is different from usual. The urethra is displaced by the fetal head and conforms to the contour of the fetal head. Therefore, immediately after inserting the catheter into the urethra, it is necessary to direct it upward and over the fetal head to guide the catheter into the bladder. Sometimes it is helpful to splint the urethra by placing a finger into the vagina, below the urethra, as the catheter is being inserted.

Hydration

The second stage of labor requires active physical work; in turn, the risk for maternal dehydration increases. Prolonged fasting in labor is associated with increased production of ketones, which may result in ketosis for the laboring woman. Consequences of limited fluid intake or limited nutritional intake can include a rising maternal pulse rate and limited energy or fatigue. Theoretically, ketosis can cause dysfunctional uterine contractions, although this has not been definitively shown.

In 2016, the American Society of Anesthesiologists revised its position to recommend that women whose labors are progressing normally be offered modest amounts of clear liquids.[31] Clear liquid options include water, fruit juices without pulp, carbonated beverages, clear tea, black coffee, and sports drinks.[31,32] The midwife can encourage oral hydration by offering clear liquids or ice chips in between pushing efforts.[32] If the woman is unable to maintain oral hydration, intravenous fluids may be indicated. The presence of an epidural should not alter the recommendation to maintain oral hydration during labor if the woman desires.[32]

Assessment of Pain and Coping

During the second stage of labor, new physical sensations occur as the woman nears the time of birth. Anticipatory guidance as the second stage progresses can help the woman cope with pushing sensations, perineal stretching, and the potentially uncontrollable urge to bear down.

If a woman is crying out rather than grunting and bearing down, or is unable to sustain any

position of comfort, she may be suffering acute distress instead of actively coping.[33] Some women will experience sudden fear and anxiety with the sensations of vaginal stretching, and pain relief that ameliorates this perineal sensation may be necessary so she can push. A pudendal block can be used if an epidural is not available or desired. Women who have experienced past sexual abuse or assault may be at particular risk for fear or panic that escalates when the presenting part induces new sensation in the pelvic floor or perineum.[34]

Maternal Fatigue

The amount of added energy a woman requires for the process of pushing varies greatly, as does the average duration of second-stage labor. Some women begin the active process of pushing with renewed energy and excitement. The entire energy level in the labor and birth room may increase as the activity and excitement of impending birth grow. Family members present may become engaged as well and can add to the level of encouragement provided to the woman to aid her in overcoming fatigue. Since maternal fatigue can result in a prolonged second stage, it is important to monitor maternal fatigue and to encourage periods of rest between bearing-down efforts.

Evaluation of Fetal Well-Being

The assessment of the FHR or fetal heart tones during the second stage of labor is reviewed in more detail in the *Fetal Assessment During Labor* chapter. Variant FHR patterns are more common in the second stage than in the first stage, especially variable decelerations, prolonged decelerations, and bradycardias. Thus, the FHR is evaluated more frequently in second-stage labor. Current standards recommend assessment every 15 minutes for women without a risk for fetal acidemia and every 5 minutes for women who have an increased risk for acidemia.[35] In practice, the FHR is evaluated more frequently as the second stage progresses and is often checked every two to three contractions in the final phases of the second stage.

Midwifery Management of the Second Stage of Labor

During the second stage, the midwife can offer the woman encouragement and facilitate the efforts of everyone in ways that best help the woman in labor with supportive direction.[36,37] Using this approach, suggestions are offered without an intention to coach her or provide directive instructions. Instead, supportive direction incorporates recommendations to promote effective pushing in context with what has been happening up to that point during second-stage labor. This allows for individualized support of the woman without providing authoritative or directive coaching, neither of which is tailored to the unique needs of the individual woman in labor.

The Family-Centered Birth Environment

The decision of who to include in the birth room is an important one. Home settings and freestanding birth centers generally have few or no restrictions on the number of persons present. Most hospitals allow any number of support persons in the labor room, though restrictions are likely to be placed on the number of persons able to be in the operating room if a cesarean birth is necessary. Hospitals today also often allow siblings to be present during labor and birth; if children will be present, however, the institution usually requires the presence of an adult whose primary responsibility will be care of the child. Children may want or need to leave the birth environment, so an adult who is assigned the role of caring for the child should be prepared to leave the birth room as necessary.

Maternal Pushing Efforts

Several management options may be initiated for helping women push during the second stage of labor, including passive descent, open versus closed glottis technique, and spontaneous versus directed pushing.[6,38] An initial consideration is whether a period of passive descent would be beneficial for the woman who has an epidural or who does not have an urge to push. Once active pushing begins, pushing efforts can be open glottis versus closed glottis (Valsalva).

Delayed Pushing: Passive Descent Versus Immediate Maternal Pushing Efforts

Passive descent as a management strategy was initially introduced for the purpose of reducing the incidence of instrumental birth in women using epidural analgesia. The theory behind its use is as follows: During the process of passive descent (also referred to as laboring down), the fetus descends in response to the force generated from contractions alone and begins to internally rotate once the presenting part reaches the pelvic floor. Encouraging a period of maternal rest prior to engaging in active pushing efforts allows for conservation of the woman's energy until there is a physical stimulus to actively push. Once the woman does engage in active pushing, her efforts will be more effective and efficient in the presence of physical cues from pressure of the presenting part on the pelvic floor.[39]

Several RCTs and two meta-analyses have compared maternal and fetal outcomes in women

who rested for a period of approximately 1 hour prior to pushing with those in women who started pushing as soon as the cervix was found to be fully dilated.[40-46] Most of these studies defined passive descent as no active pushing for 1 to 2 hours unless an uncontrollable urge to push developed or the fetal head was visible at the introitus. In a meta-analysis of seven RCTs, Brancato et al. found delayed pushing was associated with a modest increase in the incidence of spontaneous birth (RR, 1.08; 95% CI, 1.01–1.15; $P = 0.025$), and fewer operative births (RR, 0.77; 95% CI, 0.77–0.85; $P \leq 0.0001$), less maternal exhaustion, and no increase in adverse maternal or neonatal outcomes.[41] In contrast, the systematic review by Tuuli et al. found an increased rate of spontaneous vaginal birth associated with delayed pushing in a meta-analysis of 12 RCTs; however, this increase was smaller and not statistically significant in the RCTs that the researchers assessed to be of high quality.[40]

The findings of a recently conducted secondary analysis of data collected during an observational, cohort study conflict with the findings of previously conducted high-quality RCTs. Yee et al. compared the outcomes of nulliparous women who experienced delayed onset of pushing with the outcomes of nulliparous women who initiated pushing immediately upon entering the second stage.[47] Their findings revealed higher rates of cesarean birth (11.2% versus 5.1%, respectively; aOR, 1.86; 95% CI, 1.63–2.12) and instrumental birth (16.2% versus 11.2%, respectively; OR, 1.26; 95% CI, 1.14–1.40) in the group that used delayed pushing. Furthermore, the women in this analysis had statistically significant higher rates of postpartum hemorrhage and blood transfusion, but no increased neonatal morbidity or mortality.[47]

Most recently, a 2017 systematic review of pushing methods used during second-stage labor identified a longer second stage of labor but shorter duration of pushing and increased rates of spontaneous vaginal birth in women who delayed pushing than in women who pushed immediately.[48] There was no significant increase in third- or fourth-degree lacerations or episiotomies between women who pushed immediately and women who delayed pushing. The investigators found a positive association between delayed pushing and low umbilical cord pH, but no significant difference in NICU admission rates or 5-minute Apgar scores between groups.[45] These authors concluded that there is no conclusive evidence to support or refute any specific method of pushing during second-stage labor for women with or without an epidural, and that decisions about pushing approaches should be made based on the clinical context and the woman's preference and comfort.[44]

Given the conflicting outcomes of the three meta-analyses,[40,41,48] which included some of the same trials, and ongoing questions about safe parameters for length of the second stage, the value and strength of evidence that supports delayed pushing are considered inconclusive. The studies that found passive descent or delayed pushing to be effective noted a modest positive effect of increasing the incidence of vaginal birth in women who have an epidural with a decrease in use of vacuum or forceps of approximately 20%. The findings of the Yee et al. study suggest a relationship may exist between delayed pushing and adverse maternal outcomes in nulliparous women.[47] Thus, given positive effects are small and possible negative effects may occur, until further studies are completed, delayed pushing should be considered on an individual basis for nulliparous women who have an epidural in place. Intra-amniotic

infection, FHR patterns that indicate a risk for fetal acidemia, vaginal bleeding, or other clinical indications for expediting birth are likely contraindications for delayed pushing.

Identification of how long to wait and when to transition to active pushing efforts requires careful consideration. Although no specific guidelines exist at present, it is common practice to offer a period of 1 to 2 hours for passive descent, as these are the time periods used in most of the investigations. Rather than using arbitrary time periods, it may be prudent to encourage active pushing whenever the vertex reaches +1 station, because it is not known if there are long-term adverse effects of delayed pushing on maternal pelvic floor dysfunction. The midwife should assess the effectiveness of pushing efforts soon after they start. If the woman is able to push effectively, the midwife can support her pushing efforts until she gives birth. If her initial efforts do not result in any fetal movement and the fetal station is above 0 station, the midwife may consider allowing a time of rest to encourage passive descent.

Some authors have suggested reducing the concentration of anesthetic in the epidural infusion so that the sensory block is less strong and the woman feels a stronger urge to push. This strategy can be effective if the woman wants to have more sensation, the epidural is able to be titrated, and the woman gives her consent. Nevertheless, totally discontinuing the epidural is not recommended because it results in more pain and no increase in the spontaneous vaginal birth rate.[49,50] Completely removing pain relief from a woman who has used an epidural during labor can result in the sudden onset of intense pain, which can be more detrimental to her pushing efforts than having the epidural in place.

Open-Glottis Versus Closed-Glottis (Valsalva) Pushing

Traditionally, women have been counseled to take a deep breath, hold their breath, and push forcefully throughout the duration of a uterine contraction. The use of breath holding combined with prolonged bearing down results in a Valsalva maneuver that can decrease cardiac output and subsequent blood flow to the uterus. Conversely, open-glottis pushing is similar to the semi-voluntary grunting and pushing associated with defecation. Because the glottis is not closed, this form of maternal pushing effort does not greatly increase intrathoracic pressure and, therefore, does not affect cardiac output.

Studies that have compared Valsalva-type closed-glottis pushing to open-glottis pushing have found that Valsalva-type pushing is associated with more FHR decelerations[3]; a higher incidence of perineal trauma (lacerations, episiotomy)[51]; maternal exhaustion; and an increased risk for cystocele and urinary stress incontinence.[3,52,53] This evidence is compelling, and closed-glottis pushing is not recommended in practice today.[54,55]

Spontaneous Versus Directed Pushing

Spontaneous pushing occurs when a woman in labor responds to the strong sensations of rectal pressure and an urge to push with bearing-down efforts and open-glottis pushing. In contrast, directed or coached pushing occurs when the healthcare provider instructs the woman to push in a specific manner, including direction for the woman to take a deep breath and hold it for a sustained period, typically “to the count of 10.” Directed pushing also includes providing

direction for a woman to push before she feels an urge to do so and asking her to hold the push beyond what she would do spontaneously. **Table 28-6** summarizes the differences between spontaneous pushing and directed pushing. While there are gaps in the literature regarding the benefits of spontaneous pushing, its use has not been shown to cause harm to either the woman or her newborn.[6,39,44,56]

Table 28-6 Comparison of Spontaneous to Directed Approaches to Pushing During Second-Stage Labor

Spontaneous	Directed
Also referred to as physiologic and supportive pushing.	Also referred to as Valsalva and authoritative pushing.
Maternal bearing-down efforts begin when the woman feels an overwhelming urge to push.	Maternal bearing-down efforts begin when a healthcare provider discovers that the woman is fully dilated (10 cm) and announces that she is ready to push.
Pushing is spontaneous—in response to a woman's natural urges and sensations. Usually results in 5–6 bearing-down efforts per contraction of 3–10 seconds each.	Pushing is provider directed—with instructions from a healthcare provider about when and how long to bear down. Usually results in 3 pushes per contraction of 8–10 seconds each.
Pushing often occurs while the woman exhales, with open mouth and open glottis. Usually results in grunting and low groaning.	Pushing occurs while the woman holds her breath. Silence is encouraged. Often results in a Valsalva maneuver.

Maternal Position During the Second Stage of Labor

Principles to consider when recommending positions to promote labor progress in the second stage are reviewed in **Table 28-7**. A woman can push in any position—on her side, on her hands and knees, kneeling, standing, or squatting. The position a woman assumes during second-stage labor depends on her preference, her health status, and the health status of the fetus. Many RCTs have examined upright versus supine positions during the second stage of labor. A meta-analysis of RCTs that compared the outcomes of women without an epidural who pushed in upright positions with the outcomes of women without an epidural who pushed in supine or lithotomy positions identified several potential benefits of pushing in upright positions.[57] Pushing in upright positions was associated with a small reduction in duration of second-stage labor, fewer episiotomies and assisted deliveries, and fewer abnormal FHR patterns.[57] However, pushing in upright positions was also associated with a possible increase in second-degree perineal tears and an increase in maternal blood loss greater than 500 mL.[57] The researchers concluded that without a specific indication to adopt a particular position, women in labor should be encouraged to use the position that is most comfortable and that supports ongoing progress in second stage; this is true for women both with and without an epidural.[57,58] The choice of birthing position can be facilitated by sharing information, asking the woman about her preferences, and using supportive direction.[37]

Table 28-7 Principles to Consider When Recommending Positions in Second-Stage Labor

1. Supine and lithotomy positions are associated with uterine pressure on the vena cava, maternal hypotension, decreased uterine perfusion, and variant fetal heart rate patterns.
2. Upright positions are associated with less pain and stronger contractions.
3. When a woman rounds her back rather than arching it, the uterus and fetal presenting part are better aligned with the pelvis.
4. Leaning forward in an upright position increases the angle between the uterus and the spine, thereby directing the fetal presenting part toward the larger posterior segment of the pelvis.
5. Changing positions frequently promotes changes in pelvic diameters, which could aid the fetus finding the "best fit" during the cardinal movements.
6. Squatting increases the area within the pelvic outlet by approximately 20% and can be most beneficial in the final phase of second-stage labor when the fetal presenting part is in the plane of the outlet.
7. Regardless of the position a woman is in, her legs should not be abducted laterally in an exaggerated manner (lithotomy position) because these positions put increased stress and tension on the perineum.

Despite modest findings in RCTs, older studies that evaluated the effects of position on maternal pelvic diameters suggest that upright positions in the latter stages of the second stage may have advantages.[59,60] Radiographic studies of women in labor in upright and supine positions found that upright positions increase both the anterior–posterior and transverse diameters of the pelvic outlet, which results in approximately 20% more area in the outlet.[59,60] Although these studies are old and will not be repeated due to the adverse effects of X rays, the pelvic joints are more mobile during pregnancy and squatting does cause the sacrum to rotate backward, which creates more room in the plane of the outlet.

Preparing for the Birth

If the birth is taking place in a hospital, the midwife should be aware of institutional criteria for use of a specific location. For example, in some settings the presence of meconium-stained fluid may require added personnel to be in attendance and the location of the birth to change from a single-room maternity care room to a delivery room. General preparations for a hospital birth are outlined in **Table 28-8**.

Table 28-8 **Preparation for Birth in the Hospital**

1. Determine the most appropriate place for the birth to occur. If the woman needs to move to a different room for birth for some reason, inform the woman and her support persons and move to the appropriate setting. In most instances, there is no need to move prior to the birth to a room other than where the labor has occurred.
2. Inform the woman's partner and others present that birth is imminent and review their roles and what to expect.
3. Notify the nursing staff that birth is nearing.
4. Confirm that newborn resuscitation equipment and supplies are present and in proper working condition.
5. Confirm that necessary instruments and materials are in a place of ready access. It is helpful to have additional sterile gloves accessible, should they be needed.
6. Put on protective clothing (gown or other waterproof garment); don gloves using sterile technique.
7. Arrange sterile equipment so that it is readily accessible. Maintain the instruments in a sterile field.
8. Drape the woman in accord with the drapes provided and institutional procedure if indicated.

Care during and immediately following birth is unique, in that both the woman and the newborn may need care simultaneously. Thus, two healthcare providers should always be present. The presence of additional personnel also depends on any risk factors that may be present. It is the responsibility of the midwife to consider the unique risk factors of the woman and newborn, to request the necessary resources in anticipation of the birth, and to know how to activate the call for emergency support should an unexpected complication occur.

In general, if labor is progressing normally, it is best to begin preparations for birth before complete dilation if the woman is multiparous and at the beginning of second-stage labor if the woman is nulliparous. The midwife will need to decide when to don protective wear, including a waterproof gown or clothing and eye protection. If a water birth is planned, the midwife will need to ensure the use of longer gloves or a gown that is fully waterproof to maintain universal precautions.

Management of the Birth

Management of the birth includes using standard hand maneuvers to support the birth, immediate assessment of the newborn, and immediate assessment of maternal status after the birth. Hand maneuvers are reviewed in **Appendix 28A**. In managing the birth, the midwife may need to make quick sequential decisions about the following management techniques: (1) how to provide supportive direction to the woman in labor through the final perineal phase of the second stage of labor; (2) whether to use warm compresses, perineal massage, or a "hands-off" versus "hands-on" approach; (3) the best approach to maintaining perineal integrity while supporting birth of the head; (4) whether to cut an episiotomy; (5) where to place the newborn immediately after birth—that is, whether to facilitate skin-to-skin contact on the mother; and (6) when to clamp and cut the umbilical cord.

Perineal Integrity

As the birth begins, the first focus is on promoting perineal integrity and preventing perineal lacerations. Birth positions known to decrease the incidence of genital tract trauma include lateral and semi-upright positions.[61,62] Squatting is associated with more second-degree lacerations, perhaps because it is a position in which the midwife may have difficulty controlling extension of the fetal head. The position associated with the highest incidence of genital tract trauma is lithotomy because marked abduction of the thighs stretches the perineum transversely so that it becomes a tight band without significant ability to flex around the emerging head.

Perineal support techniques that are purported to decrease the incidence of genital tract trauma include warm compresses, perineal massage, and "hands-off" versus "hands-on" perineal support.[62] Strategies that reduce the risk of perineal tearing are described in **Table 28-9**.[61-63] Most women who give birth vaginally sustain some degree of perineal trauma.[61,62] The types of perineal lacerations are described in **Table 28-10**.[64] Severe perineal lacerations that extend through the anal sphincter are termed "obstetric anal sphincter injuries" (OASIS). Perineal lacerations can result in both short-term and long-term morbidity and pain.

Table 28-9	Strategies to Promote an Intact Perineum
Make certain the woman's thighs and legs are not abducted laterally.	
Use of warm compresses during the perineal phase may decrease the incidence of third- and fourth-degree lacerations.	
Avoid vigorous or sustained perineal massage during the second stage, as these may cause perineal edema and tearing.	
Deliver the fetus slowly between contractions controlling extension of the head so pressure on the perineum is not suddenly forceful.	
If blanching of the perineum is observed, encourage the woman to breathe instead of push, to support slower birth of the fetal head.	

Based on Albers LL. Midwifery care measures in the second stage of labor and reduction of genital tract trauma at

birth: a randomized trial. *J Midwifery Womens Health.* 2005;50:365-372[61]; Aasheim V, Nilsen ABV, Reinar LM, Lukasse M. Perineal techniques during the second stage of labour for reducing perineal trauma: updated review. *Cochrane Database Syst Rev.* 2017;6. doi:10.1002/14651858.CD006672.pub3[62]; Low LK. Promoting physiological labor and birth in supporting a physiologic approach to pregnancy and birth. In: Avery M, ed. *Supporting a Physiologic Approach to Pregnancy and Birth*. Ames, IA: Wiley; 2013: 49-77.[63]

Table 28-10 Types of Perineal Lacerations

Perineal Laceration	Description
First degree	Involves the perineal skin only
Second degree	Involves the skin, vaginal mucosa, and posterior fourchette, extending into the perineal body fascia and musculature but not the anal sphincter; includes the superficial and deep transverse perineal muscle and fibers of the pubococcygeus and bulbocavernosus muscles
Third degree	Involves the skin, vaginal mucosa, posterior fourchette, perineal muscles, and external anal sphincter 3a: < 50% of the external anal sphincter 3b: > 50% of the external anal sphincter 3c: Complete rupture of the external anal sphincter and the internal anal sphincter is torn
Fourth degree	Involves the skin, vaginal mucosa, posterior fourchette, and perineal muscles, extending through the external and internal anal sphincter and anterior rectal mucosa
Sulcus tears	Lacerations of the vaginal mucosa and underlying tissue along one or both sides of the posterior column of the vagina instead of the middle inferior part of the vagina
Labial	Laceration that extends from the fourchette anteriorly in one or both of the labia majora bodies
Periurethral	Longitudinal or transverse tear in the labia minora very near the urethra
Clitoral	Periurethral tear that extends into or near the clitoral body
Cervical	Laceration on any part of the cervix, usually on one or both of the lateral sides at approximately 3 o'clock and 9 o'clock, where the anterior and posterior aspects join

Based on American College of Obstetricians and Gynecologists. *Obstetric Data Definitions (Version 1.0).* Washington, DC: American College of Obstetricians and Gynecologists; 2014. Available at: https://www.acog.org/-/media/Departments/Patient-Safety-and-Quality-Improvement/2014reVITALizeObstetricDataDefinitionsV10.pdf?dmc=1&ts=20171212T2301409385. Accessed December 11, 2017.[64]

A meta-analysis of studies examining the effect of various perineal support techniques on perineal trauma revealed that placing warm wet compresses on the perineum continuously during the perineal phase of second-stage labor does not improve the rate of intact perineum but does appear to decrease the incidence of third- and fourth-degree lacerations.[62] Perineal massage with lubricant was associated with an increased incidence of intact perineum and fewer episiotomies, but did not appear to influence the degree of perineal trauma in women who required suturing.[62]

"Hands off" means not touching the fetal head or perineum until the fetal head is crowning (i.e., the biparietal diameter of the fetal head is through the vulva). "Hands on" entails using one hand to maintain flexion of the fetal occiput and the other hand to guard the perineal body by placing fingers on both sides of the perineum. Studies of these two techniques have not found one method to be superior to the other.[62,65] However, a multivariate analysis of the

elements of techniques associated with reduced genital tract trauma in one of the RCTs performed found that controlled birth of the fetal head between uterine contractions is associated with the lowest rate of genital tract trauma (RR, 0.82; 95% CI, 0.67–0.99).[61]

Episiotomy

Routine episiotomy was part of standard obstetric management until the 1990s, as it was historically believed that an episiotomy would decrease the incidence of perineal lacerations and brain damage in the fetus/newborn. The change from routine episiotomy to the rare use of episiotomy occurred over the course of a decade in the 1990s.

In 1989 a seminal study that found episiotomies are more likely to result in third- or fourth-degree lacerations than are spontaneous perineal tears was published.[66] In this study, the incidence of a rectal laceration in women with and without an episiotomy was 28.4% versus 2.2%, respectively (aOR, 8.9; 95% CI, 6.1–13.0).[66] Multiple subsequent studies and a Cochrane meta-analysis validated these findings.[66-70] The 2017 Cochrane systematic review of 12 randomized trials found that 30% fewer women sustained severe perineal trauma if giving birth in sites with restrictive episiotomy policies compared to giving birth in sites with routine episiotomy policies (RR, 0.70; 95% CI, 0.52–0.94); there was no difference in long-term outcomes such as pain, urinary incontinence, or dyspareunia.[67] A large national review of discharge data from hospitals, addressing translation of evidence-based guidelines into the practice of reducing episiotomy use, found that the incidence of episiotomy decreased from 20.3% in 2002 to 9.4% in 2011.[71]

Today routine episiotomy is not recommended.[55,72] The primary indication for an episiotomy is an FHR pattern that indicates a rapidly increasing risk that the fetus is developing acidemia. An additional argument can be made that an episiotomy is beneficial in anticipation of use of forceps or vacuum extractor, or in the presence of risk factors for shoulder dystocia. However, there is no evidence that an episiotomy is beneficial prophylactically in these situations, and this procedure clearly increases the risk for an extension to or through the anal sphincter.

If an episiotomy is determined to be indicated, local anesthesia or a pudendal block can be instituted and then either of two types can be performed: midline or mediolateral (**Table 28-11**). A mediolateral episiotomy is less likely to result in a third- or fourth-degree laceration than is a midline episiotomy. In the United States, there is a greater tendency to perform midline episiotomies, whereas in other Western and European countries the mediolateral episiotomy is preferred. If the distance between the posterior fourchette and the rectal sphincter is unusually short, a mediolateral episiotomy may be preferable to prevent laceration through the rectal sphincter, especially if the woman has any condition that impairs healing ability, which would increase the chance that the episiotomy repair would break down. In short, a midline episiotomy is less painful than a mediolateral episiotomy during the healing process because there are fewer nerve branches in the locale of a midline episiotomy and its repair, but the risk of a fourth-degree laceration is higher when a midline episiotomy is used.

Table 28-11 **Types of Episiotomy**

Episiotomy	Description
Midline	A midline incision that starts at the posterior fourchette and extends inferiorly through the central tendon of the perineal body, including the transverse perineal muscle. Involves the skin, vaginal mucosa, and posterior fourchette and perineal muscles, but not the anal sphincter. Equivalent to a second-degree laceration.
Mediolateral	Incision that begins at the midline in the posterior fourchette and extends laterally and inferiorly away from the rectum. May be right or left mediolateral incision.

Birth of the Fetal Head

The ideal time for birth of the fetal head is between contractions. The combination of the uterine contraction and the maternal pushing effort exerts a double force on the perineum at the moment of birth, which makes birth of the head more rapid and the release of restraining pressure more abrupt. Close communication between the midwife and the woman facilitates a team approach to help the woman gently give birth while making every effort to protect perineal integrity.

Ritgen Maneuver

The Ritgen maneuver, or modified Ritgen maneuver, is an old technique in which the clinician applies pressure on the fetal chin with one hand and pressure on the occiput with the other hand to control the birth of the head. The fetal chin is palpated behind the woman's rectum and pulled forward while the other hand maintains flexion of the occiput. The result is that the fetal head is pulled forward while maintaining flexion. The maneuver is performed between uterine contractions. The original rationale for using this procedure was to control extension of the fetal head and prevent perineal lacerations. Over time, it became a practice used when the birth needed to be expedited.[73,74] This maneuver is rarely used today, as vacuum or forceps are chosen for indicated operative vaginal birth.

The original Ritgen maneuver, which was first introduced in 1855 in Germany, recommended placing fingers in the woman's rectum. The modified Ritgen maneuver places the fingers between the rectum and the coccyx (**Figure 28-2**). The Ritgen maneuver is not associated with a decrease in perineal lacerations.[73,74] Although rarely needed, the Ritgen maneuver may expedite birth by a short period if needed and other resources are not available. The steps involved in performance of this maneuver are detailed in **Table 28-12**.

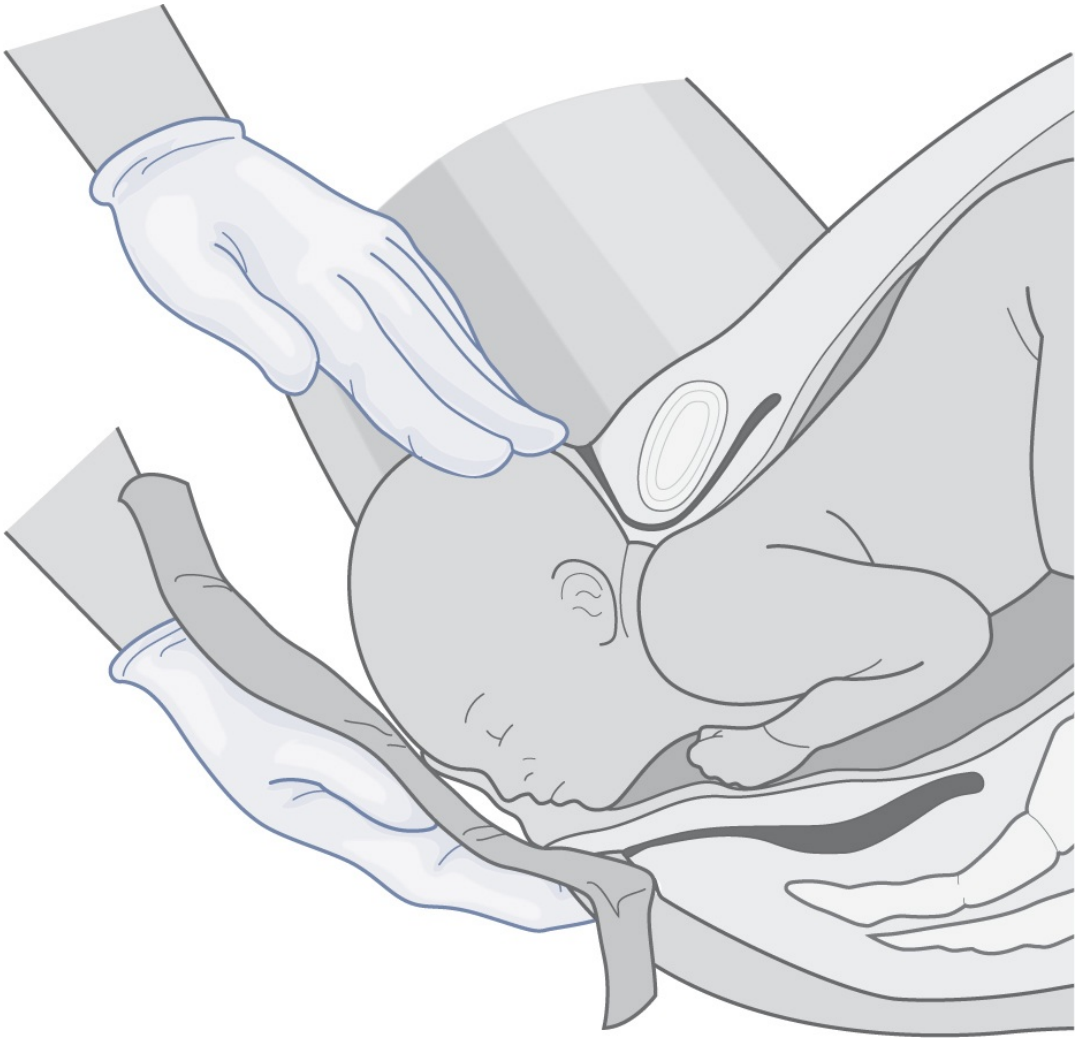


Figure 28-2 Ritgen maneuver.

Table 28-12	Steps for Ritgen Maneuver

The Ritgen maneuver is used to control the birth of the head by controlling both flexion and extension of the fetal head. The maneuver is also used to speed the birth by facilitating the process of extension through application of forward pressure on the fetal chin.

1. One hand remains on the occiput as the fetus is crowning to maintain flexion until after the biparietal diameters are visible at the introitus.
2. The other hand uses a gauze or sterile towel to protect against rectal contamination and palpates the fetal chin in the area between the maternal coccyx and rectum.
3. Forward and outward pressure is then exerted on the underneath side of the chin, and extension of the head is controlled between this hand and the hand exerting pressure on the occiput to control the pace of the birth of the infant's head.

Head-to-Body Interval and Two-Step Birth

Once the head is born, the midwife can move immediately to assist restitution and birth of the body, or wait for the next contraction to effect spontaneous restitution and birth of the shoulders before guiding the infant's body out of the vagina. The assumption is that the *one-step approach* decreases the expulsive phase duration, thereby preventing or mitigating neonatal acidemia. The *two-step approach* is a physiologic phenomenon. Although it is common practice in the United States to effect birth of the newborn quickly once the head is born, no evidence supports the contention that rapid birth is safer than a two-step birth that requires more time. Only a few studies have measured the head-to-body time interval and evaluated measures such as umbilical cord pH values and newborn hematocrit in relation to short or long head-to-body interval.[75-77]

The mean head-to-body interval using the two-step process is approximately 88 seconds, whereas that for the one-step process is approximately 24 seconds.[75] Although umbilical artery pH values have been shown to decline with longer head-to-body intervals, this decline was not statistically or clinically significant after controlling for the duration of the second stage.[75] One study of women whose births were complicated by shoulder dystocia found that a head-to-body interval of less than 5 minutes was not associated with neonatal acidemia.[76] Given these results, it appears the two-step physiologic method is safe when the fetus is not developing acidemia and is not at increased risk for acidemia.

The choice of performing a one-step or two-step method for facilitating birth is based on the individual circumstance. Because umbilical cord occlusion during the expulsive stage can be more constricting than cord occlusion during labor, fetal acidemia can also develop more quickly during this phase. Therefore, the one-step procedure may be better if the FHR pattern indicates possible developing acidemia or if other clinical factors place the fetus at increased risk for adverse effects of acidemia. The other factor to consider when making the decision about a longer expulsive stage is the risk for shoulder dystocia. Although clinical risk factors for shoulder dystocia are not strongly predictive, some authors have proposed that a rapid birth will prevent the anterior shoulder from becoming impacted behind the symphysis. However, there is little evidence to support this claim and other authors argue that utilizing the two-step approach might actually reduce rates of shoulder dystocia by allowing the shoulders to rotate into an ideal position for birth.[78,79]

Hydrotherapy and Waterbirth

Many women express interest in laboring and giving birth while submerged in water. The research regarding maternal and newborn outcomes following water immersion during labor (hydrotherapy) and/or water immersion during birth (waterbirth) is difficult to analyze. Much of the research on waterbirth has been conducted in Europe and consists of observational studies or case reports that did not have sufficient power to discern differences in non-common outcomes.[80,81] Furthermore, when women develop complications, they are generally transferred out of the hydrotherapy group, which results in differential misclassification bias that confounds the study results.[82]

Hydrotherapy During Labor

Although the research on waterbirth is not conclusive, the studies published to date have found water immersion during labor is associated with decreased use of analgesia, higher maternal satisfaction, and reduced duration of the first stage of labor.[80-83] The evidence regarding the effect of hydrotherapy on perineal lacerations is conflicting, with some studies reporting a decrease and others reporting an increase in lacerations.[80,82] Water immersion during labor has not been associated with an increase in infection, decrease in cesarean or operative birth rate, or increase in adverse neonatal outcomes.[80-83]

Waterbirth

Waterbirth refers to birth of the newborn completely underwater. The face of the newborn is gently brought to the surface within 5 to 10 seconds, while attention is paid to the length of the umbilical cord so cord avulsion does not occur.[84] Research assessing outcomes following waterbirth is sparse in contrast to the research findings regarding effects of hydrotherapy during labor. Nonetheless, to date, this body of research has identified improved maternal satisfaction and a slightly shorter duration of labor with waterbirth, although methodologic problems make it difficult to interpret the clinical significance of these results. For example, some studies evaluated addressed waterbirth, water immersion during labor, or a combination of both without clarification of which practice was being evaluated.[80] Thus, the actual effect of fetal immersion in water throughout the birth process is not well established.

Although no adverse maternal or neonatal outcomes in women who give birth in water have been identified when these studies are reviewed as a whole, the observational studies and two RCTs conducted to date are too small to detect any differences in adverse neonatal outcomes, which are rare in healthy women who are candidates for waterbirth.[80,82,85] Several case reports reporting cord avulsion, NICU admission, blood transfusion, neonatal hyponatremia, and neonatal drowning in conjunction with waterbirth have been published.[86] Nevertheless, the real incidence of adverse newborn outcomes is unknown. Schafer reviewed all reports of cord avulsion and calculated a projected rate of 3.1 per 1000 births, with 23% of these events resulting in NICU admission and 13% resulting in neonatal hemorrhage.[86] These figures are a rough estimate because no prospective studies have either tracked the incidence of adverse

neonatal outcomes or reported the incidence of cord avulsion in women who give birth outside of water. The American Academy of Pediatrics (AAP) and the American College of Obstetricians and Gynecologists (ACOG) have published a joint position statement that supports water immersion during labor, but urge caution and recommend against waterbirth until more research has been conducted.[87]

Despite a lack of strong evidence either supporting or refuting the value of waterbirth, use of water immersion during labor and birth is of interest to many women, and an option that is available in some settings. The American College of Nurse-Midwives has published guidelines for waterbirth that include indications, contraindications, protocols, and requirements for quality control.[84] These guidelines can be found in the Resources section at the end of this chapter.

Immediately Following the Birth

Placing the Newborn in Skin-to-Skin Contact

The ideal placement for the newborn immediately following the birth is on the mother's abdomen, in skin-to-skin contact, promoting a positive transition from intrauterine to extrauterine life (**Figure 28-3**). Skin-to-skin contact promotes thermoregulation of the newborn, reduces physiologic stress, enhances successful breastfeeding both initially and at 3 months postpartum, and results in improved maternal–infant bonding.[88] Evaluation of the infant, the assignment of Apgar scores, and initial vital signs can be completed while the newborn remains skin-to-skin with the mother.[88]



Figure 28-3 Skin-to-skin care.

Reproduced with permission from Melissa Scott.

Healthy newborns who have uninterrupted contact with their mother may begin to make crawling movements toward her breast after approximately 20 minutes, and by an average of 50 minutes most are correctly sucking and breastfeeding.[88] Newborn procedures should be delayed until at least an hour after the birth so skin-to-skin placement can occur while the

newborn adapts to post-gestational life.

Clamping and Cutting the Umbilical Cord: Delayed Cord-Clamping

Delayed cord-clamping is an evidence-based practice that promotes the transition from intrauterine to extrauterine life and offers other health benefits.[89-92] Delayed cord-clamping involves waiting at least 30 seconds, and as long as 5 minutes, after the birth of the newborn before the umbilical cord is clamped and cut.[90-92]

Delayed cord-clamping allows the blood that was circulating in the umbilical vessels and the placenta to return into the newborn's circulation after birth. This process, which is known as placental transfusion, increases blood volume in the newborn by approximately 30% and red cell volume by 50%.[93] The increased volume, red blood cells, stem cells, and other components of this blood have significant health benefits for all newborns; thus, delayed cord-clamping is recommended as the standard of care.[91,92,94,95]

It is unclear why clamping the umbilical cord immediately after the birth became standard practice in hospital settings in the 1950s and 1960s.[96,97] One review of historical records found references to immediate cord-clamping for purposes of avoiding staining bed linens and avoiding blood loss from the newborn.[98] Another reference suggested immediate clamping could help to reduce maternal postpartum hemorrhage and placental retention.[97] Interestingly, reviews of historical writing about the timing for cutting the umbilical cord found that the timing associated with best outcomes has long been controversial.[98] Nonetheless, until research published in the last two decades called immediate clamping and cutting of the cord into question, it was standard practice to cut the umbilical cord immediately.[90] Modern research has established that waiting for a period of time before cutting the cord is associated with significant benefits for all newborns.[99-103]

The timing and procedure for cutting the umbilical cord are presented in **Appendix 28B**, which includes discussion of how to integrate umbilical cord-clamping in complex clinical situations such as when the birth is complicated by a shoulder dystocia, tight nuchal cord, or newborn need for resuscitation.

Although the definition of delayed cord-clamping varies somewhat in different professional organization recommendations,[91,92,104] delayed cord-clamping has important health benefits for both preterm and term infants. For the term infant, this practice results in higher ferritin levels, reduces the risk of iron-deficiency anemia, and can increase stores of brain myelin, which is important for more rapid and efficient brain communication.[89,105] For the preterm infant, delayed cord-clamping results in less need for blood transfusions, decreased incidence of intraventricular hemorrhage and necrotizing enterocolitis, improved mean systemic blood pressure, and reduced hospital mortality.[99,103,104] Newborns at all gestational ages have better neurodevelopmental outcomes when cutting of the umbilical cord is delayed.[102,106,107] Waiting just a few short minutes after birth has significant health benefits and can be done in all clinical situations, with any type of birth, and across all gestational ages.[90,108]

Umbilical Cord Blood Banking

Cord blood banking involves collecting and storing umbilical cord blood after birth, whereas tissue banking involves storing the umbilical cord or placenta. A public bank stores blood or tissue at no cost to parents, but they give up ownership of the cells and the stem cells can go to any person who qualifies for them. A private bank requires fees for storage, but the stem cells are "saved" for the future for that newborn or family members. ACOG suggests that if stem cells are harvested, they should be stored in public banks because generally these banks distribute more stem cells for transplant to the public.[109]

Mousavi described the quality and quantity of 35,000 cord blood units from around the world and reported that the average cord blood collection volume is 80 mL (24 mL/kg), which contains an average of 13 billion total nucleated cells per unit.[110] Total nucleated cells contain hematopoietic (blood cell manufacturing) stem cells, along with many non-hematopoietic stem cells; these cells can help repair many different tissues in the body.[110] Stem cells are part of the body's innate healing system. Damaged tissue releases cytokines, which signal stem cells to travel to the damaged area and begin the healing process. Animal studies show that human umbilical cord blood stem cells help to heal damage in the body, no matter how they are administered.[111]

Collection of cord blood for banking requires immediate cord-clamping and rules out the benefit of a placental transfusion for the newborn. As more studies find benefits for infants when delayed cord-clamping occurs, ethical issues surrounding umbilical cord blood banking become more prescient. An informed discussion with the woman and her family is needed about the potential risks and benefits of cord blood/tissue collection.

Inspection of the Perineum and Laceration Repair

The third stage of labor begins as soon as the infant is born. This is the time during which the risk for postpartum hemorrhage is the greatest. As soon as the infant is safely being observed/cared for by an assistant, the midwife checks for vaginal bleeding while inspecting the vagina and perineum for lacerations and arterial bleeding. The systematic inspection process, technique for initiating local or pudendal anesthesia, and laceration repair are summarized in **Appendix 28C** through **Appendix 28F**.

Apgar Score

The Apgar newborn scoring system was devised during the mid-twentieth century by Virginia Apgar, an anesthesiologist who worked at a time when use of heavy maternal sedation and anesthesia was common during labor. During this era, it was not unusual for newborns to have respiratory depression and low tone at birth secondary to the large doses of morphine administered late in labor. Dr. Apgar developed the Apgar score as a way of assessing the effects of maternal anesthesia on the newborn.[112, 113] Although the actual reason Apgar developed the score is not known for certain, the simplicity of assessing the newborn to assign this score resulted in rapid adoption into clinical practice. The Apgar score is now universally used in the United States as a measure at 1 and 5 minutes of how well the newborn is adapting to extrauterine life.

Apgar scoring uses five components to assess the newborn.[113] The five components of the Apgar score include key elements of cardiovascular and circulatory status, lung function, and neuromuscular integrity. A score of 7–10 denotes a vigorous neonate, a newborn with a score of 4–6 requires some focused resuscitation efforts, and a newborn with a score of 3 or less requires intensive resuscitation procedures. Several years after the scoring system was published, an acronym was devised using Virginia Apgar's surname. Therefore, some professionals know the scoring system as Appearance, Pulse, Grimace, Activity, and Respirations (APGAR), as noted in **Table 28-13**. The Apgar score may be misinterpreted as an assessment used to detect the presence of perinatal asphyxia. Although some components of the Apgar score are related to pre-birth acid–base balance, this score is not a reliable measure of perinatal asphyxia. Apgar scores are an indicator of resuscitation success, however, and are continued every 5 minutes if resuscitation efforts are ongoing. Moreover, resuscitation efforts should not depend on the first Apgar score for initiation since the first minute of life can be a critical one.

Table 28-13 The Apgar Score

Classic Component	Acronym	Score of 0	Score of 1	Score of 2
Color	**A**ppearance	Blue/pale over entire body	Acrocyanosis Body pink	Body and extremities pink
Heart rate	**P**ulse	Absent	< 100 beats/min	≥ 100 beats/min
Reflex irritability	**G**rimace	No response to stimulation	Grimace/weak cry when stimulated	Cry or attempt to pull away when stimulated
Muscle tone	**A**ctivity	None	Some flexion	Flexed extremities that resist extension
Respiratory efforts	**R**espirations	Absent	Week, irregular, gasping	Strong cry

Based on Apgar V. A proposal for a new method of evaluation of the newborn infant. *Curr Res Anesth Analg.* 1953;32(4):260-267.[113]

To avoid subjectivity in the assignment of the Apgar score, the newborn is examined and Apgar score is assigned by a birth attendant or nurse who is not the individual responsible for conducting the birth. The 1-minute Apgar score is not associated with long-term outcomes. Neonatal mortality is associated with the 5-minute Apgar score, however, and studies in this century continue to demonstrate that relationship, even after the advent of umbilical cord blood sampling and other assessment methods.[114,115]

Conclusion

Throughout all stages of the birthing process, the midwife maintains a safe, secure, respectful environment that supports an optimal labor and birth for the woman, fetus, and newborn. To do so, the midwife must have full awareness of the desires and expectations of the individuals involved in the labor and birth. This will allow the midwife to orchestrate an optimal birth experience within the boundaries of the individual circumstances, birth environment, and clinical factors that serve to influence the care provided to the laboring family.

Resources

Organization	Description	Webpage
American College of Nurse-Midwives (ACNM)	Waterbirth resources	http://www.midwife.org/Water-Birth-Resources
California Perinatal Quality Care Collaborative (CPQCC)	Resources and a webinar on delayed cord-clamping	https://www.cpqcc.org/DCCPP
International Federation of Gynecology and Obstetrics (FIGO)	Management of the second stage of labor	http://www.sigo.it/wp-content/uploads/2015/10/guida_management_labour1.pdf

References

1. Kennedy HP, Rousseau AL, Low LK. An exploratory metasynthesis of midwifery practice in the United States. *Midwifer* 2003;19(3):203-214.
2. Roberts JE. A new understanding of the second stage of labor: implications for nursing care. *J Obstet Gynecol Neonatal Nurs*. 2003;39:794-801.
3. Friedman EA. *Labor: Clinical Evaluation and Management*. 2nd ed. New York, NY: Appleton-Century-Crofts; 1978.
4. Roberts JE. The "push" for evidence: management of the 2nd stage. *J Midwifery Womens Health*. 2002;47(1):2-15.
5. American College of Obstetricians and Gynecologists and Society for Maternal–Fetal Medicine; Caughey AB, Cahill AG, Guise JM, Rouse DJ. Safe prevention of the primary cesarean delivery. *Am J Obstet Gynecol*. 2014;210(3):179-193.
6. Kopas ML. A review of evidence-based practices for management of the second stage of labor. *J Midwifery Womens Health*. 2014;59:264-276.
7. Spong CY, Berghella V, Wenstrom KD, Mercer BM, Saade GR. Preventing the first cesarean delivery: summary of a join Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal–Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. *Obstet Gynecol*. 2012;120(5):1181-1193.
8. Kilpatrick SJ, Laros RK. Characteristics of normal labor. *Obstet Gynecol*. 1989;74(1):85-87.
9. Zhang J, Landy HJ, Brancy DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcomes. *Obst Gynecol*. 2010;116:1281-1287.
10. Cheng YW, Hopkins LM, Laros RK Jr, Caughey AB. Duration of the second stage of labor in multiparous women: Maternal and neonatal outcomes. *Am J Obstet Gynecol*. 2007;196(6):585-590.
11. Cheng YW, Shaffer BL, Nicholson JM, Caughey AB. Second stage of labor and epidural use: a larger effect than previously suggested. *Obstet Gynecol*. 2014;123(3):527-535.
12. Rouse DJ, Weiner SJ, Bloom SL, et al. Second-stage labor duration in nulliparous women: relationship to maternal and perinatal outcomes. *Am J Obstet Gynecol*. 2009;201(4):357.e1-357.e7.
13. Gimovsky AC, Berghella V· Prolonged second stage: what is the optimal length? *Obstet Gynecol Surv*. 2016;71(11):667-674.
14. Laughon SK, Berghella V, Reddy UM, Sundaram R, Lu Z, Hoffman MK. Neonatal and maternal outcomes with prolonge second stage of labor. *Obstet Gynecol*. 2014;124(1):57-67.
15. LeRay C, Audibert F, Goffinet F, Fraser W. When to stop pushing: effects of duration of second-stage expulsion efforts or maternal and neonatal outcomes in nulliparous women with epidural analgesia. *Am J Obstet Gynecol*. 2009;201(4):361.e1-361.e7.
16. Kearney R, Miller JM, Ashton-Miller J, DeLancey JOL. Obstetric factors associated with levator ani muscle injury after vaginal birth. *Obstet Gynecol*. 2006;107(1):144-149.
17. Low LK, Zielinski R, Tao Y, Galecki A, Brandon C, Miller J. Predicting birth-related levator ani tear severity in primiparou women: Evaluating Maternal Recovery from Labor and Delivery (EMRLD Study). *Open J Obstet Gynecol*. 2014;4:266-278. doi:10.4236/ojog.2014.46043.
18. Miller JM, Low LK, Zielinski R, Smith AR, DeLancey JO, Brandon C. Evaluating maternal recovery from labor and delivery: bone and levator ani injuries. *Am J Obstet Gynecol*. 2015;213(2):188.e1-188.e11.
19. Cheng YW, Caughey AB. Defining and managing normal and abnormal second stage of labor. *Obstet Gynecol Clin Nort Am*. 2017;44:547-566.
20. Leiberman E, Davidson K, Lee-Parritz A, Shearer E. Changes in fetal position during labor and their association with epidural anesthesia. *Obstet Gynecol*. 2005;105:974-982.
21. Simkin P. The fetal occiput position: state of the science and a new perspective. *Birth*. 2010;37(1):61-71.
22. Osborne C, Ecker JL, Gauvreau K, Davidson KM, Lieberman E. Maternal temperature elevation and occiput posterior position at birth in women receiving epidural analgesia. *J Midwifery Womens Health*. 2011;56(5):446-451.
23. Cheng YW, Shaffer BL, Caughey AB. The association between persistent occiput posterior position and neonatal outcomes. *Obstet Gynecol*. 2006;107:837-834.
24. Malvasi A, Tinelli A, Barbera TM et al. Occiput posterior position diagnosis: vaginal examination or intrapartum sonography? A clinical review. *J Matern Fetal Med*. 2014;27(5):520-526.
25. Le Ray C, Lepleux F, De La Calle D, et al. Lateral asymmetric decubitus position for the rotation of occipito-posterior positions: multicenter randomized controlled trial EVADELA. *Am J Obstet Gynecol*. 2016;215;511.e1-511.e7.
26. Stremler R, Hodnett E, Petryshen P, Stevens B, Weston J, Willan AR. Randomized controlled trial of hands and knees positioning for occiput posterior position in labor. *Birth*. 2005;32(4):243-251.
27. Cohen SR, Thomas CR. Rebozo technique for fetal malposition in labor. *J Midwifery Womens Health*. 2015;60(4):445-451.
28. Shaffer BL, Cheng YW, Vargas JE, Caughey AB. Manual rotation to reduce cesarean delivery in persistent occiput posterior or transverse position. *J Matern Fetal Neonatal Med*. 2011;24(1):65-72.
29. Liang CC, Wong SY, Tsay PT, et al. The effect of epidural analgesia on postpartum urinary retention in women who delive

vaginally. *Int J Obstet Anesth*. 2002;11(3):164-169.
30. Weiniger CF, Wand S, Nadjari M, et al. Post-void residual volume in labor: a prospective study comparing parturients with and without epidural analgesia. *Acta Anaesthesiol Scand*. 2006;50(10):1297-1303.
31. American Society of Anesthesiologists. Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia and the Society for Obstetric Anesthesia and Perinatology. *Anesthesiology*. 2016;124(2):270-300.
32. American College of Nurse-Midwives. Clinical Bulletin No. 16: providing oral nutrition to women in labor. *J Midwifery Womens Health*. 2016;61(4):528-532.
33. Bergstrom L, Richards L, Morse J, Roberts J. How caregivers manage distress in second stage labor. *J Midwifery Womens Health*. 2010;55(1):34-38.
34. Sperlich M, Seng JS. *Survivor Moms: Women's Stories of Birthing, Mothering and Healing After Sexual Abuse*. Eugene, OR: Motherbaby Press; 2008.
35. American College of Nurse-Midwives. Clinical Bulletin No. 11: intermittent auscultation for intrapartum fetal heart rate surveillance. *J Midwifery Womens Health*. 2015;60(5):626-632.
36. Roberts JE, Gonzalez C, Sampselle C. Why do supportive birth attendants become directive of maternal bearing down efforts in second stage? *J Midwifery Womens Health*. 2007;52:134-141.
37. Nieuwenhuijze M, Low LK, Korstjens I, Lagro-Janssen T. The role of maternity care providers in promoting shared decision-making regarding birthing positions during second-stage labor. *J Midwifery Womens Health*. 2014;59(3):277-285. doi:10.1111/jmwh.12187.
38. Association of Women's Health, Obstetric, and Neonatal Nurses (AWHONN). *Nursing Care and Management of the Second Stage of Labor: Evidence-Based Clinical Practice Guideline*. 2nd ed. Washington, DC: AWHONN; 2007.
39. Roberts J, Hanson L. Best practices in second stage labor care: maternal bearing down and positioning. *J Midwifery Womens Health*. 2007;52(3):238-245.
40. Tuuli M, Frey HA, Odibo AO, Macones GA, Cahill AG. Immediate compared with delayed pushing in the second stage of labor: a systematic review and meta-analysis. *Obstet Gynecol*. 2012;120:660-668.
41. Brancato R, Church S, Stone P. A meta-analysis of passive descent versus immediate pushing in nulliparous women with epidural analgesia in the second stage of labor. *J Obstet Gynecol Neonatal Nurs*. 2008;37:4-12.
42. Fraser WD, Marcoux S, Krauss I, Douglas J, Goulet C, Boulvain M. Multicenter randomized, controlled trial of delayed pushing for nulliparous women in the second stage of labor with continuous epidural analgesia. *Am J Obstet Gynecol*. 2000;182:1165-1172.
43. Fitzpatrick M, Harkin R, McQuillan K, et al. A randomised clinical trial comparing the effects of delayed versus immediate pushing with epidural analgesia on mode of delivery and faecal continence. *Br J Obstet Gynaecol*. 2002;109(12):1359-1365.
44. Simpson KR, James DC. Effects of immediate versus delayed pushing during 2nd-stage labor on fetal well-being: a randomized clinical trial. *Nurs Res*. 2005;54(3):149-157.
45. Gillesby E, Burns S, Dempsey A, et al. Comparison of delayed versus immediate pushing during second stage of labor for nulliparous women with epidural anesthesia. *J Obstet Gynecol Neonatal Nurs*. 2010;39(6):635-644.
46. Vause S, Congdon HM, Thornton JG. Immediate and delayed pushing in the second stage of labour for nulliparous women with epidural analgesia: a randomised controlled trial. *Br J Obstet Gynaecol*. 1998;105(2):186-188.
47. Yee LM, Sandoval G, Bailit J, et al.; Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Maternal–Fetal Medicine Units (MFMU) Network. Maternal and neonatal outcomes with early compared with delayed pushing among nulliparous women. *Obstet Gynecol*. 2016;128(5):1039-1047.
48. Lemos A, Amorim MRM, De Andrade AD, de Souza A, Filho JEC, Correia JB. Pushing/bearing down methods for the second stage of labour. *Cochrane Database Syst Rev*. 2017;10:CD009124. doi:10.1002/14651858.CD009124.pub2.
49. Mhyre JM. What's new in obstetric anesthesia? *Int J Obstet Anesth*. 2011;20(2):149-159.
50. Torvaldsen S, Roberts CL, Bell JC, Raynes-Greenow CH. Discontinuation of epidural analgesia late in labour for reducing the adverse delivery outcomes associated with epidural analgesia. *Cochrane Database Syst Rev*. 2004;4:CD004457. doi:10.1002/14651858.CD004457.pub2.
51. Sampselle CM, Hines S. Spontaneous pushing during birth: relationship to perineal outcomes. *J Nurse Midwifery*. 1999;44(1):36-39.
52. Bloom SL, Casey BM, Schaffer JI, McIntire DD, Leveno KJ. A randomized trial of coached versus uncoached maternal pushing during the second stage of labor. *Am J Obstet Gynecol*. 2005;194:10-13.
53. Schaffer JI, Bloom SL, Casey BM, et al. A randomized trial of the effects of coached vs uncoached maternal pushing during the second stage of labor on postpartum pelvic floor structure and function. *Am J Obstet Gynecol*. 2005;192(5):1692-1696.
54. American College of Obstetricians and Gynecologists. Committee Opinion No. 687: approaches to limit intervention during labor and birth. *Obstet Gynecol*. 2017;129(2):e20-e28.
55. King TL, Pinger W. Evidence-based practices for intrapartum care: the pearls of midwifery. *J Midwifery Womens Healt*

2014;59:572-585.
56. Yildirim G, Kizilkaya N. Effects of pushing techniques in birth on the mother and fetus: a randomized study. *Birth*. 2008;35(1):25-30.
57. Gupta JK, Sood A, Hofmeyr GJ, Vogel JP. Position in the second stage of labour for women without epidural anaesthesia. *Cochrane Database Syst Rev*. 2017;5:CD002006. doi:10.1002/14651858.CD002006.pub4.
58. Kibuka M, Thornton JG. Position in the second stage of labour for women with epidural anaesthesia. *Cochrane Database Syst Rev*. 2017;2:CD008070. doi:10.1002/14651858.CD008070.pub3.
59. Fenwick L, Simkin P. Maternal positioning to prevent or alleviate dystocia in labor. *Clin Obstet Gynecol*. 1987;30:83-89.
60. Russell JGB. Moulding of the pelvic outlet. *J Obstet Gynaecol Br Commonw*. 1969;76:817-820.
61. Albers LL. Midwifery care measures in the second stage of labor and reduction of genital tract trauma at birth: a randomized trial. *J Midwifery Womens Health*. 2005;50:365-372.
62. Aasheim V, Nilsen ABV, Reinar LM, Lukasse M. Perineal techniques during the second stage of labour for reducing perineal trauma: updated review. *Cochrane Database Syst Rev*. 2017;6. doi:10.1002/14651858.CD006672.pub3.
63. Low LK. Promoting physiological labor and birth in supporting a physiologic approach to pregnancy and birth. In: Avery M ed. *Supporting a Physiologic Approach to Pregnancy and Birth*. Ames, IA: Wiley; 2013: 49-77.
64. American College of Obstetricians and Gynecologists. *Obstetric Data Definitions (Version 1.0)*. Washington, DC: American College of Obstetricians and Gynecologists; 2014. Available at: https://www.acog.org/-/media/Departments/Patient-Safety-and-Quality-Improvement/2014reVITALizeObstetricDataDefinitionsV10.pdf?dmc=1&ts=20171212T2301409385. Accessed December 11, 2017.
65. Bulchandani S, Watts E, Sucharitha A, Yates D, Ismail KM. Manual perineal support at the time of childbirth: a systematic review and meta-analysis. *BJOG*. 2015;122(9):1157-1165.
66. Green JR, Soohoo SL. Factors associated with rectal injury in spontaneous deliveries. *Obstet Gynecol*. 1989;73(5 pt 1):732-738.
67. Jiang H, Qian X, Carroli G, Garner P. Selective versus routine use of episiotomy for vaginal birth. *Cochrane Database Syst Rev*. 2017;2:CD000081. doi:10.1002/14651858.CD000081.pub3.
68. Frankman EA, Wang L, Bunker CH, Lowder JL. Episiotomy in the United States: has anything changed? *Am J Obstet Gynecol*. 2009;200(5):573.e1-573.e7.
69. Albers LL, Borders N. Minimizing genital tract trauma and related pain following spontaneous vaginal birth. *J Midwifery Womens Health*. 2007;52:246-253.
70. Hartmann K, Viswanathan M, Palmieri R, Gartlehner G, Thorp J Jr, Lohr KN. Outcomes of routine episiotomy: a systematic review. *JAMA*. 2005;293:2141-2148.
71. Kozhimannil KB, Karaca-Mandic P, Blauer-Peterson CJ, Shah NT, Snowden JM. Uptake and utilization of practice guidelines in hospitals in the United States: the case of routine episiotomy. *Jt Comm J Qual Patient Saf*. 2017;43(1):41-48.
72. American College of Obstetricians and Gynecologists. Practice Bulletin No. 165: prevention and management of obstetric lacerations at vaginal delivery. *Obstet Gynecol*. 2016;128:e1-e15.
73. Jonsson ER, Elfaghi I, Rydhstrom H, Herbst A. Modified Ritgen's maneuver for anal sphincter injury atdelivery. *Obstet Gynecol*. 2008;112(2 pt 1):210-211.
74. Cunningham FG. The Ritgen maneuver: another sacred cow questioned. *Obstet Gynecol*. 2008;112(2 pt 1):212-217.
75. Locatelli A, Incerti M, Ghidini A, et al. Head-to-body interval using "two-step" approach in vaginal deliveries: effect on umbilical artery pH. *J Matern Fetal Med*. 2011;24:6:799-803.
76. Leung TY, Stoart O, Sahota DS, Suen SSH, Lau TK, Lao TT. Head to body delivery interval and risk of fetal acidosis and hypoxic ischemic encephalopathy in shoulder dystocia: a retrospective review. *BJOG*. 2011;118:474-479.
77. Zanardo V, Gabrieli C, de Luca F, et al. Head-to-body delivery by two-step approach: Effect on cord blood hematocrit. *J Matern Fetal Neonat Med* 2013;26(12): 1234-1238.
78. Kotaska A, Campbell K. Two-step delivery may avoid shoulder dystocia: head-to-body delivery interval is less important than we think. *J Obstet Gynaecol Can*. 2014;36(8):716-720.
79. Menticoglou S. Delivering shoulders and dealing with shoulder dystocia: should the standard of care change? *J Obstet Gynaecol Can*. 2016;38(7):655- 658.
80. Nutter E, Meyer S, Shaw-Battista J, Marowitz A. Waterbirth: an integrative analysis of peer-reviewed literature. *J Midwifery Womens Health*. 2014;59(3):286-319.
81. Pinette MG, Wax J, Wilson E. The risks of underwater birth. *Am J Obstet Gynecol*. 2004;190:1211-1215.
82. Bovbjerg ML, Cheuney M, Everson C. Maternal and newborn outcomes following waterbirth: the Midwives Alliance of North America Statistics Project, 2004 to 2009 cohort. *J Midwifery Womens Health*. 2016;61:11-20.
83. Cluett ER, Burns E. Immersion in water in labour and birth. *Cochrane Database Syst Rev*. 2009;2:CD000111. doi:10.1002/14651858.CD000111.pub3.
84. Kane Low L, Nutter EM. A model practice template for hydrotherapy in labor and birth. *J Midwifery Womens Health*. 2017;63(1):120-126.

85. Simpson KR. Underwater birth. *JOGNN*. 2013;42(5):588-594.
86. Schafer R. Umbilical cord avulsion in waterbirth. *J Midwifery Womens Health*. 2014;59:91-94.
87. American College of Obstetricians and Gynecologists. Committee Opinion No. 679: immersion in water during labor and delivery. *Obstet Gynecol*. 2016;128:e231-e236.
88. Moore ER, Bergman N, Anderson GC, Medley N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;11:CD003519.
89. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on materna and neonatal outcomes. *Cochrane Database Syst Rev*. 2013;7:CD004074. doi:10.1002/14651858.CD004074.pub3.
90. Mercer JS, Erickson-Owens DA. Rethinking placental transfusion and cord clamping issues. *J Perinat Neonatal Nurs*. 2012;26(3):202-217. doi:10.1002/14651858.CD003519.pub4.
91. American College of Obstetricians and Gynecologists. Committee Opinion No. 543: timing of umbilical cord clamping after birth. *Obstet Gynecol*. 2012;120(6):1522-1526.
92. American College of Nurse-Midwives. Delayed cord clamping: position statement. 2014. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000290/Delayed-Umbilical-Cord-Clamping-May-2014.pdf. Accessed December 11, 2017.
93. Yao AC, Moinian M, Lind J. Distribution of blood between infant and placenta after birth. *Lancet*. 1969;2:871-873.
94. Rabe H, Diaz-Rossello JL, Duley L, Dowswell T. Effect of timing of umbilical cord clamping and other strategies to influence placental transfusion at preterm birth on maternal and infant outcomes. *Cochrane Database Syst Rev*. 2012;8:CD003248. doi:10.1002/14651858.CD003248.pub3.
95. Lawton C, Acosta S, Watson N, et al. Enhancing endogenous stem cells in the newborn via delayed umbilical cord clamping. *Neural Regen Res*. 2015;10(9):1359-1362.
96. Bayer K. Delayed cord clamping in the 21st century: indications for practice. *Adv Neonatal Care*. 2016;16(1):68-73.
97. Downey CL, Bewley S. Historical perspectives on umbilical cord clamping and neonatal transition. *J Royal Soc Med*. 2012;105(8):325-329.
98. Niermeyer S. A physiologic approach to cord clamping: clinical issues. *Matern Health Neonatol Perinatol*. 2015;1:21. doi:10.1186/s40748-015-0022-5.
99. Raju T. Timing of umbilical cord clamping after birth for optimizing placental transfusion. *Opin Pediatr*. 2013;25(0):1-8.
100. Hutton EK, Hassan ES. Late vs early clamping of the umbilical cord in full-term neonates: systematic review and meta-analysis of controlled trials. *JAMA*. 2007;297(11):1241-1252.
101. van Rheenen P, Brabin BJ. Late umbilical cord-clamping as an intervention for reducing iron deficiency anaemia in term infants in developing and industrialised countries: a systematic review. *Ann Trop Paediatr*. 2004;24(1):3-16.
102. Andersson OB, Lindquist B, Lindgren M, Stjernqvist K, Domellöf M, Hellström-Westas L. Effect of delayed cord clamping on neurodevelopment at 4 years of age: a randomized clinical trial." *JAMA Pediatr*. 2015;169(7):631-638.
103. Fogarty M, Osborn DA, Askie L, et al. Delayed vs early umbilical cord clamping for preterm infants: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2018;218(1):1-18.
104. World Health Organization. Guideline: delayed umbilical cord clamping for improved maternal and infant health and nutrition outcomes. 2014. Available at: http://apps.who.int/iris/bitstream/10665/148793/1/9789241508209_eng.pdf. Accessed December 11, 2017.
105. Mercer J, Erickson-Owens D, Deoni S. Effects of placental transfusion on ferritin and brain myelin volume at 4 months of age [Abstract]. Session 3130: Neonatal Medicine: Clinical Trial I, Pediatric Academic Societies conference, 2016.
106. Mercer JS, Erickson-Owens DA, Vohr BR, et al. Effects of placental transfusion on neonatal and 18 month outcomes in preterm infants: a randomized controlled trial. *J Pediatr*. 2016;168:50-55.
107. Rabe H, Sawyer A, Amess P, Ayers S; Brighton Perinatal Study Group. Neurodevelopmental outcomes at 2 and 3.5 years for very preterm babies enrolled in a randomized trial of milking the umbilical cord versus delayed cord clamping. *Neonatology*. 2016;109(2):113-119.
108. Mercer JS, Erickson-Owens DA. Is it time to rethink cord management when resuscitation is needed? *J Midwifery Womens Health*. 2014;59(6):635-644.
109. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 648: umbilical cord blood banking. *Obstet Gynecol*. 2015;126(6):e127-e129.
110. Mousavi SH, Abroun S, Zarrabi M, Ahmadipanah M. The effect of maternal and infant factors on cord blood yield. *Pediatr Blood Cancer*. 2017;64:e26381. doi:10.1111/pbc.26381.
111. Meier C, Middelanis J, Wasielewski B, et al. Spastic paresis after perinatal brain damage in rats is reduced by human cord blood mononuclear cells. *Pediatr Res*. 2006;59(2):244-249.
112. Finster M, Wood M. The Apgar score has survived the test of time. *Anesthesiology*. 2005;102(4):855-857.
113. Apgar V. A proposal for a new method of evaluation of the newborn infant. *Curr Res Anesth Analg*. 1953;32(4):260-267.
114. Casey BM, McIntire DD, Leveno KJ. The continuing value of the Apgar score for the assessment of newborn infants. *N Engl J Med*. 2001;344:467-471.
115. Vahabi S, Haidari M, Akbari Torkamani S, Gorbani Vaghei A. New assessment of relationship between Apgar score and

early neonatal mortality. *Minerva Pediatr.* 2010;62(3):249-252.

APPENDIX

28A

Hand Maneuvers for Birth

BARBARA J. REALE

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Introduction

The first hand placement used during birth is directed toward preserving perineal integrity as the fetus emerges. Once the head emerges, the next hand placement is critical as it will allow the midwife to maintain two secure grips on the newborn throughout the birth and immediately after the birth. Newborns are slippery, and sometimes the combination of maternal pushing and uterine contractions results in a rapid forceful birth. It is imperative that the midwife is able to hold the newborn securely throughout the process. The hand maneuvers presented in this appendix can be used in any setting.

Controlling extension of the fetal head with one hand is the technique that has the most evidence with regard to decreasing the incidence of perineal lacerations.[1,2] Other techniques include (1) perineal massage; (2) application of warm compresses; (3) "hands off," wherein the midwife does not touch the fetal head or perineal area until the biparietal diameter is born; and (4) "hands on," which is variously described as flexion of the fetal head to control extension and manual support of the perineum. In practice, the hands-on technique refers to placement of one hand on the fetal head to control extension. The thumb and forefinger of the other hand are placed on either side of the perineum near the ischial tuberosities so that a slight amount of pressure is directed inward to counter the transverse stretching of the perineum caused by pressure from the fetal head.

Research evidence regarding use of prenatal perineal massage and application of warm compresses is of low to moderate quality, but these practices are associated with a small reduction in third- and fourth-degree lacerations in nulliparous women.[1,2] Vigorous massage can create edema and is not recommended. Although randomized controlled trials (RCTs) comparing "hands-off" and "hands-on" techniques for supporting the perineum prior to birth

have not demonstrated superior effectiveness of one technique versus the other, many midwives use some method of perineal support.[1-5] Therefore, although not conclusively evidence based, these techniques are presented here as options for use during birth.

Hand Maneuvers for Birth When the Fetus Is in an Occiput Anterior Position and the Woman Is in a Semi-Sitting, Lateral, or Dorsal Position

1. Ensure both access to the perineum and visibility of the vulvar–perineal area.
   a. The midwife stands or sits in a position that allows for a clear visual field and comfortable access to the woman's perineum.
   b. A sterile drape is placed on a surface within easy reach, on which instruments and a bowl for the placenta are placed.
2. Control extension of fetal vertex. During extension of the head, the anterior–posterior diameter of the fetal vertex gradually changes from the suboccipitobregmatic diameter (9.5 cm) to the occipitofrontal diameter (11.5 cm), and then to the occipitomental diameter (12.5 cm). Gradual extension prevents perineal trauma as these increasing anterior–posterior diameters stretch the perineum during the birth. The primary goal is to prevent sudden extension of the fetal head, which increases the likelihood of perineal laceration.
   a. To control extension of the fetal head during crowning, the midwife places the palmar side of one hand, with fingers held straight, on the portion of the occiput of the fetal vertex visible at the vaginal introitus. This should be done as the vulva begins to stretch around the presenting part, with crowning imminent (**Figure 28A-1**). Control of extension is maintained best when the *fingers are kept in a straight plane with the hand*. This finger positioning allows the midwife to control extension of the head. When just the fingertips are placed on the head, the midwife does not have as much control. If sudden extension occurs with a contraction or maternal pushing, the fingertips are likely to slide along the curve of the fetal head, touch or hurt the sensitive clitoral area, and the head will pop into the midwife's palm, placing rapid pressure on the perineum. The use of one hand to control extension leaves the other hand free to check for a nuchal cord, and handle any equipment or supplies that may be needed. Given that midwives' hands vary in size and that women give birth in many different positions, the actual placement may be less important than identifying a manner in which the individual midwife can comfortably provide the needed control and simultaneously visualize the perineum. Good body mechanics are important to prevent injury to the midwife.



Figure 28A-1 The midwife's fingers are in a flat plane with the palm of the hand to allow maximum control of extension of the fetal head.

i. If a woman is lateral (side-lying), the woman's legs should be bent at the knee, and when open, remain parallel to each other. Pillows or a peanut ball between the knees can increase comfort in this position. If the upper leg is pushed toward the woman's head and flexed more dramatically than the lower leg, the pelvis is forced out of alignment and the perineum becomes tightly stretched. When given the opportunity to raise and position the upper leg spontaneously, many women will not do so until birth is imminent.

ii. Visualization of the perineum and maintaining flexion of the fetal vertex is easiest if the midwife stands behind the woman's back. If facing the woman's abdomen, visualization of the perineum is impaired, but guiding the rest of the birth can be easier. In either position, the midwife's fingers can be placed on the occiput, exerting pressure with the palmar surfaces, toward the perineum as needed to control extension of the head during the birth.

b. If the midwife choses to use a second hand to support the perineum ("hands on" approach): Using the hand that is not controlling the pace of extension of the fetal head, a thumb and one finger is placed on the perineum near the ischial tuberosities below the fourchette, so that the midline area of the perineum, which is likely to tear, is between the thumb and the finger. Applying pressure with the thumb and fingers inward toward each other and the middle of the perineum (perineal body) reduces the transverse pressure created as the fetal head extends.

3. Check for a nuchal cord. As soon as the head has emerged, the fingertips of one hand palpate the infant's neck, sweeping the fingers along the neck in both directions, to

determine if the umbilical cord is looped around the neck. If a nuchal cord is present, slip fingertips between the cord and neck to ascertain how tight the cord is. There are four options for proceeding with the birth if a nuchal cord is present:

- **a.** *Cord reduction:* If the cord is loose, slip it forward over the head before delivery of the shoulders (referred to as reducing the cord over the head).
- **b.** *Birth through the cord:* If the cord is too tight to reduce but still has some mobility, slip it back over the shoulders as the infant is born, allowing the infant to be born through the cord.
- **c.** *Somersault maneuver:* If the cord is too tight to slip over the shoulders but loose enough to permit some movement, use one hand to keep the head close to the maternal thigh throughout the birth of the body and the other hand to "somersault" the body over the perineum, which will limit the traction placed on the cord.[6,7] The somersault maneuver is depicted in **Figure 28A-2**.
- **d.** *Clamp and cut*: If the cord is too tight to accomplish the other preferred steps, double-clamp and cut the cord between the clamps at the neck before the infant's body is born.

4. If planning a two-step process (assuming the cord has not been clamped and cut): While waiting for restitution, external rotation, and the next contraction, the midwife or assistant may wipe fluid from the neonate's face, nose, and mouth with a soft, absorbent cloth but it is not necessary to stimulate the infant at this moment. Suctioning with a bulb syringe of the fetal nasal and oral passages is not necessary.[8,9]

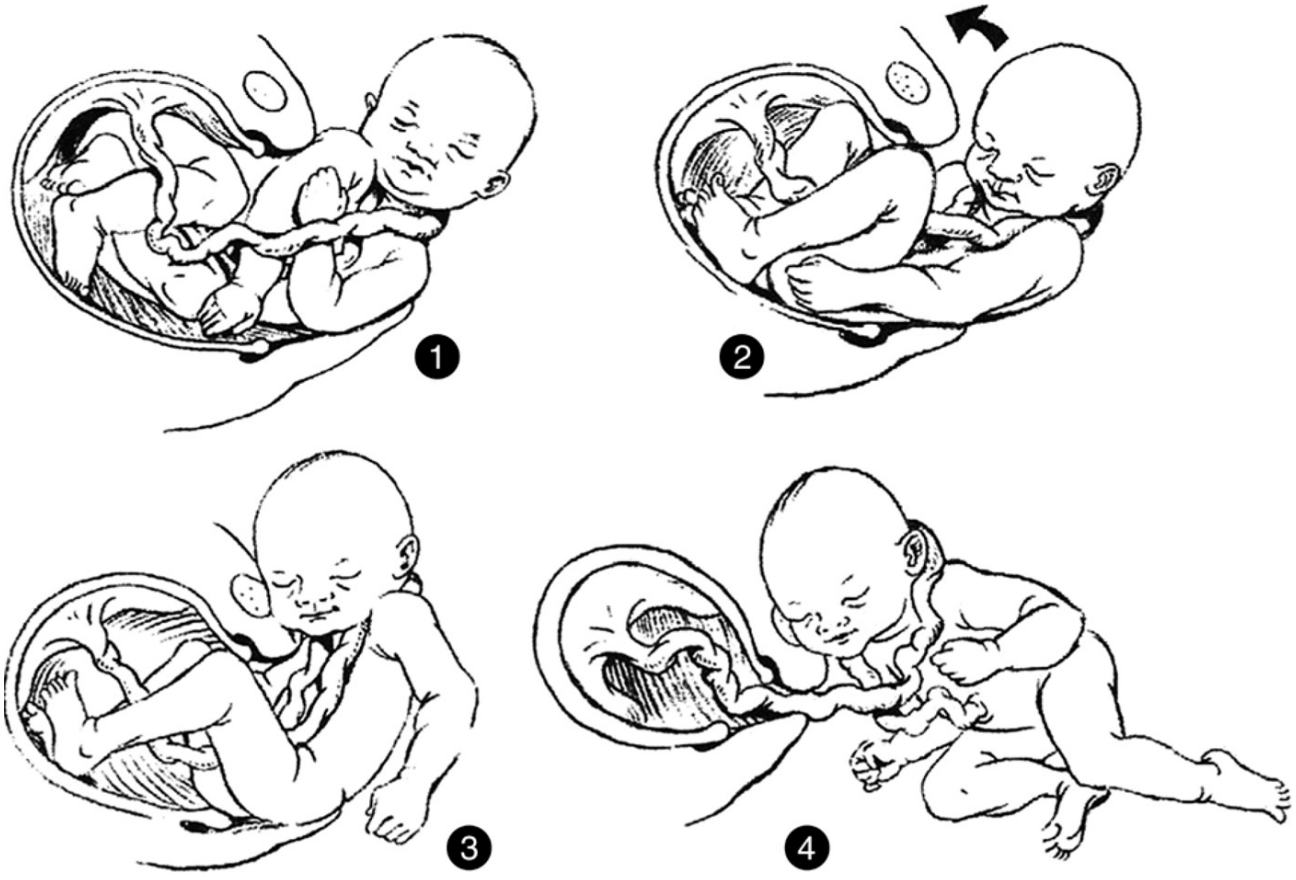


Figure 28A-2 The somersault maneuver involves holding the infant's head flexed and guiding it upward or sideways toward the pubic bone or thigh, so the baby does a "somersault," ending with the infant's feet toward the mother's knees and the head still at the perineum.

1. Once the nuchal cord is discovered, the anterior and posterior shoulders are slowly delivered under control without manipulating the cord.
2. As the shoulders are delivered, the head is flexed so that the face of the baby is pushed toward the maternal thigh.
3. The newborn's head is kept next to the perineum while the body is delivered and "somersaults" out.
4. The umbilical cord is then unwrapped, and the usual management ensues.

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   a. Wait for the next contraction and for restitution and external rotation to occur, which will be evident when the occiput rotates 90°.
   b. If there is a need to expedite the birth (e.g., the cord has been clamped and cut), rotation of the shoulders into the anterior–posterior position to facilitate restitution and external rotation can be facilitated by using a modified Rubin's maneuver if needed. Insert and place two fingers over the scapula of the anterior shoulder, and two fingers against the clavicle of the posterior shoulder. Firmly move the infant's head into the oblique diameter of the outlet.

5. Facilitate birth of the shoulders.
   a. Place one hand on each side of the head over the parietal and cheek bones so that the midwife's *fingers point toward the fetal face* and nose, as depicted in **Figure 28A-3**. The midwife's little fingers are closest to the woman's perineum and the thumb is farthest away from the perineum. This is the step that will assure all subsequent maneuvers will result in maintaining a secure hold on the newborn. Remember, *"Pinkies to the perineum."*
      i. For a fetus in the left occiput transverse (LOT) position, the fetus will be facing the woman's right leg, the midwife's left hand will be the bottom hand underneath the fetal head, and the midwife's right hand will be the hand on top of the fetal head.
      ii. For a fetus in the right occiput transverse (ROT) position, the fetus will be facing the woman's left leg, the midwife's right hand will be the bottom hand underneath the head, and the midwife's left hand will be the hand on top of the head.
      iii. Remember, "*Fetus faces right, the midwife's right hand is on top*" (**Figure 28A-3**).
   b. Assist the woman to give birth to the anterior shoulder. Avoid gripping the fetus's neck by keeping the fingers straight so the hands are flat on the sides of the head. During the next contraction, while the woman pushes, exert gentle, downward pressure on the head until the top of the anterior shoulder and axilla can be seen beneath the symphysis pubis. Maintain parallel alignment between the head and the

fetal spine during this process. Pulling down on the head alone laterally flexes the fetal head toward the posterior shoulder. This widens the angle between the neck and anterior shoulder, which can cause undue stretch and injure the infant's brachial plexus.

c. Once the anterior shoulder and axilla is visible, apply gentle upward pressure on the fetal head with both hands, lifting the infant's head following the natural arc of the curve of Carus toward the woman's abdomen. Avoid laterally flexing the infant's neck during this maneuver as well. Some midwives suggest that the perineum can be protected during the birth of the posterior shoulder by sliding the bottom hand over the shoulder and keeping the infant's arms and hands close to the body during the birth.

d. As the posterior shoulder is emerging, pivot the bottom hand under the fetal head, forming a C-shape cup with that hand facing the fetus so that the palmar side of the hand is facing the woman's perineum. Loosely grasp the infant's chest and back between the thumb and fingers. The head will be supported by the midwife's forearm in this position. If the midwife's hands have been placed properly during birth of the head, the midwife's thumb will come to rest near the scapula and the fingers will lay across the infant's thorax with the infant's neck and head resting gently between the thumb and fingers in the webbing between the thumb and fingers of the bottom hand. This is the first secure grip that will be maintained. With the bottom hand in this position, a forceful push will not result in the midwife losing a secure grip on the body. This bottom hand positioning also stabilizes the neck and head. The thumb and fingers are directed down toward the center of the infant's body which prevents circling the neck.

e. Slide the hand that was on the top of the fetal head down the infant's back; grasp one or both thighs, using this hand. This is the second secure grip that will be maintained.

6. Complete the final phase of birth. As the body of the infant emerges, the newborn can be placed immediately onto the woman's abdomen, pivoted to rest on the midwife's forearm facing the midwife, or handed to another clinician for assessment.

a. To place the infant immediately on the woman's abdomen: Using the bottom hand that is supporting the head by cupping the chest and scapula, move the infant in a smooth arc from over the perineum onto the mother's abdomen. Position the infant laterally on the woman's abdomen with the head and face slightly lower than the body to facilitate drainage of oral and nasal fluids.

i. While moving the infant onto the woman's abdomen, confirm that there is no tension on the umbilical cord.

ii. Maintain both secure grips on the newborn until the woman is safely holding her infant.

b. To hand the newborn to another clinician: Pivot the hand cupping the infant's chest and scapula in one of two ways:

i. Keeping the thumb and fingers around the newborn's shoulders, extend a finger to support the occiput and stabilize the neck. The newborn's shoulders are in the palm of the hand, and the thumb and remaining fingers are *resting*—not pressing

—against the sides of the neck. The top hand continues its grasp around legs during this maneuver.

ii. Alternatively, the bottom hand can slide up toward the head and grasp the two parietal bones between the thumb and fingers, with the occiput resting in the palm of the hand. With this maneuver, the neck is stabilized because the scapula rests on the bottom of the midwife's palm (**Figure 28A-4**).

iii. The newborn's hips and legs are then tucked securely between the midwife's bottom arm and body at waist level. The newborn's back is supported on the midwife's lower arm. The newborn's head is held firmly, in a slightly dependent and lateral position to encourage postural drainage. This is the third secure grip that can be maintained regardless of the woman's position. An initial assessment should be conducted at this time. The midwife now has one hand (the original top hand) free to clamp and cut the umbilical cord when necessary.



Figure 28A-3 Placement of hands for birth of the body.



Figure 28A-4 Stabilize the neck while preparing to hand the newborn to another provider.

Hand Skills for Birth of a Fetus in the Occiput Posterior Position

The same hand maneuvers used to facilitate birth for the woman whose fetus is in the occiput anterior (OA) position are applicable for the fetus in the occiput posterior (OP) position, with one exception: The direction of pressure exerted to maintain flexion of the head is maintained by upward pressure instead of downward pressure because the occiput is closer to the rectum than it is to the symphysis. Once the biparietal diameters are visible, releasing the upward pressure on the occiput, in a controlled manner, controls extension.

This technique is also used for the fetus in a face presentation. In a face presentation, the mentum (chin) will be under the symphysis and the rest of the head will be born by flexion of the neck. A face presentation that rotates so the mentum is posterior will not deliver vaginally because the neck is too short. Once the head is out, the remaining hand maneuvers are the same as described previously.

Hand Skills for Birth When the Woman Is in a Hands-and-Knees Position

While the mechanisms of labor are not changed by a maternal hands-and-knees position, the manner in which the infant emerges appears reversed and requires variations in the direction of support and positioning of hands as a result. Visibility and access to effect maneuvers are enhanced in this position. However, while the woman can hear the midwife, she is not able to see the midwife when in this position. As a result, anticipatory guidance to the woman about what to expect, and where she will need to position herself to receive the newborn, should be communicated prior to birth if possible. In addition, there may be increased stress on the anterior labia and on the periurethral area when the woman assumes a hands-and-knees

position for birth. **Figure 28A-5** shows a birth when the woman is in a hands-and-knees position and the fetus is in an occiput anterior position.



Figure 28A-5 Birth of a fetus with a compound anterior arm while the woman is in a hands-and-knees position.

Photograph courtesy of Tekoa L. King, CNM, MPH.

1. As the head emerges, maintain flexion by directing pressure on the occiput in the direction of the anus.
2. Allow the head to extend gradually, maintaining a steady but gentle counterpressure on the vertex. Maintaining flexion until the occiput passes under the symphysis pubis, and extension begins, will reduce the possibility of perineal trauma.
3. It is possible to support the perineum with the other hand alone or with a warm moist towel. Many women appreciate moist heat on the rectal area as the head crowns and extends. If stool is present, cover the anus with a towel to prevent stool from coming into contact with the fetus.
4. The hand maneuvers involved in checking for a nuchal cord, and watching for restitution and external rotation are the same as the maneuvers for birth when the woman is in any other position. In the hands-and-knees maternal position, the hand checking for the presence of a nuchal cord will be palm up and sliding back under the infant's head.
5. To effect birth of the shoulders: Some practitioners guide the anterior shoulder under the symphysis first just as though the woman were in a dorsal position; others reverse the

order and deliver the shoulder nearest the woman's sacrum first.

6. As the upper half of the infant is born, follow the curve of Carus and hold the arms close against the baby's body to protect the perineum and anterior structures.
7. The newborn can then be passed to the woman through her legs, keeping a secure hold on the infant until the woman has a firm grasp on her newborn. The midwife then moves to face the woman and helps her to sit down. It is important to observe for tension on the umbilical cord if this maneuver is selected.
8. Alternatively, the woman can carefully lift one leg, with the newborn and cord being passed under her leg as she completes a turn to sit down facing the midwife. The newborn can then be placed on her abdomen.
9. Immediately after birth, many women will naturally start to sit back. If the woman sits before passage of the newborn to her arms, she may sit on the cord or newborn. To prevent this problem, the midwife instructs the woman and others assisting with the birth on what to expect both prior to and during the birth process.

Hand Maneuvers for Birth of the Infant When the Woman Is in a Deep Squatting or Supported Squat Position

Upright and squatting positions during the second stage of labor and/or birth are associated with a shorter duration of the second stage of labor, fewer abnormal fetal heart rate patterns, and reduced severity of maternal pain. These positions are also associated with an increase in the incidence of second-degree perineal lacerations and more frequent estimated blood loss of more than 500 mL. A squatting position enlarges pelvic diameters and gravity may aid fetal descent. Depending on placement of the woman's legs, this position can tighten the transverse tension on the perineum.[10-12]

To facilitate visibility, access, and control when the woman is in a squatting position, the midwife sits on a low stool or the floor in front of the woman. In a deep squat, the woman's legs are bent and abducted, with weight bearing distributed between her legs and feet. This position brings the perineum close to the surface she is squatting on, so the infant emerges just inches above the surface. Moreover, the curve of Carus is such that the natural arc of the infant's path is into the woman's arms as an extension of the arc.

In the supported squatting position, the woman's buttocks can be partially supported by the edge of a broken birthing bed, a birthing chair, or a stool. The woman may use a squatting bar or handles on the stool, which then distributes her weight bearing between her hands, buttocks, legs, and feet. The woman may also be supported in a semi-squat position by another person. This person may stand against a wall for back support, holding the woman under her arms and across her chest. Both people bend their legs and squat gently, while the woman bears down with support from behind. It is helpful to place a pillow on the floor between the pair's legs so that there is a place to slide down onto and sit after the birth. There is usually ample space between the perineum and floor for assisting with birth in a supported squat. The perineum may be less taut when the woman is in a supported squatting position compared to the deep squatting position.

The following hand maneuvers are performed to support the woman giving birth in a

squatting position:

1. As the vertex appears at the vaginal introitus, flexion of the crowning vertex should be maintained with the palmar side of the fingers and hand. Perineal support can be provided in the same manner described previously for other positions, but is done by feel—not by sight—in this position.
2. It is important to watch the introitus while the head extends and is born, paying attention to the labial borders. When the vertex emerges with the woman in a squatting position, the pressure exerted on the introitus is distributed equally between the labia and the perineum. Lacerations along the labial borders are more common but tend to be shallow if extension of the head is controlled.
3. Using the maneuvers described previously, the newborn may be delivered to the maternal abdomen, where the woman can easily grasp and hold her baby if she is supported.
4. The hand maneuvers involved in checking for a nuchal cord, wiping the infant's head, watching for restitution and external rotation, and delivering the anterior and posterior shoulders and body are the same as described previously.

References

1. Aasheim V, Nilsen ABV, Reinar LM, Lukasse M. Perineal techniques during the second stage of labour for reducing perineal trauma. *Cochrane Database Syst Rev.* 2017;6:CD006672. doi:10.1002/14651858.CD006672.pub3.
2. Albers LL, Borders N. Minimizing genital tract trauma and related pain following spontaneous vaginal birth. *J Midwifery Womens Health*. 2007;52:246-253.
3. Albers LL. Midwifery care measures in the second stage of labor and reduction of genital tract trauma at birth: a randomized trial. *J Midwifery Womens Health*. 2005;50:365-372.
4. Kopas ML. A review of evidence-based practices for management of the second stage of labor. *J Midwifery Womens Health*. 2014:59:264-276.
5. Bulchandani S, Watts E, Sucharitha A, Yates D, Ismail KM. Manual perineal support at the time of childbirth: a systematic review and meta-analysis. *BJOG*. 2015;122:1157-1165.
6. Schorn MN, Blanco JD. Management of the nuchal cord. *J Nurse Midwifery.* 1991;36:131-132.
7. Mercer JS, Skovgaard RL, Peareara-Eaves J, Bowman TA. Nuchal cord management and nurse-midwifery practice. *J Midwifery Womens Health*. 2005;50:373-379.
8. Keleher J, Bhat R, Salas AA, et al. Oronasopharyngeal suction versus wiping of the mouth and nose at birth: a randomised equivalency trial. *Lancet.* 2013;382:326-330.
9. Foster JP, Dawson JA, Davis PG, Dahlen HG. Routine oro/nasopharyngeal suction versus no suction at birth. *Cochrane Database Syst Rev.* 2017;4:CD010332. doi:10.1002/14651858.CD010332.pub2.
10. Golay J, Vedam S. The squatting position for second stage labor: effects on the evaluation and progress of labor and on maternal and fetal well-being. *Birth*. 1993;20(2):73-78.
11. Moraloglu O, Kansu-Celik H, Tasci Y, et al. The influence of different maternal pushing positions on birth outcomes at the second stage of labor in nulliparous women. *J Matern Fetal Neonat Med*. 2017:30(2):247-249.
12. Gupta JK, Hofmeyr GJ, Smyth RMD. Position in the second stage of labour for women without epidural anaesthesia. *Cochrane Database Syst Rev.* 2004;1:CD002006. doi:10.1002/14651858.CD002006.pub2.

APPENDIX

28B

Umbilical Cord-Clamping at Birth

DEBRA A. ERICKSON-OWENS AND JUDITH S. MERCER

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Introduction

A delay in clamping the umbilical cord results in a placental transfusion that supplies the newborn with a major source of iron-rich red blood cells, stem cells, and plasma. Placental transfusion continues for several minutes after birth if the umbilical cord is not clamped, and results in approximately 30% more blood volume and 50% more iron-rich blood cells being delivered to the newborn compared to immediate clamping of the umbilical cord.[1] Recent randomized controlled trials have demonstrated both short- and long-term benefits of delayed cord-clamping (DCC) for term and preterm newborns, without any adverse effects.[2] The focus of this appendix is on the use of DCC for term newborns.

Overview

The World Health Organization recommends DCC for up to 3 minutes.[3] The American College of Nurse-Midwives (ACNM) position statement (2014) on delayed umbilical cord-clamping recommends waiting at least 5 minutes before clamping this cord.[4] Immediate cord-clamping in term infants is usually defined as clamping and cutting the umbilical cord within 30 seconds after birth.[5] The definition of DCC varies but is often defined as clamping that occurs 1 to 3 minutes after birth.[3-7] A delay of 5 minutes or more is encouraged when the infant is held skin-to-skin (above the level of the placenta).[5,8] Benefits for preterm infants include improved hemodynamic stability resulting in less intraventricular hemorrhage, reduced rates of neonatal transfusion in the neonatal intensive care unit (NICU), higher blood volume and blood pressure, and less bronchopulmonary dysplasia.[2,9,10] Benefits for term infants include higher birth weights and increased early hematocrit levels, better iron stores in infancy along with less anemia, and more myelin in early-developing areas in the brain.[8]

Key Points

- Delay of cord-clamping substantively increases iron stores in early infancy. Inadequate iron stores in infancy may have an irreversible impact on the developing brain despite oral iron supplementation.
- Gravity influences the amount of placental transfusion the newborn receives. Placing the newborn skin-to-skin with the mother immediately after birth requires a longer delay of cord-clamping.
- Uterotonics can be safely administered before clamping the umbilical cord.
- Cord milking is a safe alternative to DCC when it is necessary to cut the cord prematurely.
- The concern that DCC can cause hyperbilirubinemia or symptomatic polycythemia is unsupported by the available research.
- In complex clinical situations, the management of DCC is inconsistent between types of birth settings.
- A change in cord-clamping practice requires collaboration between all types of providers who attend births.

Management Steps

Briefly review the benefits of placental transfusion with the laboring woman and her support persons. Explain that DCC at the time of birth is standard practice, unless the woman requests otherwise.

There are several issues to consider for the management of cord-clamping including: timing, position of the newborn, use of uterotonics, cord milking, the influence of the birth setting, and complex clinical situations

Timing

- Gravity affects the amount of placental transfusion that the newborn receives. Holding the newborn below the level of the placenta accelerates the transfusion, while holding the newborn above the level of the placental slows the transfusion. Holding the baby skin-to-skin requires a little longer delay.[4,5,11-13]
- Once the transfusion is complete, the umbilical cord will become pale, white, and flat and look obviously emptied, at which point the umbilical cord can be clamped and cut.[4,13] "Wait for white" is a mnemonic that is used to signal when the transfusion is complete.
- Cessation of palpable pulsations of umbilical cord vessels means only that the umbilical arteries have constricted; it does not indicate that it is time to clamp the cord. There is still more blood left behind in the placenta that will be transferred to the newborn during the third-stage uterine contractions.[12]

Position

- Place the newborn and woman skin-to-skin. Dry and cover the newborn with a warm blanket, and leave the umbilical cord intact for at least 5 minutes or until the placenta is ready to emerge. Only newborns with good tone and respiratory effort should go immediately onto the maternal abdomen.[5,12]
- For any woman who does not want skin-to-skin placement of her newborn, place the newborn on a clean pad, between the woman's legs, and wait for the transfusion to complete.[12]

Use of Uterotonics

- Uterotonics can be safely administered before clamping the umbilical cord.[7,14]
- The use of uterotonics may speed the rate of placental transfusion by increasing the strength and frequency of uterine contractions. A widely held misconception is that the newborn will be over-transfused; however, the evidence is clear that over-transfusion does not occur.[13]

Umbilical Cord Milking

When it is necessary to transfer the newborn to a warmer for resuscitation, an alternative procedure is to milk the umbilical cord.[5,12,13] While milking the cord does not provide as much blood to the newborn as DCC, it does provide the newborn with some additional blood and red cell volume.[4,5]

To milk (or strip) the cord, a portion of the cord nearest the introitus is grasped between the thumb and the forefinger and the cord is then compressed (milked) down the length of cord toward the newborn's umbilicus 3 to 5 times. Following a vaginal birth, start at the introitus; at time of cesarean birth, start near the insertion site on the placenta. Take about 2 seconds to move over the length of the cord; wait about 2 seconds for cord to refill after milking, and then milk again.[13,15]

Complex Clinical Situations

Tight Nuchal Cord

Approximately one-third of all newborns are born with a nuchal cord. Usually this is a nonemergent event. A tight nuchal cord is less common but can be associated with hypovolemia in the newborn. Keeping the cord intact is strongly encouraged and can be supported by performing the somersault maneuver (**Figure 28A-2**).[16] This can be followed by transitioning (or resuscitation) with an intact cord.[17]

Shoulder Dystocia

Hypovolemia frequently accompanies a shoulder dystocia. After a birth that is complicated by a shoulder dystocia, assess the newborn and use techniques such as DCC or cord milking to restore blood volume before clamping. If the shoulder dystocia is accompanied by a tight nuchal cord, avoid clamping before birth of the newborn's shoulders.[18]

Slow-to-Start Newborn Requiring Neonatal Resuscitation

If the newborn has poor tone or is "slow to start," a plan of action is needed to support a safe neonatal transition.[13,18] Providers in out-of-hospital settings usually resuscitate the newborn at the perineum while maintaining an intact cord.[19] The hospital setting is challenging in this respect, and the provider may or may not be able to resuscitate with an intact cord (except if part of a research study).

Resuscitation with an Intact Cord (Delayed Cord-Clamping)

The newborn can be placed on a clean pad at the perineum or held below the level of the placenta to accelerate placental transfusion.[5] According to the Neonatal Resuscitation Program (NRP) guidelines, breathing, heart rate, and tone need to be assessed. Heart rate can be assessed by lightly feeling the stump of the cord. The heart rate is often normal even when the newborn is not initially breathing. Resuscitation, as recommended by NRP, can then proceed. The midwife should check the heart rate again; if it has not improved, all the usual resuscitation maneuvers can be instituted at the perineum without detaching the newborn. Once the newborn is breathing and tone is regained, the newborn can be placed skin-to-skin with the woman. This method of resuscitation has been practiced safely for many years in out-of-hospital settings.

Resuscitation with Cord Milking

When DCC is not feasible, and the heart rate is low, the cord can be milked 3 to 5 times toward the newborn to accelerate transfer of blood volume before clamping.[5,13] The newborn can then be moved to the newborn warmer and the usual NRP resuscitation maneuvers can be instituted.

Umbilical Cord Blood Gas Sampling

Within the hospital setting, a cord blood gas sample may be required or be a component of a standard protocol. A provider usually double-clamps (and cuts) the cord immediately after birth to collect a section of umbilical cord that can be used to obtain blood samples from the umbilical artery and umbilical vein. However, Andersson et al. demonstrated a technique that supports cord gas sampling while maintaining an intact cord.[20] With this procedure, an assistant holds the newborn immediately after birth. The midwife then uses both hands to collect the umbilical cord blood gas samples. Using a tuberculin syringe(s) with a very small-

gauge needle (25 or 27 gauge), the sample is collected from an umbilical artery first and then the umbilical vein. A small 2 × 2 gauze pad is placed over the insertion site and very gentle pressure applied to stop the oozing. The DCC is maintained and, if appropriate, the newborn is placed skin-to-skin with the woman.

References

1. Yao AC, Moinian M, Lind J. Distribution of blood between infant and placenta after birth. *Lancet*. 1969;2(7626):871-873.
2. Rabe H, Diaz-Rossello JL, Duley L, Dowswell T. Effect of timing of umbilical cord clamping and other strategies to influence placental transfusion at preterm birth on maternal and infant outcomes. *Cochrane Database Syst Rev*. 2012;8:CD003248. doi:10.1002/14651858.CD003248.pub3.
3. World Health Organization. Guideline: delayed umbilical cord clamping for improved maternal and infant health and nutrition outcomes. 2014. Available at: http://apps.who.int/iris/bitstream/10665/148793/1/9789241508209_eng.pdf. Accessed December 11, 2017.
4. American College of Nurse-Midwives. Delayed cord clamping: position statement. 2014. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000290/Delayed-Umbilical-Cord-Clamping-May-2014.pdf. Accessed December 11, 2017.
5. Mercer JS, Erickson-Owens DA. Rethinking placental transfusion and cord clamping issues. *J Perinat Neonatal Nurs*. 2012;26(3):202-217; quiz 18-19.
6. Andersson O, Lindquist B, Lindgren M, Stjernqvist K, Domellöf M, Hellström-Westas L. Effect of delayed cord clamping on neurodevelopment at 4 years of age: a randomized clinical trial. *JAMA Pediatr*. 2015;169(7):631-638.
7. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on maternal and neonatal outcomes. *Cochrane Database Syst Rev*. 2013;7:CD004074. doi:10.1002/14651858.CD004074.pub3.
8. Mercer J. Delayed Cord Clamping and Infant Brain Study (IBS). Clinicaltrials.gov Identifier NCT01620008. 2012. Available at: https://clinicaltrials.gov/ct2/show/NCT01620008. Accessed December 11, 2017.
9. March MI, Hacker MR, Parson AW, Modest AM, de Veciana M. The effects of umbilical cord milking in extremely preterm infants: a randomized controlled trial. *J Perinatol*. 2013;33(10):763-767.
10. Katheria AC, Leone TA, Woelkers D, Garey DM, Rich W, Finer NN. The effects of umbilical cord milking on hemodynamics and neonatal outcomes in premature neonates. *J Pediatr*. 2014;164(5):1045-50.e1.
11. Yao AC, Lind J. Effect of gravity on placental transfusion. *Lancet*. 1969;2(7619):505-508.
12. Mercer JS, Erickson-Owens DA, Collins J, Barcelos MO, Parker AB, Padbury JF. Effects of delayed cord clamping on residual placental blood volume, hemoglobin and bilirubin levels in term infants: a randomized controlled trial. *J Perinatol*. 2017;37(3):260-264.
13. Katheria AC, Lakshminrusimha S, Rabe H, McAdams R, Mercer JS. Placental transfusion: a review. *J Perinatol*. 2017;37(2):105-111.
14. Vain N. Delayed cord clamping and use of oxytocin. Clinicaltrials.gov Identifier NCT02618499. 2015. Available at: https://clinicaltrials.gov/ct2/show/NCT02618499. Accessed December 11, 2017.
15. Erickson-Owens DA, Mercer JS, Oh W. Umbilical cord milking in term infants delivered by cesarean section: a randomized controlled trial. *J Perinatol*. 2012;32(8):580-584.
16. Mercer JS, Skovgaard RL, Peareara-Eaves J, Bowman TA. Nuchal cord management and nurse-midwifery practice. *J Midwifery Womens Health*. 2005;50(5):373-379.
17. Katheria AC, Brown MK, Rich W, Arnell K. Providing a placental transfusion in newborns who need resuscitation. *Front Pediatr*. 2017;5:1.
18. Mercer JS, Erickson-Owens DA. Is it time to rethink cord management when resuscitation is needed? *J Midwifery Womens Health*. 2014;59(6):635-644.
19. Fulton C, Stoll K, Thordarson D. Bedside resuscitation of newborns with an intact umbilical cord: experiences of midwives from British Columbia. *Midwifery*. 2016;34:42-46.
20. Andersson O, Hellstrom-Westas L, Andersson D, Clausen J, Domellof M. Effects of delayed compared with early umbilical cord clamping on maternal postpartum hemorrhage and cord blood gas sampling: a randomized trial. *Acta Obstet Gynecol Scand*. 2013;92(5):567-574.

APPENDIX

28C

Genital Tract Injury: Immediate Postpartum Inspection of the Vulva, Perineum, Vagina, and Cervix

BARBARA J. REALE

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Introduction

Inspection of the vulva, perineum, vagina, and cervix for the presence of lacerations or hematomas takes place immediately after the birth. During this inspection, the midwife identifies:

- The presence of any arterial bleeding that needs immediate repair
- The location and type of lacerations
- Whether the lacerations require repair[1,2]
- The presence or absence of a hematoma

Key Points

Pain Relief

Inspection of the vagina and cervix can be uncomfortable or painful. The first step is to determine whether a woman is bleeding heavily from a laceration. If so, hemostasis must be achieved prior to or simultaneously with analgesic considerations. Hemostasis may be achieved with manual pressure, application of hemostats, or suture.

Once hemostasis is achieved, the midwife must determine whether the woman needs an

analgesic for the inspection. This decision will depend both on how extensive the inspection is likely to be and what the woman's pain threshold is. If an extensive inspection or repair is likely, a woman may need an intravenous dose of an opiate such as fentanyl or morphine. If she has an epidural, the anesthesiologist should be made aware that an inspection is needed so that the epidural can be used to provide the necessary pain relief. If the woman has a patient-controlled epidural analgesia pump, she should be asked to press the button for another dose. A pudendal or local injection of anesthetic may be given as needed.

Adequate Lighting

Ensure that an adequate lighting source is available. Inadequate lighting can result in misidentification of a laceration or a prolonged inspection and repair. It may be necessary to move the woman to a different place or different room to ensure adequate visualization.

Positions for the Woman and the Provider

Position the woman in a manner that will maximize her comfort and also allow the midwife to conduct an adequate examination. For most inspections, having the woman assume a modified lithotomy position by reclining and flexing her knees and placing her feet on the bed or other surface is sufficient. However, if an upper vaginal vault or cervical laceration is suspected, positioning the woman in foot rests or stirrups may be preferable if available.

Visualization

The midwife will need to insert three or four fingers into the vagina to provide space to allow visualization of the cervix and possibly the apex of a vaginal tear. Alternatively, a Sims retractor may be inserted with pressure against the anterior vaginal wall for better visualization of the vagina. Actions to be taken include:

- Assessing whether the laceration is hemostatic or bleeding
- Identifying the exact location of the laceration so that it can be described in the woman's chart and to other providers who may be involved in the woman's postpartum care
- Determining which type of laceration is present (**Table 28-10**) and whether the laceration requires repair

Equipment

- Sterile gloves
- Sterile gauze pads (or lap sponge pads detectable by X ray)
- Tissue forceps
- Vaginal retractor (Sims)
- Two ring forceps
- Antiseptic solution

Procedure for Examination of the Vulva and Perineum

1. Inspection of the vulva and the perineum is achieved by using sterile, gloved fingers to gently separate the labia. A sterile cloth or gauze may be used to gently clean away blood or clots that obscure a laceration.
2. The periurethral, periclitoral, labial, fourchette, perineal, and rectal areas are completely visualized.
3. The edges of irregular lacerations may be visually approximated best if a tissue forceps is used to carry one side of the laceration to the other. This helps the midwife begin a plan for repair of the laceration.
4. If a third- or fourth-degree laceration is suspected, don an additional glove and insert the index finger into the rectum to assess the integrity of the rectal sphincter. With palmar side up, gently lift upward, to expose the full extent of the laceration. Remove this glove and discard it after the rectal examination.

Procedure for Thorough Examination of the Vaginal Vault

For the purpose of this procedure, the vaginal vault is divided into four sections: posterior, anterior, and two lateral walls. Each section is visualized to identify lacerations in the vaginal mucosa. A systematic approach to this examination ensures that all areas are quickly, yet thoroughly inspected. Gauze sponges should be counted prior to use.

1. A 4 × 4 gauze sponge folded in fourths and clamped with a long-length ring forceps can be used to clean away blood. A second ring forceps can be used to gently lift a prolapsed cervix, or a Sims retractor used to elevate the anterior wall, allowing an unobstructed view of the lateral, posterior, and anterior fornices of the vaginal vault.
2. To examine each section of vaginal wall, insert the full length of several fingers of one sterile, gloved hand palmar side down and exert pressure away from the area being examined. Once one area has been inspected, release the pressure and reposition the vaginal hand in the new area to be inspected before force is applied again.
3. Insert the ring forceps with the gauze sponge on it by sliding it down the top of the vaginal fingers. This maneuver minimizes the amount of gauze touching the vaginal walls, as the gauze is abrasive to these tissues. The gauze serves as a sponge to blot blood and other fluids from the exposed area, thus, facilitating visualization. If the gauze sponge becomes saturated, remove the ring forceps, dispose of the saturated gauze sponge, clamp on a clean one, and reinsert the ring forceps. During this procedure, the midwife should be careful to monitor use of gauze sponges so none is inadvertently left in the vagina. Dedicating an area on a delivery table or drape for saturated sponges to be counted is helpful.

Procedure for Inspection of the Cervix

An assistant, if one is available, will make this procedure easier and more efficient.

1. Insert three or four sterile, gloved fingers of the nondominant hand palmar side down along the posterior vaginal wall to the area just in front of the cervix. This hand is used to exert pressure to retract the posterior vaginal wall and facilitate visualization of the cervix.
2. Once the cervix is in view, using the dominant hand, grasp the anterior lip of the cervix with one of the ring forceps. The first forceps is used to help bring the anterior part of the cervix into view. The second ring forceps is used to grasp the posterior part of the cervix (**Figure 28C-1**).
3. Hold the handles of *both* ring forceps in the dominant hand. Pull on the forceps gently, if necessary, to bring the cervix more into view. Move the handles of the forceps toward one side of the perineum, thereby slightly pulling the cervix so the lateral apex of the cervix can be visualized.
4. Visually inspect the area of the cervix between the two ring forceps on one side.
5. If necessary, confirm the visual inspection by using the index finger of the vaginal hand to feel the edge of the cervix.
6. Repeat the procedure, moving the handles of the forceps toward the other side of the perineum to visualize and inspect the other lateral side of the cervix.
7. If the cervix is very patulous, all of the cervix may be difficult to visualize adequately between the ring forceps placed on the anterior and posterior lips of the cervix. In such a case, the entire circumference of the cervix can be visualized by placing one ring forceps on the anterior lip of the cervix and the second forceps next to it. Release the first forceps and place it on the other side of the second. Continue to leap-frog or "walk" the ring forceps around the cervix. This technique can also be used when the posterior lip of the cervix cannot be located easily.
8. If there are no cervical lacerations, remove the ring forceps and the vaginal hand.
9. If the cervix is bleeding from a cervical laceration, use the forceps to exert direct pressure on the laceration to decrease or stop the bleeding, prior to repair. This method can help to significantly reduce the amount of blood lost.

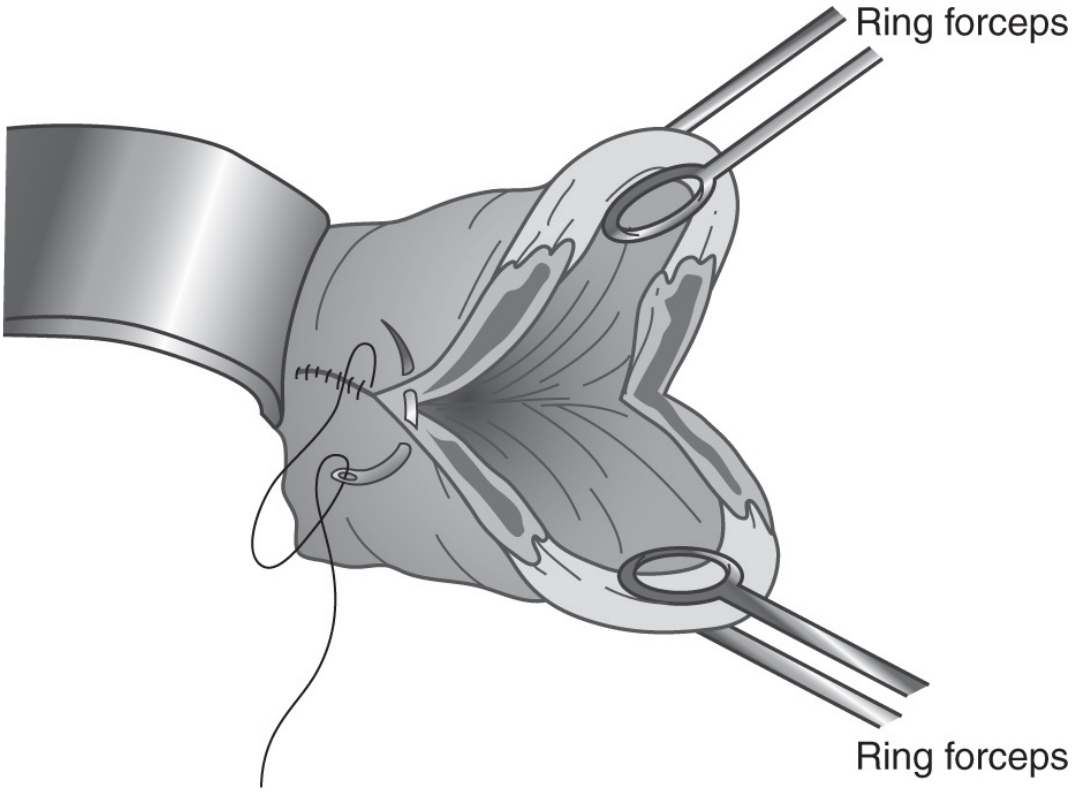


Figure 28C-1 Visualization of the cervix.

Hematomas

During pregnancy, the vagina and vulva have rich and full vascular beds that are at risk for experiencing trauma during childbirth. When one of these vessels is torn or damaged, a hematoma can occur and enlarge very quickly.[3] Most hematomas develop from vessels torn during a vaginal or perineal laceration, but vessels can also be injured withoutobvious laceration. Hematomas develop rapidly and are extremely painful for women who do not have regional anesthesia. A classic presentation is pain that is unrelieved or worsens despite administration of pain medication. The most common symptom in women who have epidural anesthesia is rectal pressure or rectal pain. Hematomas are reviewed in more detail in the *Third Stage of Labor* and *Postpartum Complications* chapters.

Because hematomas often occur secondary to a tear in an artery, significant blood can be lost and the woman can develop signs of shock. The most common locations where hematomas develop are the vulva, the vagina, and the retroperitoneum.[4]

Vulvar hematomas develop secondary to damage to one of the branches of the pudendal artery. Because the Colles' fascia and urogenital diaphragm or anal fascia prevent their expansion, these hematomas grow superficially toward the skin and loose subcutaneous tissue. They present as swellings in the labia majora.

Vaginal hematomas are the result of injury to the uterine artery. These vessels are not surrounded by fascia, and bleeding into the paravaginal area can be extensive.

Retroperitoneal hematomas that extend between the folds of the broad ligament are rarer than the other two types. These hematomas are caused by injury to the branches of the internal

iliac artery and are more common following uterine rupture or as an extension of a paravaginal hematoma. Hemorrhage can be significant, and hemodynamic instability can develop quite quickly. Bleeding can be either arterial or venous. Women who are hemodynamically stable usually have a venous bleed; conversely, women who become hemodynamically unstable are more likely to have an arterial bleed.

References

1. Cioffi JM, Swain J, Arundell F. The decision to suture after childbirth: cues, related factors, knowledge and experience used by midwives. *Midwifery.* 2010;26(2):246-255.
2. Cioffi JM, Arundell F, Swain J. Clinical decision-making for repair of spontaneous childbirth trauma: validation of cues and related factors. *J Midwifery Womens Health*. 2009;54(1):65-72.
3. Mirza FG, Gaddipati S. Obstetric emergencies. *Semin Perinatol.* 2009;33(2):97-103.
4. Elharmeel SMA, Chaudhary Y, Tan S, Scheermeyer E, Hanafy A, van Driel ML. Surgical repair of spontaneous perineal tears that occur during childbirth versus no intervention. *Cochrane Database Syst Rev.* 2011;8:CD008534. doi:10.1002/14651858.CD008534.pub2.
5. Mirza FG, Gaddipati S. Obstetric emergencies. *Semin Perinatol.* 2009;33:97-103.

APPENDIX

28D

Local Infiltration for Laceration Repair

BARBARA J. REALE AND TEKOA L. KING

The editors acknowledge Lisa Kane Low, who contributed to this appendix in the previous edition.

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Local infiltration of an anesthetic solution can be placed in the perineum just prior to birth before cutting an episiotomy. In addition, it can be administered specifically in the areas or structures involved that need to be sutured after a spontaneous laceration occurs.

Anesthetic Agent for Local Injection

The medication recommended for local infiltration of tissue that requires sutures is 1% lidocaine hydrochloride (Xylocaine) 10 mg/mL. Lidocaine is available either with or without added epinephrine. Epinephrine produces vasoconstriction—an action that helps retain the drug in the tissue being sutured longer, thereby extending the period of anesthesia.

Pharmacokinetic studies have found that lidocaine is detected in maternal plasma within a minute of injection and that placental transfer occurs quite rapidly.[1] It is theorized that potential adverse effects of epinephrine on the uterine blood flow and fetus could be significant. Because of this risk, lidocaine with added epinephrine should *not* be used prior to birth. In contrast, lidocaine *with* added epinephrine is used for local infiltration after birth because less overall drug is needed and the anesthetic effects last longer.[2]

Lidocaine Toxicity

Lidocaine toxicity can cause light-headedness, dizziness, tinnitus, abnormal taste, facial tingling, circumoral numbness, and confusion. Severe toxicity results in loss of consciousness and respiratory arrest. If lidocaine without epinephrine is injected intravascularly, the first signs may be hypotension and bradycardia. If lidocaine with epinephrine is injected intravascularly, the first signs may be hypertension and tachycardia.

Toxicity is usually the result of intravenous injection, rapid absorption from a very vascular area such as mucous membranes, or overdose. Thus, a maximum dose that is safe to use is specified for lidocaine. Toxic levels of lidocaine can be administered if accidental intravascular injection occurs or if the total volume of medication injected is not monitored. Toxic levels are based on the weight of the woman, the concentration of the medication, and an identified safe range for administration.

Case reports of toxic levels of lidocaine in the fetus have been described secondary to direct injection into the fetal scalp. The risk of this unanticipated event is more likely if the fetus is in an occiput posterior (OP) position or if large amounts of lidocaine are injected more than 4 minutes prior to the birth.

Lidocaine Dose and Pharmacokinetics

- The recommended dose of lidocaine 1% *without* added epinephrine is typically 5 to 10 mL (50–100 mg). Peak plasma concentrations occur in 3 to 15 minutes, and the onset of action may take up to 3 minutes. It is important to wait until the anesthetic has taken effect. The duration of action is approximately 30 to 60 minutes.[2-4]
- The maximum dose of lidocaine 1% is based on case studies of toxicity, older animal studies' and manufacturers' recommendations.[2,3]
  - Lidocaine 1% *without* epinephrine: 30 mL (300 mg).[2,3] In Europe, the maximum recommended dose is 20 mL (200 mg), so publications may report either 200 mg or 300 mg.[2]
  - Lidocaine 1% *with added* epinephrine: 50 mL (500 mg).

Equipment for Local Infiltration

Equipment used to perform local infiltration is listed in **Table 28D-1**.

Table 28D-1	Equipment Necessary for Local Infiltration of Anesthetic Solution
Sterile gloves	
1% lidocaine (10 mg/mL)	
A 10- to 20-mL syringe	
Needles:	
18-gauge, 1½-inch needle to draw up the anesthetic from its original container	
22-gauge, 1½-inch needle for episiotomy, perineal or vaginal laceration, or a vaginal sulcus tear	
25-gauge, 1-inch needle for periurethral or clitoral lacerations	

Local Infiltration Prior to Cutting an Episiotomy

Local infiltration prior to cutting an episiotomy is done when the perineal body has been

thinned and flattened considerably by the crowning of the fetal head or pressure from the presenting fetal part. At this point, there is typically little anteroposterior tissue and only one plane that requires anesthesia. While the thin and flattened perineal body allows for easier administration of anesthetic and identification of the area where infiltration has occurred, it also presents the challenge of potentially piercing the posterior perineal wall and inserting local anesthetic uselessly into the vagina or dangerously into the fetus. To avoid this risk, the following procedure is recommended.

General Principles

1. Always inject slowly.
2. Aspirate regularly during injection to check for blood return and to avoid intravascular injection.

Procedure

1. Place two fingers of the nondominant hand 3–4 cm into the vagina between the fetal presenting part and the flattened perineal body. Bring a small amount of pressure onto the posterior perineal surface to create a space between the area where the local infiltration is going to occur and the presenting fetal part.
2. Either of two techniques for local infiltration of the perineal body may be used.
   a. Insert the needle at the center of the fourchette and direct the needle straight down the midplane toward the rectum. After aspirating, up to a maximum of 10 mL (100 mg) of lidocaine is injected as the needle is slowly withdrawn. The desired effect is to create a large, visible, and palpable perpendicular wheal approximately 2–2.5 cm wide and extending from top to bottom in the center of the perineal body (**Figure 28D-1**).
   b. Insert the needle point at the center of the fourchette and aspirate. The needle is then introduced in a manner that creates an upside-down fan shape centrally in the perineum body while the medication is being injected. This technique provides more anesthesia in a wider section of the perineum, but takes more time to perform and involves pulling the needle back up and repositioning it. The goal is to avoid multiple sticks by drawing the needle up but not removing it before re-angling and moving the needle down the perineum in the next direction (**Figure 28D-2**).
3. If the plan is to cut a mediolateral episiotomy, the direction of the needle and resultant wheal should not be placed centrally but instead directed to the side, following the intended path of the mediolateral cut that will be made.

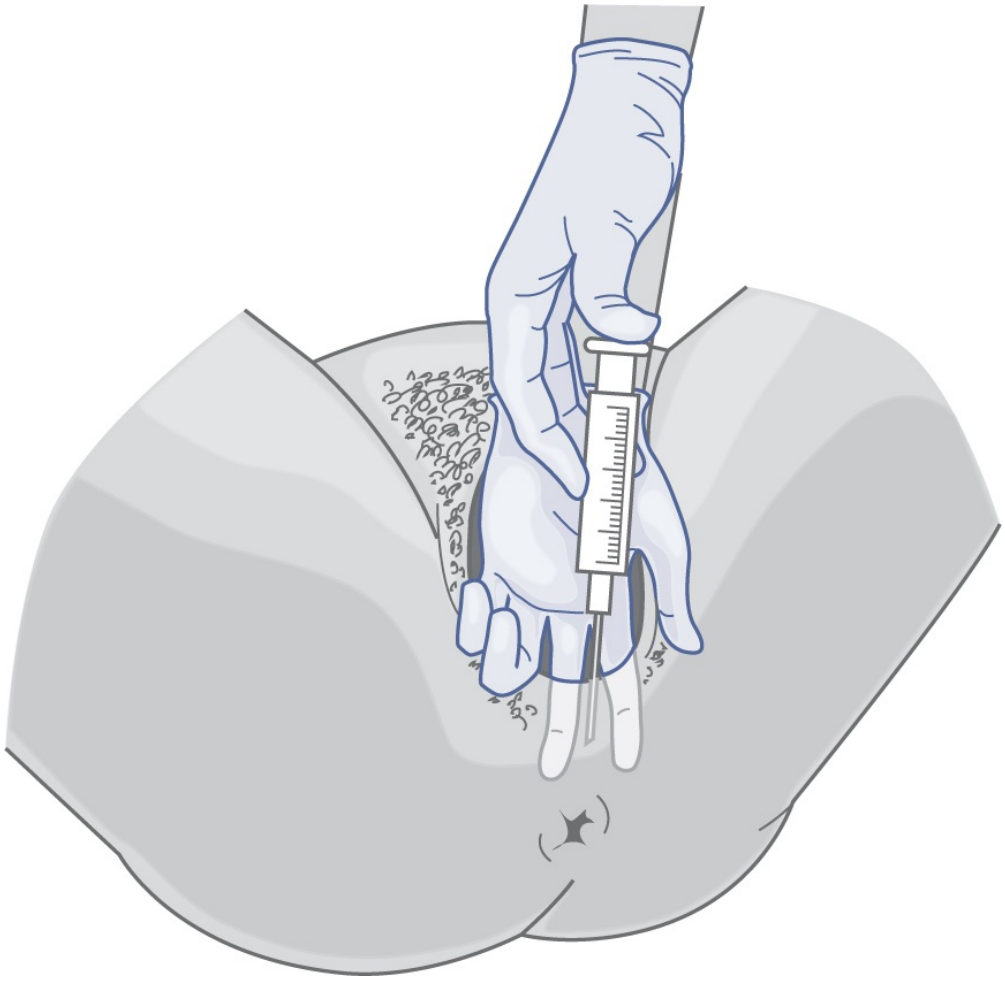


Figure 28D-1 Technique 1 for local infiltration for a midline episiotomy.

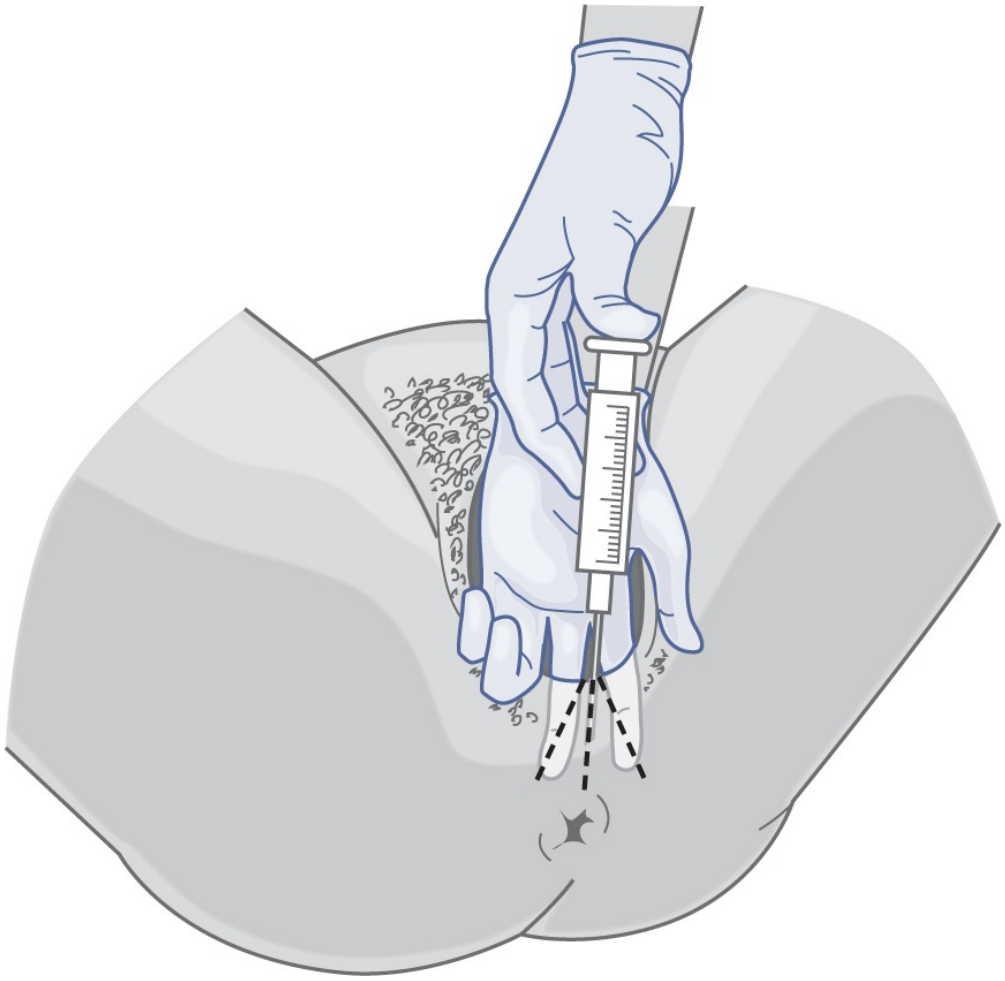


Figure 28D-2 Technique 2 for local infiltration for a midline episiotomy.

Local Infiltration for Repair After Birth

Local infiltration for repair of a laceration may be needed if pre-birth anesthesia has dissipated, a post-birth pudendal block has failed, or local infiltration is the mode of choice. The primary disadvantage of local infiltration for repair is that it distorts the tissue, thereby making tissue approximation potentially more challenging.

Procedure

1. Identify all tissue that requires anesthesia and repair.
2. Place the needle tip at the end or corner of the laceration nearest the posterior fourchette, and insert it for the length of the wound, reaching the areas where the suture needle will be either entering or exiting the tissue.
3. Aspirate to confirm that the needle is not in a vessel.
4. Inject the anesthetic as the needle is withdrawn along the line of the wound edge.
5. Discontinue injecting medication when the needle point is withdrawn to where it entered the wound, without fully withdrawing the needle.

6. Redirect the needle along the open wound in any tissue just under the wound that will require suturing.
7. Inject anesthetic as the needle is withdrawn back to the point of entrance.
8. Repeat this process until the area that needs suturing has been anesthetized.
9. Once the area requiring anesthesia is fully infiltrated, withdraw the needle. Another, noncontiguous area can then be anesthetized following the same process.

References

1. Phillipson EH, Kuhnert BR, Syracuse CD. Maternal, fetal, and neonatal lidocaine levels following local perineal infiltration. *Am J Obstet Gynecol*. 1984;149(4):403-407.
2. Rosenberg PH, Veering BT, Urmey WF. Maximum recommended doses of local anesthetics: a multifactorial concept. *Reg Anesth Pain Med*. 2004;29:564-575.
3. Lidocaine hydrochloride: drug summary. *Prescribers Digital Reference*. Available at: http://www.pdr.net/drug-summary/Lidocaine-Viscous-Solution-lidocaine-hydrochloride-2696. Accessed December 13, 2017.
4. Colacioppo PM, Riesco MLG. Effectiveness of local anaesthetics with and without vasoconstrictors for perineal repair during spontaneous delivery: double blinded randomised controlled trial. *Midwifery*. 2009;25:88-95.

APPENDIX

28E

Pudendal Nerve Block for Vaginal Birth

DEBORAH ANDERSON

The editors acknowledge Barbara J Reale for contributions to this appendix.
The editors acknowledge Lisa Kane Low, who contributed to this appendix in the previous edition.

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Pudendal nerve block is a regional anesthetic technique that anesthetizes the pudendal nerve, resulting in a reduction or elimination of pain perception in the muscles and tissue innervated by the pudendal nerve. Pudendal nerve block offers the following advantages:

- Primarily used to achieve analgesia for operative vaginal birth and/or repair of genital tract lacerations. It may also be used for late second stage pain relief with spontaneous vaginal birth, or for women who do not have adequate or satisfactory pain relief during perineal repair following instillation of local anesthesia to the perineum.
- Provides anesthesia to the vulva and anus, including the clitoris, labia majora, labia minora, and perineal body, thereby offering broader anesthesia coverage than is accomplished with local infiltration. Pudendal anesthesia does not abolish the pain of uterine contractions and cervical dilation, nor is it effective for repair of lacerations of the upper vagina and cervix, or for manual exploration of the uterine cavity.
- Does not alter the strength or frequency of uterine contractions, but may decrease the urge to push during second stage labor.
- Confers minimal risk to the parturient, fetus, or newborn when administered correctly, using the appropriate medication within the safe dosage range.

Anesthetic Agent for Pudendal Block

The anesthetic agent commonly used in pudendal nerve block is 1% (10 mg/mL) lidocaine hydrochloride (Xylocaine) without added epinephrine.[1] Lidocaine is rapidly absorbed, crosses the placenta, and is detectable in maternal venous and fetal scalp blood within 5 minutes after

injection, with peak levels attained between 10 and 20 minutes after injection.[2] A maximum block may take up to 20 minutes to achieve because the pudendal nerve is a large peripheral nerve with a smaller surface area to volume ratio, and a myelin sheath. Hence, the speed of onset of anesthetic effect is delayed, and time to maximum block takes longer to achieve than the usual 5 minutes for subcutaneous infiltration of the perineum.[3]

The duration of anesthesia depends on the success of the pudendal block and on the medication used. The average duration of action of lidocaine is 30 to 60 minutes. Safe dosing of 1% lidocaine is 3mg/kg (204 mg in a woman who is 150 lbs) to 4.5mg/kg (300 mg in a woman who is 150 lbs). The maximum recommended dose is 300 mg.[3,4]

Equipment for Pudendal Block

The equipment needed to perform a pudendal block is listed in **Table 28E-1**. The needle guide, procedure needle, and syringe used for this technique are shown in **Figure 28E-1**.[1]

Table 28E-1	Equipment for Pudendal Block
Sterile gloves	
Prepackaged pudendal nerve block set, which includes:	
Needle guide (e.g. Iowa trumpet)	
12- to 15-cm (5- to 6-inch) 20- or 22-gauge procedure needle	
20-mL syringe	
18- to 20-gauge needle for drawing up the anesthetic	
Local anesthetic: lidocaine (Xylocaine) 1%	

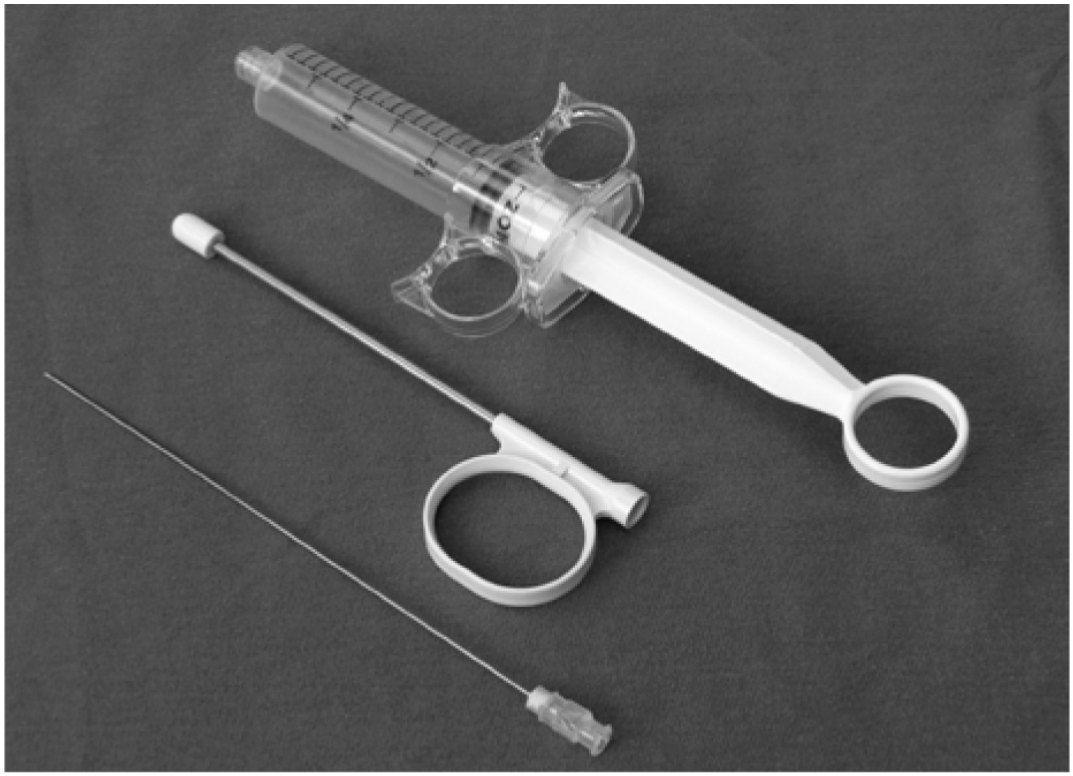


Figure 28E-1 Pudendal nerve block set: needle, guide, and syringe.

Reproduced with permission from Anderson D. Pudendal nerve block for vaginal birth. *J Midwifery Womens Health.* 2014;59(6):651-659.[1] © 2014, with permission from Wiley.

Pudendal Block for Perineal Anesthesia

The pudendal nerves are the primary source of sensory innervation to the lower vagina, vulva, perineum and anus, and provide motor innervation to the perineal muscles, urethral sphincter, and external anal sphincter. Infiltration of local anesthesia around the trunk of the pudendal nerve via pudendal nerve block inhibits nerve transmission and results in pain relief to these areas. To target the pudendal nerve effectively and safely, an understanding of the location of the pudendal nerve and internal pudendal artery is essential.

Anatomic Location of Pudendal Nerve

The pudendal nerve derives from sacral roots S2-S4. It courses between the piriformis and coccygeus muscles, and exits the pelvis through the greater sciatic foramen. It then courses on the posterolateral surface of the sacrospinous ligament, medial to the internal pudendal vessels that lie on the posterior aspect of the ischial spine. Inferior to the ischial spine, the nerve travels along the lateral pelvic wall through Alcock's canal and divides into three branches (inferior rectal nerve, perineal nerve, and dorsal nerve of the clitoris).[5]

Key Principles for Achieving an Effective Pudendal Nerve Block

1. To target the pudendal nerve effectively and safely, an understanding of the location of the pudendal nerve and internal pudendal artery is essential.
2. Understand the key anatomic landmarks used to locate the optimal point of lidocaine infiltration to most effectively target the pudendal nerve.
3. Allow for a sufficient amount of time to effect adequate analgesia.

Contraindications to Pudendal Nerve Block

Contraindications to pudendal nerve block are coagulation disorders, infection in the vagina, allergy to local anesthetic, and women who are not able to cope with the procedure, or who decline pudendal nerve block.

Procedure for Administration of a Pudendal Nerve Block[1]

1. Explain the risks and benefits of pudendal nerve block and obtain consent.
2. Draw up 20 mL of 1% lidocaine into the syringe, attach the pudendal needle and clear the air from the syringe and needle. Place the needle with attached syringe into the guide.
3. Palpate the ischial spine.
   a. If the location of the ischial spine is unclear, palpate the sacrospinous ligament, and follow it laterally to its attachment point on the ischial spine. The sacrospinous ligament is a firm band of tissue that travels between the sacrococcygeal joint and the ischial spine. If the location of the sacrospinous ligament is unclear, palpate the sacrococcygeal joint (the junction between the coccyx and the sacrum), find the attached sacrospinous ligament, and walk your fingers laterally along the ligament to the ischial spine.
4. With the needle tip withdrawn into the guide, insert the guide with attached syringe into the vagina. Place the tip of the guide on the sacrospinous ligament, 10 mm medial to the ischial spine (**Figure 28E-2**).
   a. To prevent inadvertent needle sticks, make sure the needle tip is withdrawn into the guide during insertion into the vagina.
   b. To perform a right-sided block, place the right index finger on the ischial spine. Place the middle finger on the adjacent sacrospinous ligament. Holding the guide and attached syringe in the left hand, glide it along the right hand to the point between the tips of the index and middle finger.
   c. For a left-sided block, place the left index finger on the ischial spine and hold the guide and attached syringe in the right hand.
   d. The correct location of the guide directs the needle tip to the optimal point of infiltration (10 mm medial and 10-15 mm posterior to the ischial spine). The needle guide allows only 10-15 mm of needle to protrude from its end (distance varies with manufacturer of the guide).
   e. Placing the pudendal nerve block while the fetal head is deep in the pelvis can be challenging as the procedure often stimulates an urge to push. Placing it between contractions and encouraging the parturient to breathe deep between contractions

may help her to overcome the urge to bear down during the procedure.

5. Stabilize the guide.
   a. A gentle forward pressure of the index finger on the ischial spine may help to stabilize it.
6. Advance the needle through the guide and through the sacrospinous ligament to the optimal point of infiltration.
   a. Caution! When the needle is fully advanced through the guide, it will protrude from the tip of the guide. To prevent needle stick injuries, keep the needle retracted into the guide until you are ready to advance the needle through the ligament. Make sure that your fingers are not in front of the tip of the guide before advancing the needle.
7. Aspirate.
   a. This is a necessary safety measure to prevent intravascular administration. Aspiration is performed before all subsequent injections.
8. Inject 10 mL of lidocaine 1% into the first side.
   a. Alert the woman to the forthcoming injection.
9. Repeat the procedure on the contralateral side.

2. Allow sufficient time to achieve an effective block.
   a. The maximum analgesic effect may take up to 20 minutes to achieve. To test for an effective block, stroke the skin adjacent to the anus. An effective block will prevent the reflexive constriction of the anal sphincter. Repeat on the opposite side.
   b. If the block is ineffective or unilateral, 5 additional mL of 1% lidocaine may be administered. Keep track of the total dose administered and do not exceed 30mL (300 mg) of 1% lidocaine.
   c. In some cases, such as a need for an urgent birth where it may not be possible to wait up to 20 minutes to achieve maximum anesthetic effect, practitioners may first infiltrate the perineum with local anesthetic, then administer the pudendal nerve block. This will provide a more rapid anesthetic block to the perineum and help with pain relief if the effect of the pudendal nerve block is not complete before the procedure.

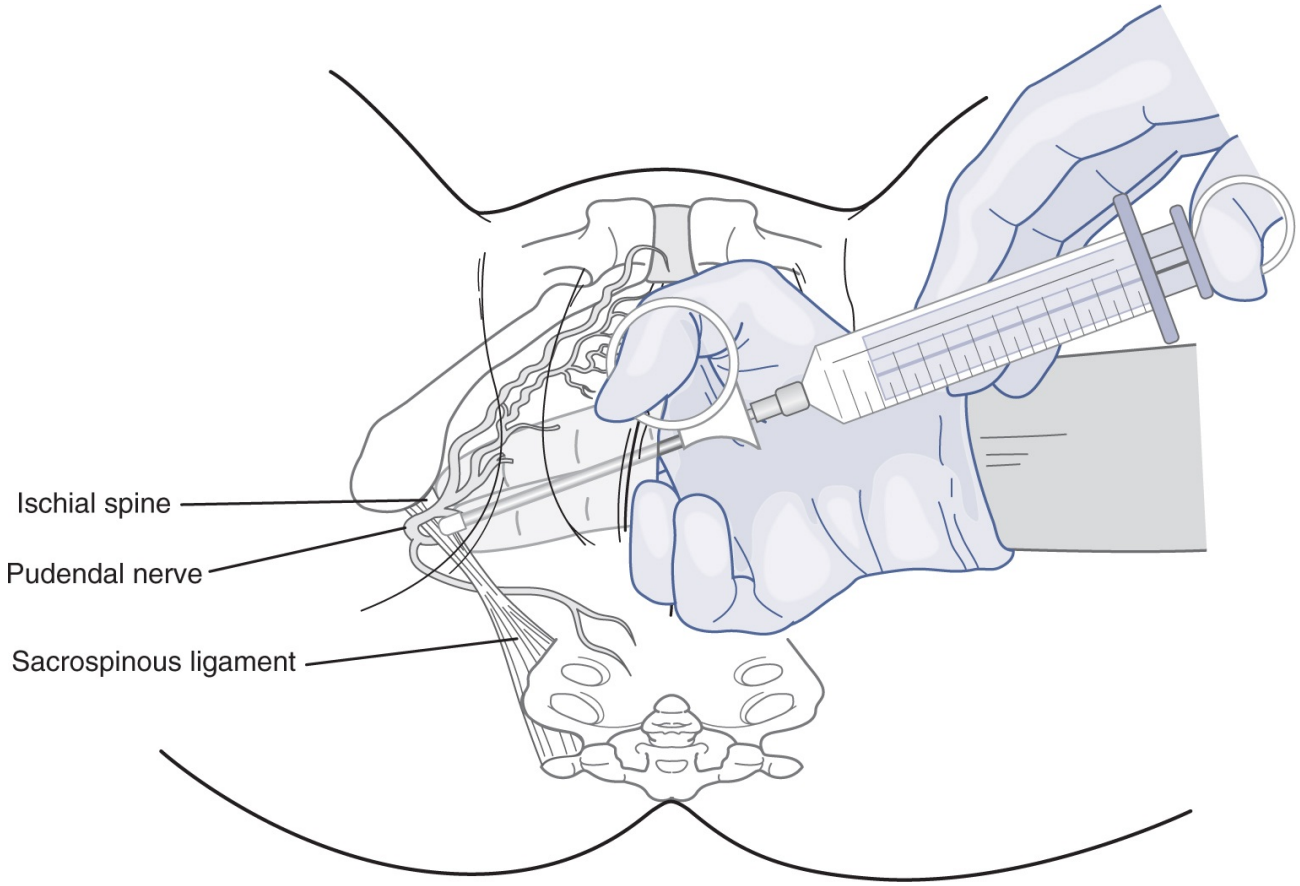


Figure 28E-2 Pudendal block procedure.

Variations of Pudendal Block Procedure: Multiple-Injection Approach

Variations of the standard procedure have been published and are summarized here.[1,4,6] The single-injection approach may minimize the risks of pudendal nerve block complications; the multiple-injection approach may decrease the chance of inadequate block. However, no studies have been conducted comparing the safety or efficacy of the single and multiple injection techniques.

Variation 1[1,6]

Follow the procedure outlined above until the point of administration of 1% lidocaine. Instead of a single injection approach:

- Inject 3 mL of 1% lidocaine into the sacrospinous ligament 10 mm medial and posterior to ischial spine.
- Advance the needle through the sacrospinous ligament and inject the remaining 7 mL.
- Aspirate before all injections.
- Repeat on other side.

Variation 2[1,4]

Follow the procedure outlined above until the point of administration of 1% lidocaine. Instead of a single injection approach:

- Inject 1 mL of 1% lidocaine into the mucosa and create a mucosal wheal just beneath the tip of the ischial spine.
- Advance the needle to the sacrospinous ligament and infiltrate 3 mL into the sacrospinous ligament.
- Advance the needle through the sacrospinous ligament and inject another 3 mL of 1% lidocaine posterior to the ligament.
- Withdraw the needle into the guide, place it just above the ischial spine, and administer an additional 3mL of 1% lidocaine.
- Aspirate before all injections.
- Repeat on other side.

Outcomes Associated with Pudendal Block

Approximately 3% to 50% of pudendal blocks will be ineffective on one or both sides.[1] Ford has suggested that poor knowledge and variable performance of pudendal nerve block may be contributing to suboptimal effectiveness.[3] Nevertheless, a pudendal block provides excellent analgesia when it is effective.

Complications of pudendal nerve block are rare but can be serious. These include hematoma with or without infection, localized infection (retropsoas and subgluteal abscess), infection at the site of injection, systemic toxicity from inadvertent intravascular administration or excessive doses of anesthetic, temporary ischial paresthesia, sacral neuropathy, and inadvertent needle injection of anesthetic into the fetal scalp, mother, or provider.[6-9]

References

1. Anderson D. Pudendal nerve block for vaginal birth. *J Midwifery Womens Health*. 2014;59(6):651-659.
2. Zador G, Lindmark G, Nilsson BA. Pudendal block in normal vaginal deliveries: clinical efficacy, lidocaine concentrations in maternal and foetal blood, foetal and maternal acid-base values and influence on uterine activity. *Acta Obstet Gynecol Scand Suppl*. 1974;(34):51-64.
3. Ford JM, Owen DJ, Coughlin LB, Byrd LM. A critique of current practice of transvaginal pudendal nerve blocks: A prospective audit of understanding and clinical practice. *J Obstet Gynaecol*. 2013;33(5):463-465.
4. Cunningham FG. Obstetrical anesthesia. In: Cunningham FG, ed. *William's Obstetrics*. 24th ed. New York: McGraw-Hill; 2014:504-522.
5. Standring S, ed. *Gray's Anatomy: The Anatomical Basis of Clinical Practice*. 39th ed. Edinburgh: Elsevier Churchill Livingstone; 2005.
6. Kurzel RB, Au AH, Rooholamini SA. Retroperitoneal hematoma as a complication of pudendal block. diagnosis made by computed tomography. *West J Med*. 1996;164(6):523-525.
7. Svancarek W, Chirino O, Schaefer G Jr, Blythe JG. Retropsoas and subgluteal abscesses following paracervical and pudendal anesthesia. *JAMA*. 1977;237(9):892-894.
8. Langhoff-Roos J, Lindmark G. Analgesia and maternal side effects of pudendal block at delivery. A comparison of three local anesthetics. *Acta Obstet Gynecol Scand*. 1985;64(3):269-272.
9. Pages H, de la Gastine B, Quedru-Aboane J, Guillemin MG, Lelong-Boulouard V, Guillois B. Lidocaine intoxication in newborn following maternal pudendal anesthesia: Report of three cases. *J Gynecol Obstet Biol Reprod (Paris)*. 2008;37(4):415-418.

APPENDIX

28F

Repair of Genital Tract Lacerations and Episiotomy

BARBARA J. REALE

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Approximately 85% to 90% of nulliparous women and 60% of multiparous women sustain some genital tract laceration during vaginal birth.[1,2] These lacerations can be either minor or extensive. Lacerations to the perineum often need repair, whereas lacerations to other parts of the genitalia usually need repair only if bleeding is present. In addition, an episiotomy—a surgical incision of the posterior perineal body—can be performed to expedite birth when indicated. Repair of genital tract lacerations is an essential midwifery skill.

This appendix reviews general principles for suturing, different stitches used in repairing genital tract lacerations, and techniques for repairing common genital tract lacerations sustained at birth. A brief review of current controversies regarding different repair techniques is included. The last section reviews the technique for cutting an episiotomy. Genital tract lacerations are categorized according to the extent of tissue and muscle involvement, as noted in Table 28-10. Lacerations are spontaneous events; in contrast, an episiotomy is intentional.

Figure 28F-1 illustrates the general anatomy of a perineal laceration. The anatomy that can be disrupted includes the skin, vaginal mucosa, hymenal ring, fourchette, bulbocavernosus muscle, transverse perineal muscle, and—if the laceration extends into the rectum—the external rectal sphincter, internal rectal sphincter, and rectal mucosa.

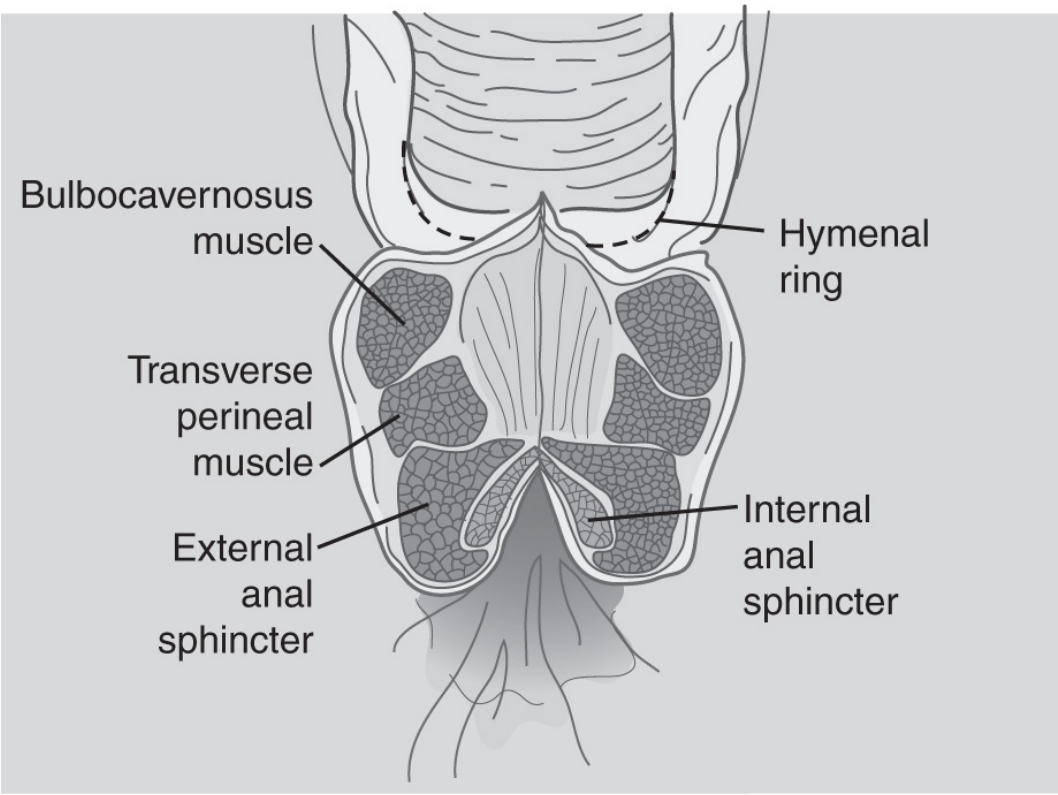


Figure 28F-1 Anatomy of a perineal laceration or midline episiotomy.

Equipment for Repair of Genital Tract Trauma

- Sharp scissors
- Sterile gauze sponges (4 × 4)
- Needle holder (also termed a needle driver)
- Tissue forceps with and without teeth
- Suture
- 1% lidocaine *without* epinephrine
- Syringe and 18-gauge needle for lidocaine
- 22-gauge needle for local infiltration

Optional as needed:

- Iowa trumpet and needle for pudendal block
- Sims retractor

Third- or fourth-degree laceration:

- Gelpi retractors
- Allis clamps

General Principles and Suture Technique

Repair of a genital tract laceration or episiotomy accomplishes three goals: (1) hemostasis, (2) return to functional integrity, and (3) cosmetic repair. Multiple sources exist electronically on the Internet that demonstrate various aspects of perineal laceration repair for learners who need a visual demonstration. Active practice using models and under direct supervision of an experienced midwife or physician cannot be underestimated.

Achieving Hemostasis

Hemostasis must be achieved first before a wound is closed. Blood clots increase the inflammatory reaction and serve as a culture media for bacteria. Large clots can cause tissue necrosis by creating pressure on adjoining tissues.

Hemostasis is accomplished by tying off bleeding vessels, by eliminating dead space, and by using techniques that minimize trauma to the tissue. The following techniques also aid in promoting hemostasis:

1. Gently probe the depth of the wound before suturing to identify the true depth that needs repair beneath the mucosal layer.
2. Start any line of suture at least 1 cm beyond the apex of the wound to include any retracted blood vessels.
3. Use stitch types that facilitate hemostasis in the most vascular areas, such as the continuous locked or blanket stitch.
4. Use an atraumatic (tapered) needle instead of a cutting needle.

Promoting Wound Healing

Wound healing involves three phases: inflammation, proliferation, and maturation.[3] Although healthy women tend to heal well, it is critical that aseptic technique is maintained to avoid the risk of infection. Asepsis may require changing gloves following the birth and redraping the area if necessary to create a sterile field; using an extra sterile glove over the sterile glove(s) already on an examining hand when checking the rectum and discarding the extra glove immediately after use; and careful placement of suture on the sterile field as the process of the repair is conducted.

During the process of wound repair, the following techniques promote wound healing:

- Use the smallest suture, suture gauge, and needle size that are needed.
- Use an atraumatic round needle.
- Precise approximation of tissues: Place sutures once where desired. Do not remove and replace sutures to obtain a more perfect result.
- Do not place sutures too close together or make them too tight. Tissue strangulation can result from too-tight sutures.
- Avoid repeated unnecessary sponging, prodding, and probing of the wound. Dab—do not wipe—to avoid creating additional trauma to the tissue.

- Avoid use of instruments that crush tissue.
- Remove blood clots and debris before closure.
- Establish visible hemostasis before closure to avoid formation of hematomas.
- Ensure closure obliterates all dead space.

Selection of Suture Material

Types of suture include standard synthetic suture, rapidly absorbing synthetic suture, glycerol-impregnated catgut suture, standard synthetic suture with rapidly absorbing synthetic monofilament suture, and catgut.[4] Synthetic sutures are preferred, as they are associated with less pain in the immediate postpartum period.[3,5] Synthetic suture is also less likely to require removal and resuturing when compared to catgut.[3,5] Catgut has the additional disadvantage of causing a marked tissue inflammatory reaction that produces edema, which places tension on the sutures and may cause tissue necrosis. Ultimately scarring can result, which results in a weaker functional repair. The two most common absorbable synthetic suture types are polyglycolic acid (Dexon, Davis & Geck Ltd., United Kingdom) and polyglactin 910 (Vicryl, Ethicon Ltd., United Kingdom).

Selection of Suture Gauge and Needle Type

Muscle heals by deposition of scar tissue and requires a stronger suture than does other tissue, which regenerates itself. The larger the gauge number, the finer the suture (e.g., 4-0 is finer than 6-0 or 8-0). The suture gauges used for various conditions are listed in **Table 28F-1**.

Table 28F-1 **Suture Gauge and Common Sites for Use of Suture Material**

	Gauge	Common Repair Sites	Usual Types of Stitches
Thinner ↓ Thicker	4-0	Clitoral lacerations	Continuous, interrupted
		Shallow periurethral lacerations	Continuous, interrupted
		Anterior wall of rectum (repair of fourth-degree laceration)	Continuous, locked
	3-0	Vaginal mucosa	Continuous, locked
		Deep periurethral lacerations	Subcutaneous, continuous
		Pelvic muscles that are not extensive	Deep interrupted
	2-0	External anal sphincter	Continuous, deep interrupted
		Cervical lacerations	Continuous, deep interrupted
		Lateral wall lacerations	Continuous, deep interrupted
		Deep pelvic muscles	Deep interrupted

The use of an atraumatic or tapered round needle is recommended instead of a cutting needle to reduce tissue trauma and the risk of increased hemostasis (**Figure 28F-2**). A cutting needle is triangular in shape, and each edge has a sharp surface; in contrast, the atraumatic needle is a round needle with a tapered point. The latter moves through soft tissues more easily than a

cutting needle and is less likely to lacerate a blood vessel. The anatomy of a needle is identified in **Figure 28F-3**.

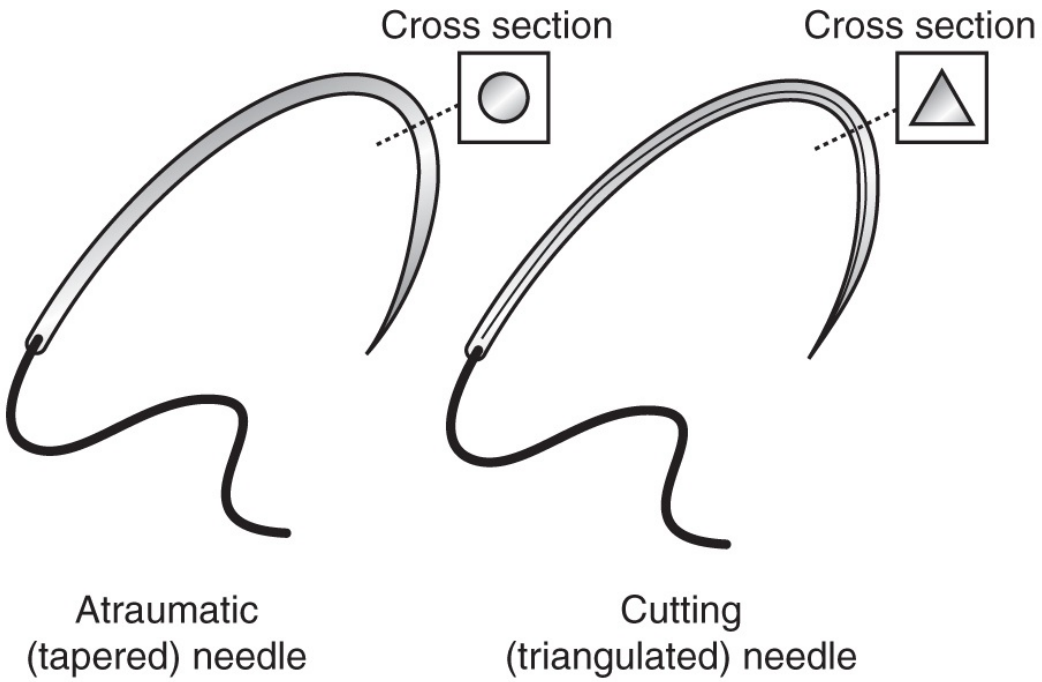


Figure 28F-2 Types of curved needles usually attached to suture material (swaged).

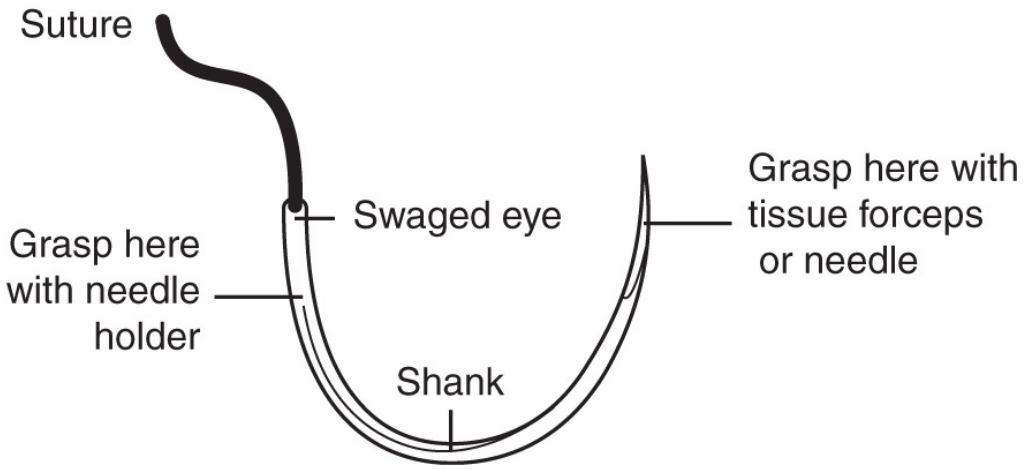


Figure 28F-3 Anatomy of a needle.

Suturing

Key Points

1. General suturing techniques and appropriate sutures for different tissue types should be reviewed before beginning the repair. The tissue should be carefully visualized and

landmarks identified.

2. Excess suture can be gathered up in the palm of the hand holding the needle holder, so as to prevent the length of suture from dangling over or dragging across areas of contamination.
3. The needle is clamped by the tip of the needle holder so that the plane of the curve of the needle is perpendicular to the plane of the needle holder. Clamp the needle just proximal to the junction of the needle and the suture.
4. Do not clamp the needle holder at the junction of the needle with the suture, or on the suture, to avoid weakening the suture.
5. The needle holder is controlled by wrist action. It can be held so that the thumb holes are in the palm of the hand and secured by the ring and little fingers. With this positioning, the thumb and middle fingers are on either lateral side of the shaft, with the index finger on one side (usually the upper) of the broad side of the shaft (rather than placing fingers through the holes). Some midwives refer to this placement as "shaking hands" with the needle holder (**Figure 28F-4)**.
6. Because of the need for universal precautions, the midwife always should use forceps to grasp or "pick up" the point of the needle, once it has come through tissue, to guide it to completion of the stitch.
7. The needle should be guided through tissue along the angle of its curve. When following the circle of the needle, rather than pulling it straight up, the suture will lie in the smallest space possible, a track left by the needle. If the needle is pulled straight, its arced shape will enlarge the space it creates in the tissue and the suture will lie in a larger track, which may result in a less stable stitch, or collection of blood and fluids. Turning the wrist to have the same angle as the needle promotes smooth movement.
8. Suturing is begun by holding the needle with its curved midportion turned so the tissue can be entered with the point of the needle perpendicular to the tissue. Therefore, the hand holding the shaft of the needle holder will be positioned so that the back of the hand is up and then further pronated as the needle point is positioned for entry. As the needle enters the tissue, the wrist is rotated approximately 180° so that the needle follows the necessary directional path for exit, completing the stitch.
9. As the point of the needle emerges, it should be grasped either by tissue forceps (without teeth) or by releasing the needle holder from its original position and repositioning the needle holder to grasp the emerging point (**Figure 28F-5**). Tissue forceps always should be used when sewing deep to moderately deep wounds. In such a case, the amount of tissue is often large enough that it is impossible to release the needle holder and grasp the other end of the needle, without losing the needle in the tissue. When shallow stitches are being made, such as subcuticular stitches, the needle holder may be released and reclamped to the other end of the needle, to complete the stitch, without concern for the needle being retracted into the tissue. Do not grasp the point of the needle with fingers—doing so may cause a needle puncture.
10. Avoid an excessive number of needle punctures, an unnecessary number of stitches, placement of stitches in very close proximity, and misplacement of stitches that have to be removed. Excessive suture in the wound slows the healing process by causing an

inflammatory reaction to foreign material.



Figure 28F-4 One method for holding a needle holder.

Knots

A square knot is the basic knot used for repairing an episiotomy or a laceration (**Figure 28F-6**). A square knot results after a left-handed overhand knot is created, followed by a right-handed knot. Most experts advocate completing not only the square knot, but also one more action to ensure that the stitch does not come undone. This knot, which may be colloquially termed a “knot and a half,” is made in case either the first or third action becomes loose or the suture dissolves more rapidly than anticipated. In either case, the complete square knot would remain.

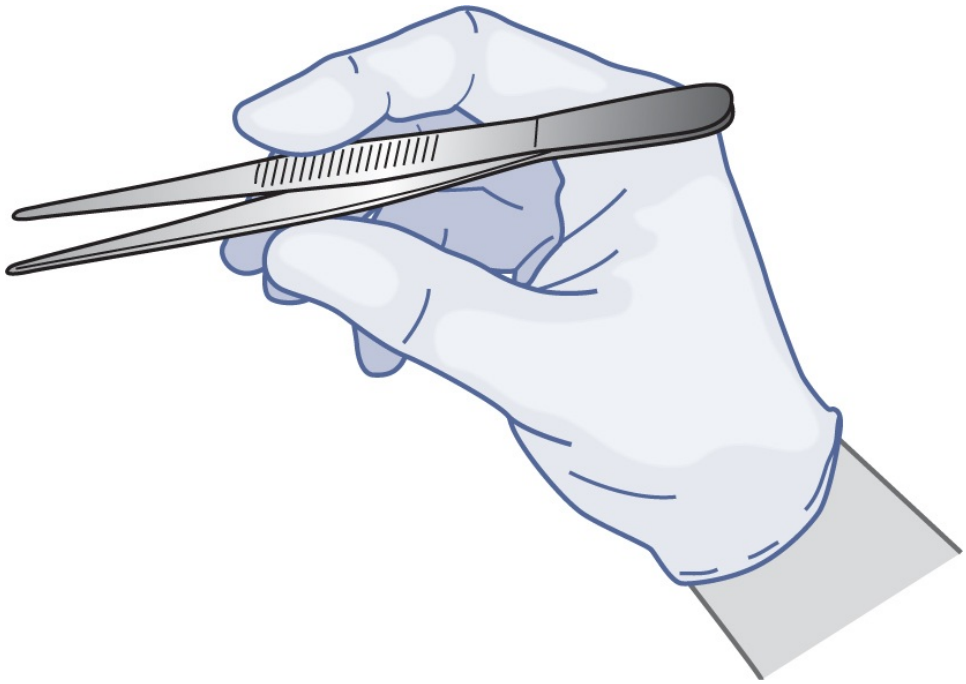


Figure 28F-5 Tissue (pickup) forceps without teeth.

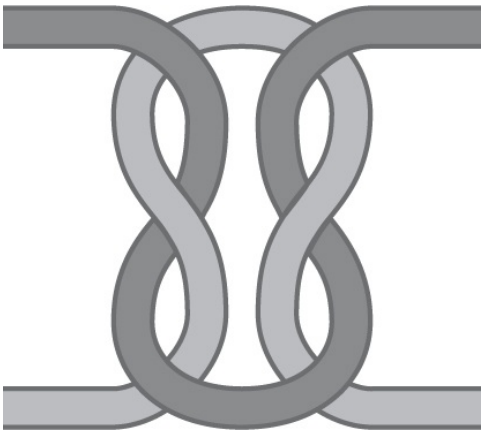


Figure 28F-6 Square knot.

A square knot can be tied by hand, by instrument, or by both, as all of these options can ensure that the knot is appropriately tied and placed flat against the tissue. Instrument ties are recommended, as they are safest for the provider. Another advantage of an instrument tie is that the knot can be tied with a short suture length, whereas a hand tie requires longer suture to complete tying the knot.

Attaining skill in tying knots requires practice. Multiple resources on this topic exist on the Internet, including a variety of digital recordings illustrating how to tie a knot by hand or instrument in real time (a topic beyond the scope of this appendix). Knot-tying boards are available, although not essential for practice.[6]

Key Points for Tying Knots

1. The knot should be as small as possible to minimize the use of excessive suture material; in turn, the ends should be as short as possible. Extra ties do not necessarily provide extra security, but they do provide additional and unwarranted suture material that can irritate healing tissue.
2. Friction between the strands, also called sawing, should be avoided as much as possible because it can weaken the integrity of the suture.
3. Crimping of the suture should be avoided except when grasping the free end of the suture during an instrument tie.
4. Sutures used simply to approximate tissue should not be tied with excessive tension because this condition may contribute to tissue strangulation.
5. As the knot is tied, tension should be as equal as possible on each step and the final tension should be made as nearly horizontal as possible. The midwife may need to change personal position to tie the most effective knot.

Types of Stitches

Challenges in any discussion of the types of stitches used in repairing genital tract lacerations include the existence of many, often minor, variations to a basic technique and multiple names for both the same stitch and those variations. The most commonly used stitches—continuous, continuous locked, interrupted, subcuticular, figure of eight, and crown—are illustrated in **Figure 28F-7**. The description of each of these techniques in this section usually assumes a right-handed practitioner; the direction should be reversed if suturing in a left-handed manner.

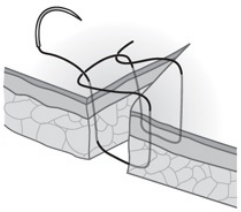
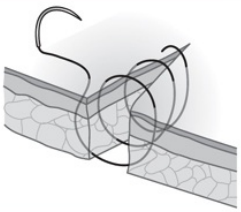
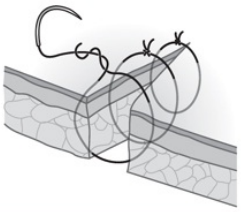
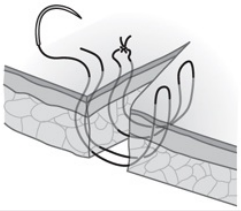
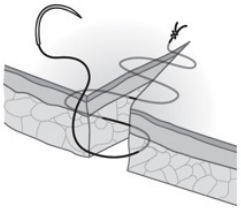
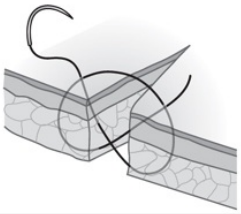
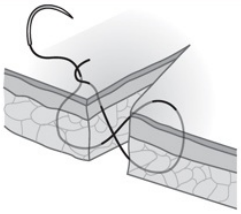
Suture	Alternative nomenclature	Illustration
Continuous locked	Blanket	
Continuous	Running	
Interrupted		
Double interrupted for third-degree laceration		
Subcuticular	Mattress	
Crown		
Figure of eight		

Figure 28F-7 Sutures.

Continuous Locked (Blanket) and Continuous (Running) Stitches

A continuous stitch is often called a running or “whip” stitch. However, each stitch can also be

locked to provide additional security. This locking is the differentiating factor between the two most common types of continuous stitches.

1. The needle is inserted into the right side of the wound and brought through the incision to emerge on the left side of the wound. The amount of tissue on each side should be approximately the same. Based on the amount of the tissue, it may be advisable to bring the point of the needle out into the midline of the wound to verify positioning before continuing to the left side.
2. An anchoring knot is tied on the left side of the stitch and one end is cut short.
    a. For a continuous locked (blanket) stitch, the point emerges in front of the previous stitch but under a loop or section of the suture. As the needle exits the tissue, the section of suture remains behind it. This maneuver locks the stitch.
    b. For a continuous (running) stitch, the needle point emerges in front of the previous stitch—*not* under a loop or section of the suture.
3. The needle and suture should be guided through the tissue, following the curve of the needle.
    a. A continuous locked (blanket) stitch is secured or locked along the left side of the wound line in right-handed suturing and locked along the right side of the wound in left-handed suturing.
    b. A continuous (running) stitch is made by repeating stitches from right to left (or left to right if left-handed) approximately 0.5 to 1 cm apart, with insertion and exit points being in parallel. The needle enters the tissue on the one side and carries the suture under and perpendicular to the line of the laceration across the laceration to the opposite side, where it exits into the vaginal mucosa. The suture is then carried from the exit point, diagonally across the uppermost surface of the wound to a reentry point 0.5 to 1 cm from the last entry point, and then repeats the traverse under the wound perpendicularly.

These steps should be repeated until the wound is closed.

Interrupted Suture

An interrupted stitch consists of a single stitch and knot. Both ends of the suture are cut short.

1. The needle is inserted into the side of the wound and brought through the wound to emerge on the other side of the wound, directly across from the origination. The suture should not be drawn completely to the end of the suture, as an end is needed for tying.
2. The amount of tissue picked up by the needle on each side of the laceration should be approximately the same, unless the depths of the sides of the wound are uneven (e.g., in a mediolateral episiotomy). If this is a deep stitch, with a large amount of tissue to bring together, it is advisable to bring the needle out in the midline of the wound and then take another stitch into the other side of the wound. This approach will verify positioning and ensure that the needle grasps the appropriate amount of tissue on each side.
3. Tie a square knot using both ends, by hand or with instruments, to secure the stitch. If the

interrupted stitch is used for deep muscle, the needle and suture should include the apex of the wound to avoid leaving any unsutured dead space behind the closed wound.

Separate interrupted stitches should be inserted as needed by following these steps.

Subcuticular (Mattress) Stitch

A subcuticular stitch is a buried horizontal stitch used for closing the subcuticular perineal skin during a laceration or episiotomy repair so that the suture is not visible on the skin.

1. An anchoring stitch is placed in the appropriate site going through the skin if necessary, a knot is tied, and one end is cut short. When subcuticular sutures are used to close the perineum during repair of a second-degree laceration, this anchoring stitch is not needed because the subcuticular stitches will follow a series of running continuous stitches.
2. Use of tissue forceps with teeth, to hold the skin and adjacent tissue stable while placing a stitch, may be helpful. Pick up the tissue ahead of where the needle will enter and exit.
3. Insert the needle on one side of the incision. Unlike with other stitches, this insertion is shallow—"one cell layer" deep, although it is difficult to ascertain that benchmark. The suture is placed immediately below the skin layer and will run parallel to the line of the wound under the skin.
4. Gently guide the suture through the tissue for approximately 0.5 cm in length.
5. Make the next stitch in the same manner on the opposite side of the wound. The entry point on this side should be directly across from the exit point of the preceding stitch on the opposite side of the wound. If exit and entry points are not directly across from each other, the skin will appear "bunched up." Careful placement will result in smooth, even closure.

These steps are repeated until the wound is closed.

Figure of Eight Stitch

A figure of eight stitch is used to provide hemostasis and tie off actively bleeding vessels. It is also used for securing the rectal sphincter muscle.

1. Start the stitch by entering the tissue on one side behind the bleeding vessel and bringing it out on the other side, also behind the bleeding vessel. This suture should not be tied or cut. An end for tying should remain loose.
2. Continuing with the same needle and suture, create a second, parallel stitch in front of the bleeding vessel, starting on the side in front of the vessel where the first stitch was placed and emerging again on the other side in front of the vessel.
3. Create another end for tying by cutting the needle off from the suture. The two ends of suture can be tied diagonally across the tissue surface, creating a figure of eight. The suture constricts the bleeding vessel within its configuration.

Crown Stitch

The crown stitch is used to reapproximate the bulbocavernosus (bulbospongiosus) muscle. This step restores the anatomy of the perineum, introitus, and labial junction at the mucocutaneous junction. When the bulbocavernosus muscle is torn, the ends usually retract superiorly and posteriorly. The crown stitch is placed in a manner that securely grasps each end, and brings both ends down and medial, to approximate them.

1. Begin by entering the tissue in an upper diagonal slant at the upper-left side of the wound line at the junction of the perineum and the vagina. Adequate space should be allowed away from the edge of the incision to enable subcutaneous continuous stitches and potentially a subcuticular closure over the completed crown stitch. Do not tie or cut the suture.
2. Direct the needle so that it follows a sequence of moving upward, then laterally, downward, and medially so that it exits in the same plane, but posterior to the original entry site. Experience is essential to perform this technique well because the amount of lateral thrust must be just enough to reach the retracted cut end of the bulbocavernosus muscle if possible. The more lateral tissue obtained, the more vascular the area and the higher the risk of a potential hematoma. Grasping the two ends of the suture a short distance from the tissue and gently pulling them can provide verification that the bulbocavernosus muscle has been included in the suture. If the bulbocavernosus muscle has been sutured, the entire left-side labia will be pulled.
3. Insert the needle into the tissue at the right side of the wound directly across from the exit point on the left side. Direct it in a manner that mirrors the action in Step 2. The correct placement through the bulbocavernosus muscle can be ascertained using the same technique as described in Step 2.
4. After cutting off the needle, the two ends of suture should be next to each other and are knotted, with the ends cut short. Care must be taken to only closely approximate the muscle in repairing it; pulling and suturing the two sides together too tightly may result in increased scar tissue and dyspareunia.

Repair of a Second-Degree Laceration or an Episiotomy

Following are the steps that are used to repair a second-degree laceration or episiotomy. As with several other midwifery skills, this is only one of several possible methods. This method includes both continuous and interrupted stitches. Research has shown that closures dependent only on interrupted suturing techniques are associated with more pain.[7]

Repair of midline and mediolateral episiotomies is essentially the same. However, in a mediolateral episiotomy, the medial aspect of the incision tends to retract more than the lateral aspect, indicating the need for special care to avoid the rectum when suturing. In addition, the stitches for this repair follow the incisional line and are performed at a slant, rather than straight across as done for repair of a midline episiotomy or laceration, with slightly more tissue on the lateral side than on the medial side. When completed, the stitches may be closer

than for a midline closure. Sometimes a laceration may occur at an angle, and the same comments may apply.

Key Points Prior to Repair

1. An aseptic field must be established and meticulously maintained. Establish this field by clearing away soiled materials, irrigating or cleansing soiled skin areas, and, if needed, placing clean or sterile drapes under the buttocks.
2. Good visualization of the area of repair is critical. Lighting and positioning may need to be adapted. If a woman is positioned using stirrups, the time should be limited to less than 90 minutes to decrease the risk of impaired circulation or thrombophlebitis.
3. Visualize the vulva, labia, vestibule, vaginal walls, cervix, and perineum to identify all sites of bleeding and tissue disruption prior to beginning the repair.
4. Identify and tie off any actively bleeding vessels with a figure of eight stitch prior to repairing the main laceration.
5. Identify the depth of the lacerations in all directions. Shallow lacerations usually do not include muscle, but occasionally the muscle is nicked. In these instances, where a muscle is mostly intact, a well-placed continuous suture can give enough support to the muscle to facilitate well-approximated healing. Deep lacerations bifurcate muscles. The two ends of a bifurcated muscle can be properly approximated with well-placed interrupted sutures. When severed perineal muscles are not well approximated, the end result may be weakness and a gaping introitus.
6. Provide appropriate anesthesia. Local infiltration or a pudendal block can be performed when needed.
7. Through the procedure, maintain good communication among the woman, the midwife, and other support personnel as appropriate.

Technique for Repair of Second-Degree Perineal Laceration or Episiotomy

1. A sponge or other absorbent material that has a tail, which remains outside the vagina, may be inserted as a tampon if necessary to maintain a dry field. Do not use a tampon without a tail. Some portion should always be visible outside of the vagina so removal when the procedure is over is assured.
   a. Insert two fingers along the posterior vaginal wall, apply downward pressure, and slide the rolled tampon against the posterior aspect of the fingers toward the posterior fornix of the vagina.
   b. Secure the tail with a clamp to a drape where it can be seen and will not be forgotten or left in the vagina when the repair is complete.
2. Check the integrity of the anal sphincter. Feel the depth of the defect and ascertain that the anal sphincter is intact. Place a second glove over the examining hand if necessary to insert an examining finger into the anus to determine the integrity of the sphincter and rectal mucosa; discard this glove immediately after examining the rectum. Probe the tissue gently to avoid causing additional trauma.

3. Before starting to sew, gently support the tissue so that it falls together naturally and identify key landmarks that will be anatomically approximated, including the hymenal ring, apex of vaginal mucosa, mucocutaneous junction, and perineal apex. It may be helpful to use a tissue forceps with teeth to pick up one side of the laceration and carry it over to the other side to visualize how it should come together, before beginning suturing.
4. The tissue between the vaginal mucosa and rectum consists of muscle, fascia, and other tissue fibers that are collectively referred to as the recto-vaginal septum. It is repaired in stages, and in different planes. From above, sutures are placed vaginally with insertion directed down into the septum. Laterally, sutures are placed below the perineal skin, and directed inward.
5. Repair the vaginal mucosa and underlying recto-vaginal fascia: The vaginal mucosa and fascia are repaired with a 3-0 synthetic absorbable suture using an atraumatic, tapered needle with locked continuous suturing (**Figure 28F-8**).
    a. Visualize the vaginal apex by placing both fingers along the wound edges and applying gentle downward pressure. The anchoring stitch is placed approximately 1 cm beyond the vaginal apex of the wound, and provides hemostasis for any retracted blood vessels. This stitch is tied and one side cut close to the knot, leaving an approximately 0.5-cm tail.
    b. The vaginal mucosa and fascia are repaired from the posterior aspect of the wound in the vagina to the hymenal ring, using locked continuous (blanket) stitches. They should be approximately 1 to 1.5 cm apart. The first one or two stitches should be deep enough to include adequate tissue on each side of the wound to close it completely. The farther the wound extends posteriorly, the more stitches that are needed to bring the septum together, up to the level of the perineal muscles. Thereafter, the stitches should be shallower and include only the vaginal mucosa. The muscle should stay exposed so that it can be visualized and repaired.
    c. Avoid placing sutures through or over the hymenal ring, as they may cause discomfort in the healing process and subsequent dyspareunia (although proof that dyspareunia occurs is lacking). Instead, place the needle so that it locks the last vaginal stitch as it is directed downward to bring the suture from the vagina behind the hymenal ring into the perineal plane. It exits in the submucosal tissue of the wound, in the perineum.
    d. If the wound is shallow and appears to have just nicked the muscle, continuous suturing of the *subcutaneous tissue* to the apex of the perineal laceration is done as described later in this procedure. In contrast, if the wound appears deep enough to have significantly separated the perineal muscles, correct placement of deep interrupted stitches will give excellent support for an accurately reconstructed perineal body.
    e. For a deep repair, the first needle and suture are set aside, to be used again later in the subcutaneous and subcuticular repairs. It is important to “guard” the needle not in use by clamping the swaged end of the needle with the tip of the needle pointed toward and barely touching the needle holder. The needle and holder are then placed

onto a sterile drape.

6. *Muscle layer:* A layer of interrupted stitches is used to bring together the bulbocavernosus and perineal muscles. To determine the depth of suturing required, insert a finger below the vaginal mucosal area that has been repaired. If there is more than 1 cm of space below the sutured mucosa, interrupted stitches are placed to well approximate the muscles and close this space (**Figure 28F-9**).
   a. A 2-0 absorbable synthetic suture with an atraumatic needle is used for the interrupted stitches.
   b. Repair may begin with the crown stitch, to bring together the bulbocavernosus muscle (**Figure 28F-10)**. Since this muscle retracts posteriorly and superiorly, the crown stitch is best suited to grasp the large muscle ends to approximate them from top to bottom. It may be helpful to visualize a torn bulbocavernosus muscle as a tubular elastic ring with ends that retract when cut at the base. The crown stitch grasps the muscle from top to bottom on each edge.

      Some midwives choose to repair the muscles only with horizontal interrupted stitches. This preference is based upon a historical concern that a crown stitch may increase the risk of subsequent dyspareunia. Placing a horizontal stitch through the bulbocavernosus muscle may partially approximate it, particularly if the stitch goes through only the lowermost part of the muscle. No evidence exists to support one method over the other, but the integrity of this muscle clearly has functional significance.

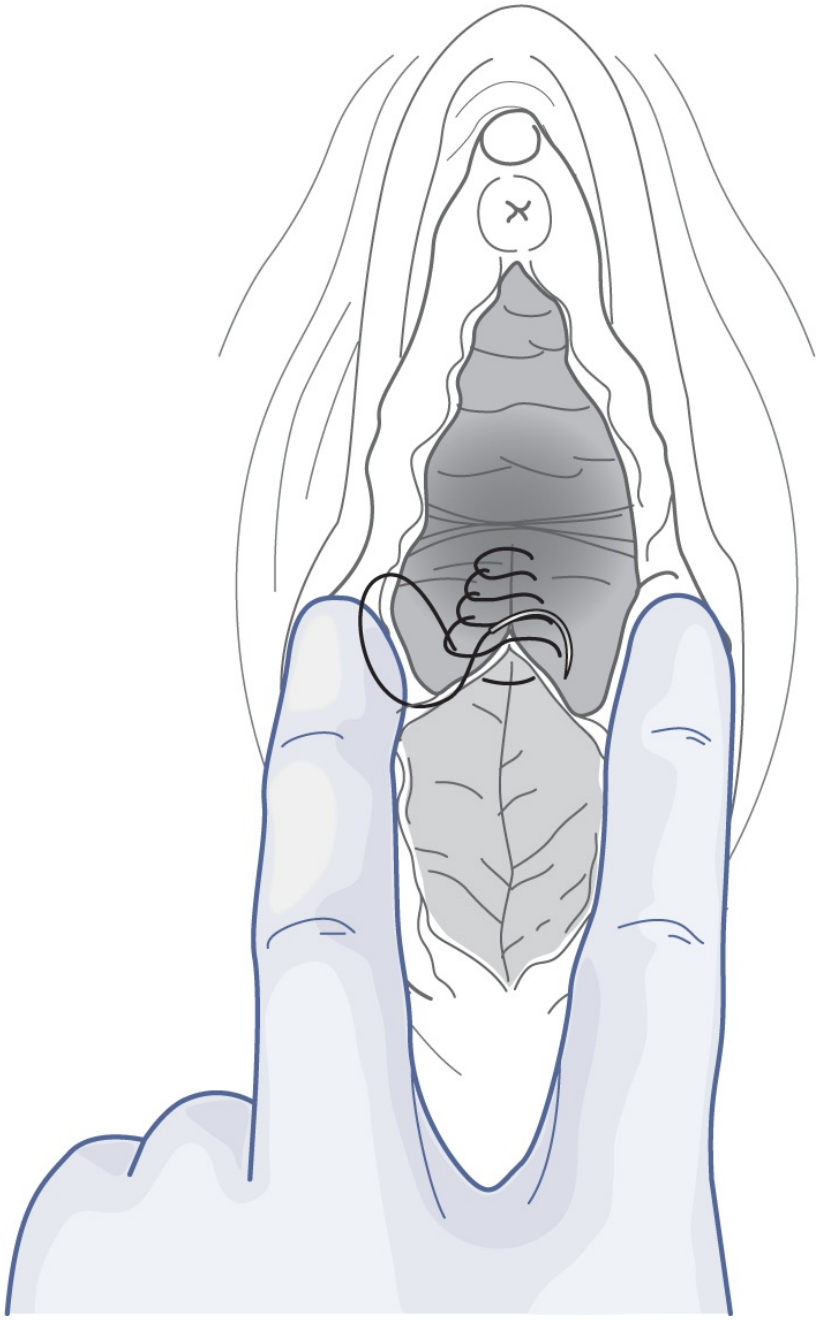


Figure 28F-8 Repair of vaginal mucosa and recto-vaginal fascia using continuous locked (blanket) stitches.

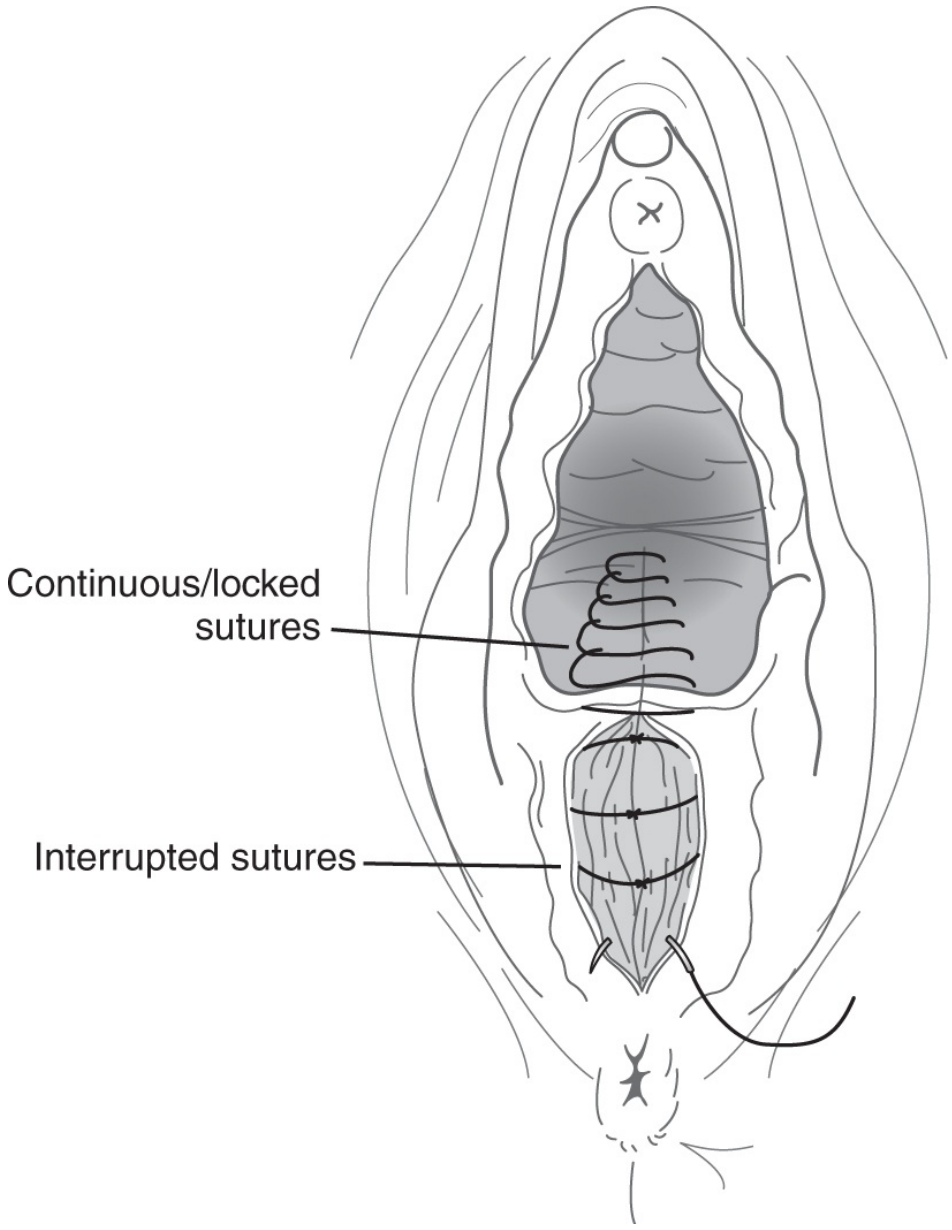


Figure 28F-9 Repair of muscles using interrupted stitches.

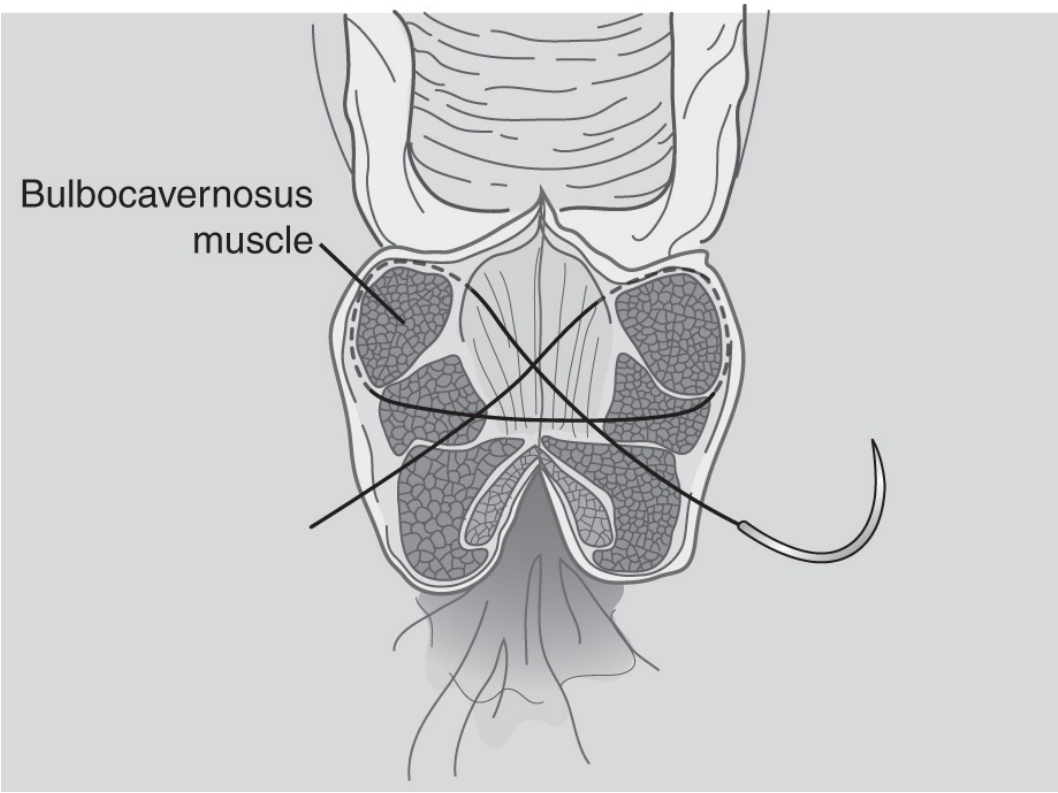


Figure 28F-10 Crown suture.

c. The perineal muscles retract laterally. Place deep interrupted stitches in a horizontal plane to ensure that the stitches do not accidentally go into the rectal mucosa. With the woman supine, the needle holder will be perpendicular to the floor and the introitus. The amount of tissue to be taken up is often too much for one bite, so the first action directs the needle to come out into the back of the defect. The needle is then grasped with a tissue forceps (without teeth) and replaced correctly in the needle holder, and a second stitch is taken to incorporate the other side. It is important that needle exit and entry points are directly across from each other.

d. After the interrupted stitches are placed, perform a rectal examination to determine whether any of the stitches can be palpated within the rectal mucosa. To maintain sterility, either a new glove is placed over the hand used for the examination and then discarded, or the single glove must be replaced. If a suture is palpated in the rectum, the repair must be dismantled until the suture is removed to prevent infection or creation of a sinus tract. The risk of a rectal suture is diminished if all interrupted stitches are placed carefully in the horizontal plane.

7. *Subcutaneous tissue*: At this point, the suture that had been set aside in Step 5 is used to repair the subcutaneous tissue under the fourchette and down the length of the laceration toward the anus. The goal is to obliterate space that would be created by closing the skin edges over a subcutaneous defect.

a. Close the perineal subcutaneous fascia using continuous (unlocked, running) stitches, as illustrated in **Figure 28F-11.**

b. Resume using the original suture that was used to close the vaginal mucosa. Close

the subcutaneous tissue with a running stich, moving down the defect toward the inferior perineal apex close to the anus. Do not bring the needle out too close to the skin, as the layer of tissue immediately under the skin will be closed with a final layer of subcuticular stitches.

c. Place stitches approximately 1 cm apart.

d. Bring the needle out in the perineal apex of the laceration.

e. Change the direction of the needle on the needle holder for the subcuticular stitches.

8. *Subcuticular stitches*: Leave sufficient and equilateral room on both sides for the final layer of continuous stitches, with the last stitch exiting at the central point of the perineal apex. This stitch should be shallow and immediately inside, but not through, the skin.

a. Perform the subcuticular, or mattress, stitch using the same suture that was used for the subcutaneous stiches.

b. Complete stitches alternatively from side to side, moving superiorly toward the fourchette. Each stitch starts exactly parallel to the point on the opposite side where the last suture emerged (**Figure 28F-12).**

i. Bring the suture across to see where to place the needle for the next stitch.

ii. Shorter stitches are usually placed at the perineal apex near the anus, whereas longer stitches can be made closer to the mucocutaneous junction.

iii. Do not pull too tightly—edges should not overlap.

c. Approximate the mucocutaneous junction exactly, and then continue a few subcutaneous stitches back toward the hymenal ring. The depth of the fourchette or space between the mucocutaneous junction and the hymenal ring varies among women. Some will require one or two stitches, and some may require as many as three or four stitches.

d. *Final knot*: Bring the needle under and posterior to the hymenal ring. The suture will now be tied with a square knot, across the laceration. The needle should next enter close to the exit point of the preceding stitch and should be carried straight under the laceration, exiting on the opposite side. Do not pull the suture through the tissue entirely, but instead leave a loop that becomes one side of the tied knot. Cut the needle off of the opposite side and tie the knot with the two ends across the laceration, just posterior to the hymen. The knot should be made securely, but not tight enough to cause tissue strangulation. Placement of the knot behind the hymen decreases the risk of discomfort for the woman.

9. After suturing, remove the tampon, if one was used. Inspect and verify the suture line is intact and without any hematoma or active bleeding along the suture lines. Other actions include ascertaining the firmness of the fundus and removing any blood clots. Depending on the circumstances, a rectal examination to rule out any rectal sutures or hematomas may be performed. Wash the perineal area, thighs, and buttocks as needed. Assist in helping the woman assume a comfortable position. Apply ice to the repaired laceration.

10. During and after the procedure, the midwife should continually communicate with the woman and family and provide anticipatory guidance and health education.

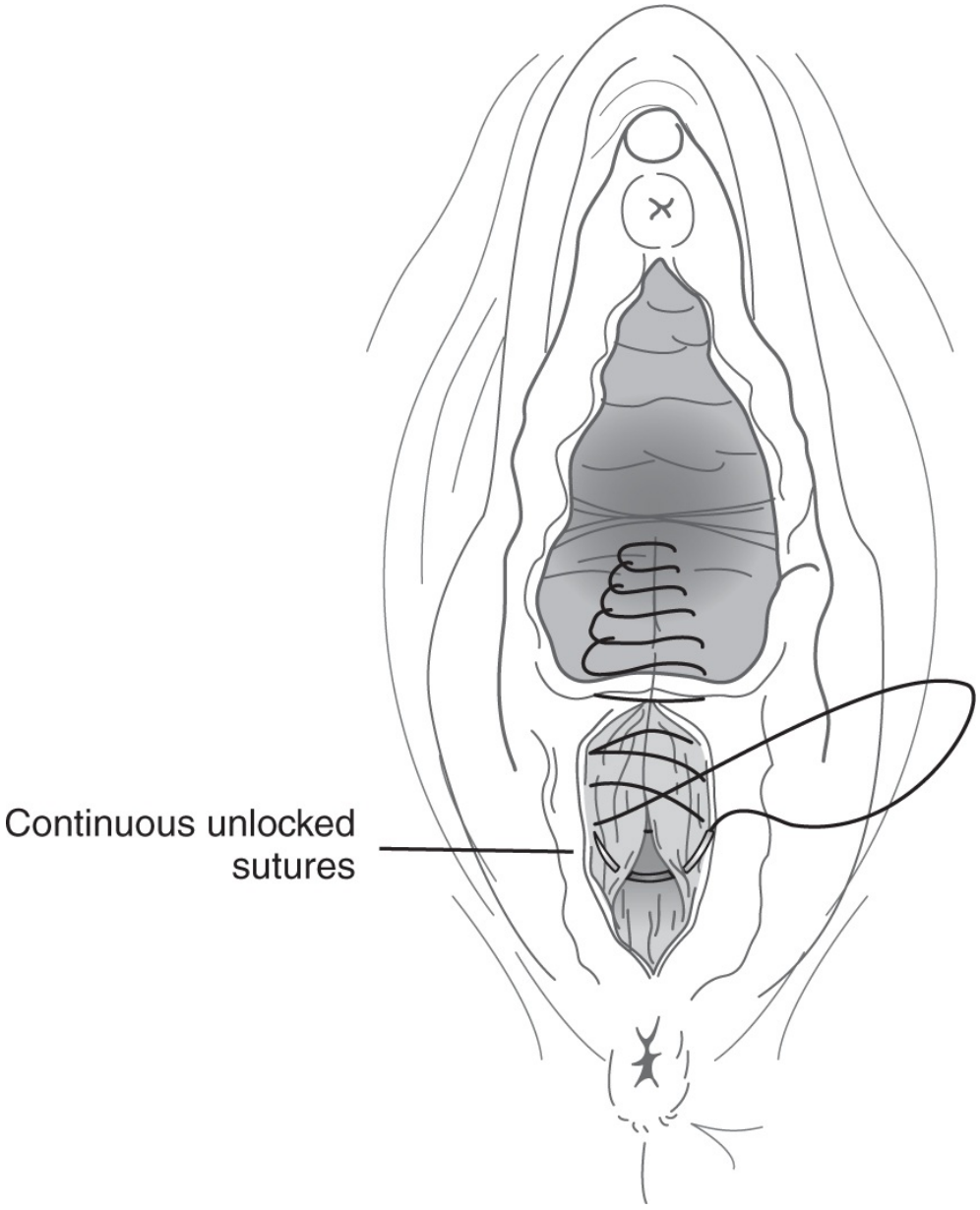


Figure 28F-11 Repair of perineal subcutaneous fascia using continuous stitches.

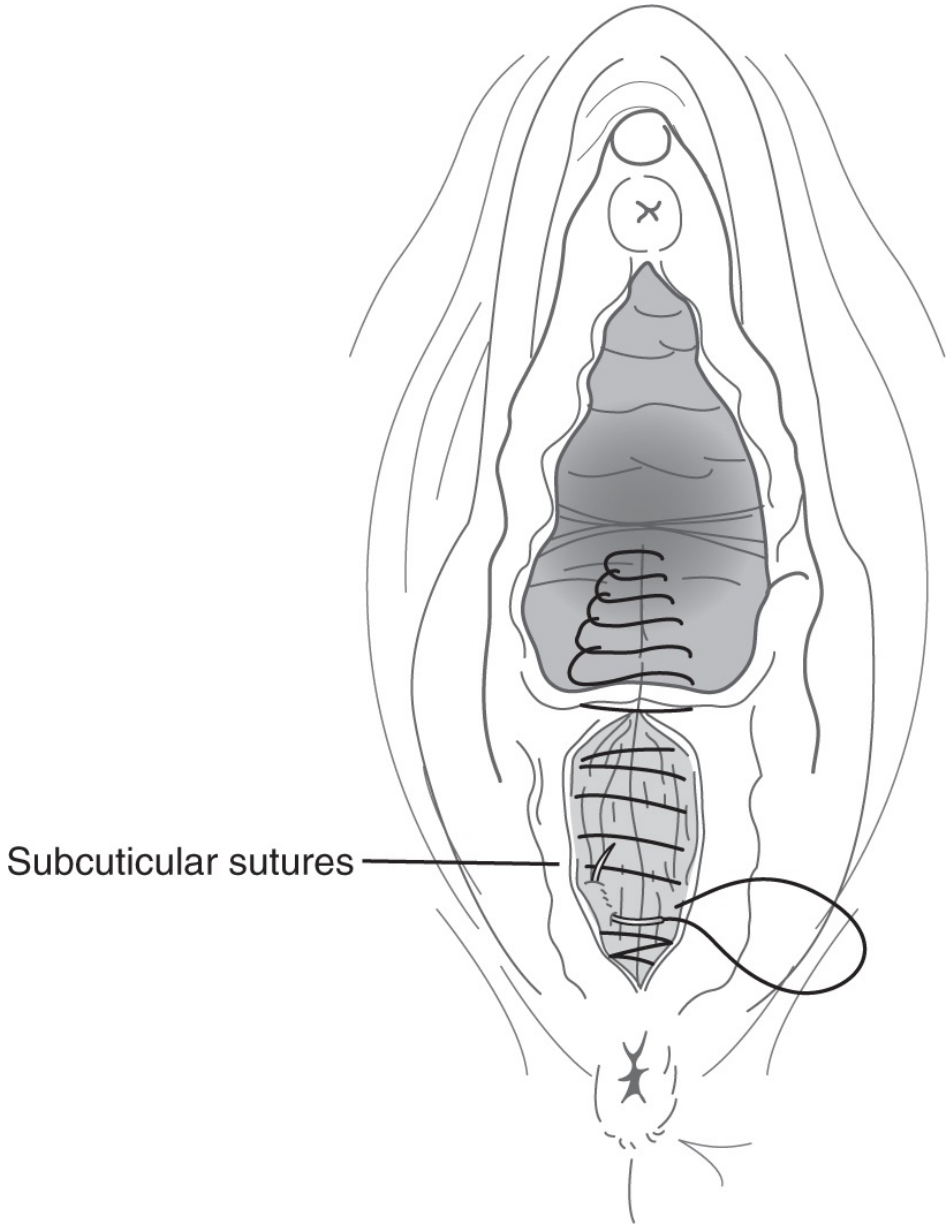


Figure 28F-12 Reapproximation of skin using subcuticular (mattress) stitches.

Repair of First-Degree Lacerations

The majority of first-degree lacerations are small skin disruptions that barely lacerate the vaginal mucosa and will heal spontaneously without suturing. More-extensive first-degree lacerations can be repaired using continuous running stitches. In some situations, a single interrupted stitch or two may accomplish closure. The midwife should assess the area to be repaired, approximate the laceration prior to suturing, and visualize all appropriate landmarks; the midwife should then plan the number of stitches that may be required in advance with the principle of placing the fewest possible to accomplish hemostasis and tissue edge reapproximation. A finer-gauge suture, such as 4-0 polyglactin on a small needle, may provide adequate tensile strength and be less traumatic.

Repair of Sulcus Tears

Sulcus tears are a type of second-degree laceration in which the vaginal mucosa and underlying tissue are lacerated along one (unilateral) or both (bilateral) sides of the posterior column of the vagina, instead of up the middle. The posterior column of the vagina is a longitudinal ridge of the inner surface of the lining extending the length of the vagina. The lateral edges of the column meld into the lateral vaginal walls and create a slight groove (sulcus) from which this type of laceration derives its name.

Significant blood loss can occur with deep sulcus tears, requiring prompt placement of figure of eight stitches. Repair of sulcus tears differs only from the repair of the vaginal mucosa if the tear is bilateral. In this event, a midline tear will bifurcate in the posterior vagina and the laceration can have a Y shape. Two apices and two separate lines of continuous locked stitches are needed to close the separate tears in the Y portion of the laceration. At their common base, one line of sutures is tied with a final stitch and a square knot, while the other line of sutures continues to draw the larger base opening together. Sulcus tears usually are deep lacerations and may require two layers of deep interrupted stitches when the deep muscle layer is repaired. To provide better access for placing the deep interrupted stitches, the vaginal mucosa may be repaired with a few stitches placed in the common base of the bilateral tear for approximation of the tissues, followed by interrupted stitches, followed by the return to the repair of the vaginal mucosa. Because of the depth and potential difficulty with visualization, a second person may be requested to assist with the repair process to aid in visualizing the wound.

Repair of Periurethral, Clitoral, and Labial Lacerations

Periurethral and labial lacerations usually are longitudinal lacerations that follow the general contour of the vestibule of the perineum. They vary in depth as well as in length, with some extending superiorly into the glans of clitoris, or inferiorly exposing the crus of the clitoris. At other times, they may be transverse lacerations extending into the labia minora and occasionally across the labia majora so that it is torn into two sections.

Repair of periurethral lacerations depends on their depth, bleeding, and the need for a cosmetic repair. Repair of any periurethral laceration close to the urethra should be preceded by insertion of a urinary catheter to prevent accidental closure of the urethra. Small superficial periurethral lacerations that are not bleeding will often heal spontaneously without stitches. Although rare, bilateral labial lacerations directly across from each other may heal together, fusing the labia. At least one of these lacerations should be closed to prevent this outcome. When significant labial edema is present, bilateral suturing and postpartum treatments such as initial application of cold packs can reduce the extent of labial edema.

When repair is indicated, most periurethral lacerations should be repaired with 4-0 suture using an interrupted technique. Prior to beginning the repair, the midwife should determine the best approach, using the least number of stitches while accomplishing reapproximation and hemostasis. There is no precise pattern or sequence of stitches to use, as each laceration is

unique.

Repair of labial lacerations may also be done with 4-0 gauge suture using an interrupted stitch. Interrupted stitches can be approximately 0.5 to 1 cm apart and should not be tied too tightly, as this tissue can very easily become edematous. Continuous sutures are not recommended for labial lacerations because they can constrict edematous tissue and distort the healing process.

Repair of Third- and Fourth-Degree Lacerations

The experienced midwife may be credentialed to perform repairs of third- or fourth-degree lacerations. Appropriate education, experience, and institutional privileges are needed in such a case.

Key Points

1. Good visualization and adequate anesthesia are essential to accurately repair a third- or fourth-degree laceration. Assistance is likely needed to secure the visualization needed.
2. Fourth- and third-degree lacerations are always repaired before repairing the rest of the perineal laceration, if present.
3. The goal is to reconstruct the muscular cylinder of the rectal sphincter, which is approximately 2 cm thick and 3 cm long.
4. The area may be irrigated with saline prior to being repaired. In contrast to practice years ago, antiseptic solutions generally are no longer employed, as they may irritate this tissue.

Repair of Fourth-Degree Lacerations

Fourth-degree lacerations involve the external anal sphincter, internal sphincter, and anterior rectal mucosa. The rectal mucosa and the internal anal sphincter are always repaired before the external rectal sphincter is repaired. After identifying the tear in the anterior rectal wall and the torn ends of the anal sphincter, the repair begins by rejoining the anterior rectal mucosa. This area is repaired in two layers with 3-0 or 4-0 polyglactin suture on an atraumatic needle.

1. The first layer starts at the internal apex and consists of a row of interrupted stitches placed in the rectal submucosa to approximate the rectal mucosa without placing stitches in the lumen of the bowel. The use of a smaller, atraumatic curved needle is essential to take smaller amounts of tissue into each stitch, thereby decreasing the risk of extending into the lumen of the bowel.
2. The second layer is the internal anal sphincter; it appears as a shiny pink, thick tissue that lies just above the rectal mucosa. It often retracts laterally and superiorly. Repair is made with a continuous technique using 3-0 polyglactin suture. This repair also starts at

the internal apex and is brought out to the cutaneous junction, where it is tied off.

3. After verification that no sutures are in the rectal lumen, repair of the external anal sphincter as described in the next section, followed by standard repair of the laceration or episiotomy, is completed. Special care must be taken in rebuilding the muscle layers of the perineal body, which have been totally lacerated.

Repair of Third-Degree Lacerations

A third-degree laceration differs from a fourth-degree laceration in that it does not include the rectal mucosa. Otherwise, repair of the anal sphincter is the same for both types of lacerations.

1. Identify the torn external anal sphincter. As the torn ends retract, they are found flush with, or as dimples in, the lateral walls at the bottom of the perineal aspect of the wound near the surface. The muscle fibers of the sphincter are visually different from the surrounding fascia. Often one side is dimpled, and the muscle can be visualized emerging from the other side.
2. Grasp both torn ends with an Allis clamp and pull them toward each other, causing them to meet. The suture of choice is 2-0 synthetic, absorbable suture. Some midwives prefer to first anchor a 3-0 absorbable suture within the inferior apex of the skin extension of the laceration and take a few subcuticular stitches in the posterior anal capsule, then lay this suture aside until the end. This approach is intended to avoid later difficulty in repairing the subcuticular area over the rectal sphincter, as it is sometimes difficult to visualize this apex after repair of the sphincter.
3. Repair a lacerated external anal sphincter with either doubled interrupted stitches or a figure of eight stitch after approximating the torn ends grasped by the Allis clamps.
   a. Inclusion of the anterior and posterior fascial layers surrounding the sphincter muscle strengthens the repair.
   b. The internal and external sphincters may both be lacerated. Knowledge of the anatomy is essential to identify the anal sphincter.
   c. The sphincter can be repaired by one of two methods: The first places the torn section “end to end” and the second uses an “overlapping” technique (**Figure 28F-13)**. The overlapping technique is superior in that it is associated with less fecal incontinence and pain.[7-10] The overlapping method is recommended for a complete tear but cannot be used for an incomplete tear (grade 3a or partial-thickness grade 3b).[7]
   d. Technique for a doubled interrupted stitch:
      i. Put the needle into the right side of the muscle and bring it out in the left side of the muscle.
      ii. Put the needle in the left side of the muscle near where it came out and bring it out in the right side.
      iii. The suture now has two strands and will be tied off on the side where it was started.
   e. *End-to-end repair*: Place four doubled interrupted sutures, starting at the posterior

aspect as illustrated in **Figure 28F-13A**, with one in each quadrant at the 6 o'clock, then 9 o'clock, then 12 o'clock, and then 3 o'clock positions.

f. *Overlapping technique*: Using the Allis clamp, overlap the ends of the sphincter and place three doubled interrupted sutures in both parts of the muscle where they overlap (**Figure 28F-13B**).

4. After repairing the sphincter and before continuing with the remainder of the repair, perform a rectal examination to confirm that sutures have not been placed in the rectal mucosa as previously described. Repair of the laceration or episiotomy then continues in standard fashion.
5. When a woman has a third- or fourth-degree perineal laceration, it is common practice to order stool softeners, oral analgesics, and sitz baths for her initial postpartum recovery. Antibiotics to prevent wound infection are considered on an individual basis.[7]

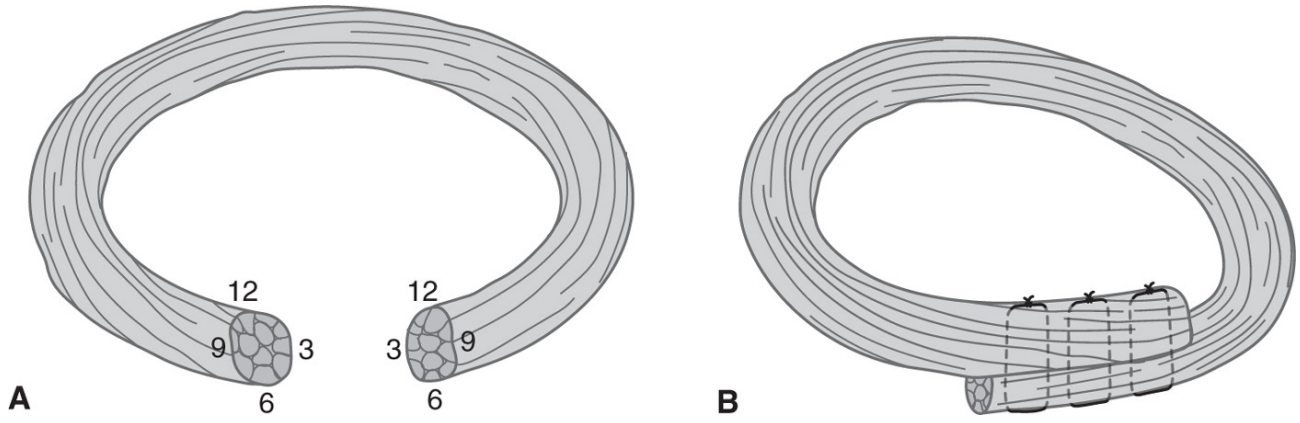


Figure 28F-13 Repair of anal sphincter. **A.** End-to-end technique. **B.** Overlapping technique.

Repair of Cervical Lacerations

Cervical lacerations can be difficult to identify and can bleed extensively. An assistant may be invaluable in this situation to hold the retractors that will enable good visualization.

Key Points

1. Good visualization is necessary, including effective use of the ring forceps on the cervix to bring the cervix and its laceration into full view, and a well-contracted uterus so any potentially obscuring bleeding is minimal (**Figure 28F-14).**
2. If an assistant is not available, use of an instrument such as a Gelpi or Sims retractor can provide better visualization. Since cervical lacerations can extend into the lower uterine segment, it is critical to be able to visualize the apex with certainty.
3. Anesthesia will be of benefit and allow relaxation that will promote better visualization.
4. Cervical lacerations more than approximately 1 cm in length or a laceration of any length that is bleeding should be repaired.

5. Cervical lacerations are repaired with 2-0 absorbable synthetic suture.

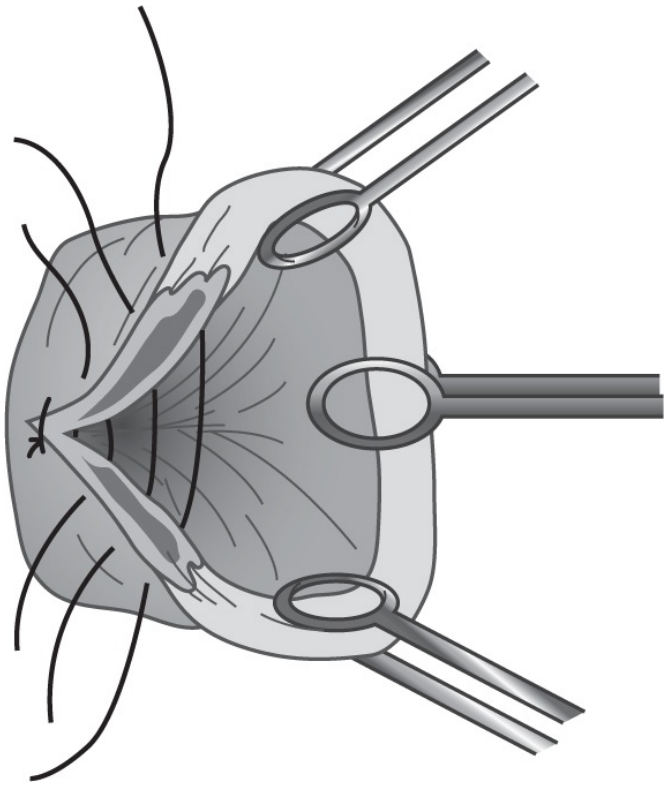


Figure 28F-14 Repair of cervical laceration using interrupted stitches.

Steps to Repair Cervical Lacerations

1. Approximation of the two sides of the tear is facilitated by the placement of a ring forceps on each side of the laceration. The forceps should be placed close enough to the edges to be of use, but far enough away from the edges that the stitches can be taken without sewing through the ring of the forceps.
2. Cervical lacerations are repaired with interrupted stitches, a continuous stitch, or a continuous locked stitch if additional hemostasis is required.
3. The first stitch should be placed approximately 1 cm above or beyond the apex of the laceration to include any retracted blood vessels.
4. Care should be taken in the placement of each stitch approximating the two sides of the tear so that only the sides of the wound are included in the stitch, thereby maintaining the patency of the cervical canal.

Controversies Regarding Laceration and Episiotomy Repair

Controversy has emerged about when to repair genital lacerations versus when to allow them to heal spontaneously. Because every laceration is unique, meaningful evidence has been

difficult to find regarding this question. In general, if the edges of the laceration appear to be well approximated when the tissue is not being handled or when the woman's legs are relaxed in a natural position, then the use of suture for repair may be avoided. Today, some professionals question whether even a second-degree laceration might heal in that manner, but available evidence on this topic is scant and inconsistent.[11]

To Repair Skin Edges or Not to Repair Skin Edges

Some authors have suggested that leaving the skin edges unsutured may result in less pain or dyspareunia, as the sutures themselves can cause irritation. A randomized trial of two-layer versus three-layer repair found that leaving the skin edges unrepaired does result in less pain and dyspareunia at 3 months postpartum, without disadvantages such as infection or repair breakdown.[12]

Episiotomy

Multiple randomized trials and a Cochrane meta-analysis have demonstrated that routine episiotomy is associated with an increased risk for severe perineal lacerations.[13] Consequently, this procedure is no longer a standard practice at all births.

Yet, because every birth is unique, cutting and repair of an episiotomy is a skill to be included in the repertoire of a midwife so that, when needed, it can be performed competently. An understanding of perineal anatomy is critical to the discussion of episiotomies/lacerations, and relevant information can be found in the *Anatomy and Physiology of the Female Reproductive System* chapter.

Episiotomies should be discussed with women prior to labor so they are aware of the indications and the procedure should it become necessary. The one accepted indication for an episiotomy is a need to expedite birth secondary to fetal bradycardia or other fetal heart rate pattern that suggests a significant risk of newborn acidemia. Other suggested but controversial indications include operative birth, suspected shoulder dystocia, and short perineum (with a mediolateral episiotomy to avoid a spontaneous fourth-degree laceration).

Key Points: Preparation

1. During labor, when it becomes apparent that an episiotomy may be necessary, the midwife should discuss the indication and recommendation with the woman and her support persons. Continuous communication as the procedure is performed informs the woman of events, especially if she has regional anesthesia and impaired sensation.
2. Appropriate equipment should be available.
3. Anesthesia is a prerequisite before an episiotomy is performed, and the level of anesthesia should be appropriate for the extent of surgical repair. Although nitrous oxide or parenteral opioids may be useful as analgesia, an epidural or a pudendal nerve block will be more effective. Under some circumstances, local infiltration may be performed prior to cutting an episiotomy, although this intervention may distort tissue and cause

edema. A description of the techniques for local infiltration and pudendal block can be found in Appendix 28D and Appendix 28E, respectively. A woman's allergies may preclude use of some anesthesia methods.

Midline Versus Mediolateral Episiotomy

Two types of episiotomy are performed: midline incision and mediolateral incision. The midline episiotomy is more commonly used in the United States.

Midline Episiotomy

The midline (or median) episiotomy has a vaginal apex at the central tendinous point of the perineum and separates the bulbocavernosus and superficial transverse perineal muscles. Depending on the depth of the cut, the deep transverse perineal muscle may also be cut into two sides. The perineal apex is above the anal sphincter.

The primary advantage of a midline episiotomy is ease of repair and better cosmetic result. Although retrospective studies of postpartum pain have found that midline episiotomies are associated with less pain than mediolateral episiotomies, prospective studies have not found a significant difference. Because the inferior apex of the midline episiotomy is directly above the rectal sphincter, extension into a third- or fourth-degree laceration is possible.[14]

Mediolateral Episiotomy

A mediolateral episiotomy also has a vaginal apex at the central tendinous point of the perineum. This incision cuts through the bulbocavernosus and the superficial and deep transverse perineal muscles, and into the pubococcygeus (levator ani) muscle. Whereas a midline incision is directly anterior–posterior, the mediolateral is cut on the diagonal (right or left) starting at the midline of the posterior fourchette, with the points of the scissors directed right or left toward the ischial tuberosity on the same side as the incision (**Figure 28F-15).**

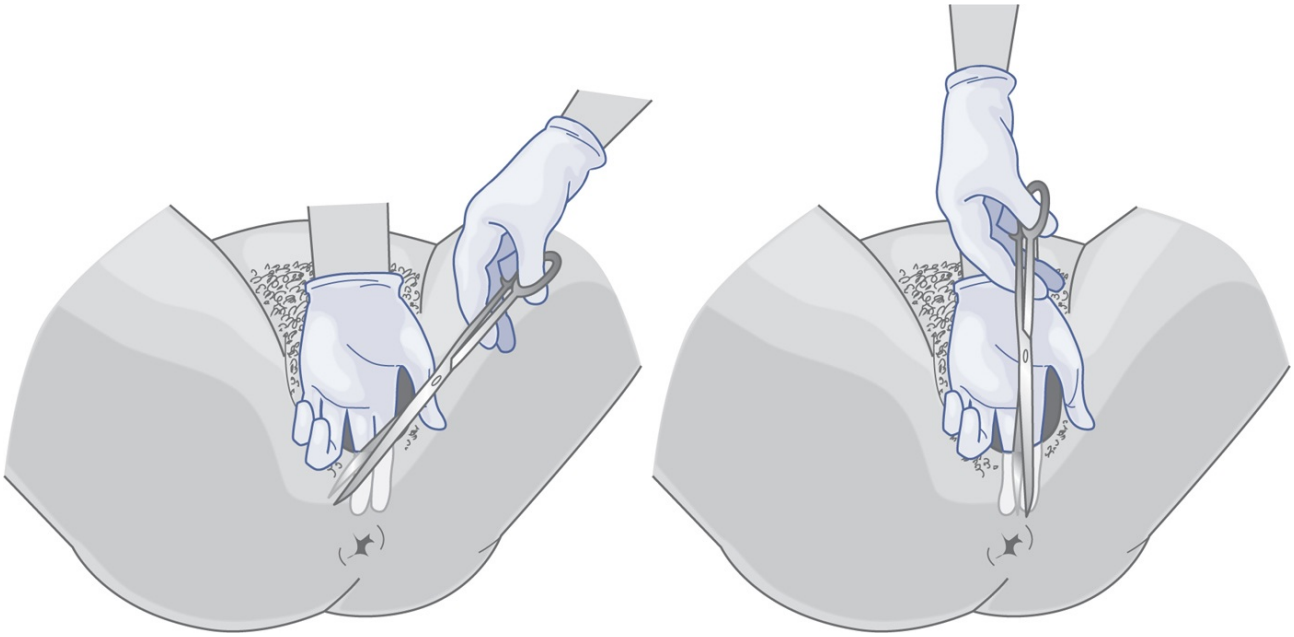


Figure 28F-15 Types of episiotomies.

The primary advantage of a mediolateral episiotomy is that fewer extensions into a third- or fourth-degree laceration occur, as compared to the outcomes of midline episiotomies. Some studies have found mediolateral episiotomies to be associated with more blood loss; others have not. Compared to midline episiotomies, mediolateral episiotomies can be more challenging to repair. The superior side of the wound has a larger area of exposed tissue than the inferior side, requiring larger "bites" of tissue superiorly when stitching.

When to Cut an Episiotomy

There is little evidence to guide a midwife regarding when to cut an episiotomy. Performing this procedure too early may result in extra blood loss. Waiting until the perineum is paper thin and the tissue bulging, however, may result in a rapid and perhaps uncontrolled birth of the fetal head, with resulting extension of the episiotomy. Many midwives advocate incision when the head is crowning and not retracting between contractions, such that birth is expected within a few contractions. When the head is well applied to the perineum, it also provides a type of tamponade to decrease bleeding.

After the episiotomy is performed, the midwife should continue to apply gentle pressure to the fetal head to control and direct a slow extension of the head. This will help avoid a rapid birth, sudden pressure on the perineum, and extension of the incision. Pressure on the wound to control bleeding may also be required between maternal pushing efforts.

Technique for Performing an Episiotomy

1. The midwife places the index and middle fingers into the vagina, palmar side down and facing away from the woman. The fingers should be slightly separated and provide gentle outward pressure on the perineal body to create adequate space between the

perineum and the head, and to allow the scissors to slip between the fingers using them as a guide. Thus, the fetal head is not touched, yet protected. The pressure also slightly flattens the perineal area, providing an easier incision.

2. Place the blades of the scissors in the appropriate direction—either straight anterior–posterior or diagonally, depending on the type of episiotomy to be cut. One blade of the scissors should be against the posterior vaginal wall, while the other blade should be against the skin of the perineal body, with the point where the blades cross at the midline of the posterior fourchette.
3. Locate the external anal sphincter by palpation using fingers in the vagina and the thumb of the same hand placed externally to the perineal body. Knowing the location of the external anal sphincter minimizes hesitation in cutting an adequate episiotomy due to fear of accidentally cutting through the sphincter.
   a. When cutting a midline episiotomy, adjust the length of the blades of the scissors on the perineal body to the projected length of the incision and terminate above the anal sphincter.
   b. When cutting a mediolateral episiotomy, position the blades so that there is approximately 1 cm of levator ani muscle between the incision and the sphincter. Another way to visualize this is to cut laterally from the midline at a 40–60° angle.[15] This directs the incision far enough laterally to avoid the sphincter during the process of cutting the episiotomy and when performing the repair. Take care not to start the incision on the lateral aspect of the fourchette or to direct the incision too far laterally to avoid incising the Bartholin gland on that side.
4. Incise the perineum with a single cut that is sufficient to open the perineal body and extend into the vaginal vault. Incising in this manner opens the vagina and the perineum. Performing a single incision through both the vagina and the perineum with one cut avoids the need for multiple incisions and increased trauma to the tissue.
5. After the initial incision, observe and evaluate the area to identify any active bleeding. Adequate visualization usually requires use of sponges to blot away blood, although excessive and aggressive blotting or using the sponges to wipe can further harm the tissue.
6. If a second incision is necessary, it can be performed at this time. Extension of an incision is best made by using fingers in the vagina to guide the incision and protect the fetal head.
7. By palpating for a band of tight, restricting vaginal tissue just inside the introitus, the midwife can further evaluate the adequacy of the incision. If such a band is present, it can be determined whether the band must be cut to be released.

References

1. Albers L, Garcia J, Renfrew M, McCandlish R, Elbourne D. Distribution of genital tract trauma in childbirth and related postnatal pain. *Birth*. 1999;26:11-17.
2. Smith LA, Price N, Simonite V, Burns EE. Incidence and risk factors for perineal trauma: a prospective observational study. *BMC Pregnancy Childbirth*. 2013;13:59. doi:10.1186/1471-2393-13-59.
3. Leffell DJ, Brown MD. *Manual of Skin Surgery: A Practical Guide to Dermatologic Procedures*. New York, NY:

Wiley; 1997.
4. Kettle C, Dowswell T, Ismail KMK. Absorbable suture materials for primary repair of episiotomy and second degree tears. *Cochrane Database Syst Rev.* 2010;6:CD000006. doi:10.1002/14651858.CD000006.pub2.
5. Bharathi A, Reddy DB, Kote GS. A prospective randomized comparative study of Vicryl Rapide versus chromic catgut for episiotomy repair. *J Clin Diagn Res.* 2013;7(2):326-330. doi:10.7860/JCDR /2013/5185.2758.
6. Ethicon. Knot tying manual. Available at: http://surgsoc.org.au/wp-content/uploads/2014/03/Ethicon-Knot-Tying-Manual.pdf. Accessed December 12, 2017.
7. American College of Obstetricians and Gynecologists. Practice Bulletin No. 165: prevention and management of obstetric lacerations at vaginal delivery. *Obstet Gynecol.* 2016;128:e1-e15.
8. Fernando RJ, Sultan AH, Kettle C, Radley S, Jones P, O'Brien PM. Repair techniques for obstetric anal sphincter injuries. *Obstet Gynecol.* 2006;107(6):1261-1268.
9. Leeman L, Spearman M, Rogers R. Repair of obstetric perineal lacerations. *Am Fam Physician.* 2003;68(8):1585-1590.
10. Fernando RJ, Sultan AH, Kettle C, Thakar R. Methods of repair for obstetric anal sphincter injury. *Cochrane Database Syst Rev.* 2013;12:CD002866. doi:10.1002/14651858.CD002866.pub3.
11. Elharmeel SMA, Chaudhary Y, Tan S, Scheermeyer E, Hanafy A, van Driel ML. Surgical repair of spontaneous perineal tears that occur during childbirth versus no intervention. *Cochrane Database Syst Rev.* 2011;8:CD008534. doi:10.1002/14651858.CD008534.pub2.
12. Gordon B, Mackrodt C, Fern E, Truesdale A, Ayers S, Grant A. The Ipswich childbirth study: 1. A randomized evaluation of two stage postpartum perineal repair leaving the skin unsutured. *Br J Obstet Gynecol.* 1998;105:435-440.
13. Jiang H, Qian X, Carroli G, Garner P. Selective versus routine use of episiotomy for vaginal birth. *Cochrane Database Syst Rev*. 2017;2:CD000081. doi:10.1002/14651858.CD000081.pub3.
14. Sooklim R, Thinkhamrop J, Lumbiganon P, et al. The outcomes of midline versus medio-lateral episiotomy. *Reprod Health*. 2007;4:10-16.
15. Ma K, Byrd L. Episiotomy: what angle do you cut to the midline? *Eur J Obstet Gynecol Reprod Biol.* 2017;213:102-106.

CHAPTER

29

Complications During Labor and Birth

AMY MAROWITZ

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Introduction

Even under the most normal of circumstances, intrapartum complications can occur. While many of these situations develop gradually or are associated with an already known high-risk condition, some will arise suddenly and without warning. This chapter builds on material reviewed in the *Pregnancy-Related Conditions* and *Medical Complications in Pregnancy* chapters. An overview of selected complications and conditions that can occur during the intrapartum period is presented. Familiarity with intrapartum complications and emergencies is essential to midwifery practice and requires frequent review of current management guidelines. Additional resources and study may be necessary for a more thorough exploration of the underlying pathophysiology associated with certain preexisting high-risk conditions.

When intrapartum complications are encountered, initiation of interventions often follows a specific progression as the situation evolves. This requires the midwife to be vigilant for worsening signs or symptoms. In addition, the need for intervention may disrupt the normal process of labor and will necessitate a careful evaluation of the risk–benefit ratio of intervention versus nonintervention. Shared decision making with the woman and her support persons regarding changes in the plan of care is an important component of midwifery management and should be used if possible when a deviation from normal is encountered.[1]

If an obstetric emergency occurs during the intrapartum period, prompt recognition and action is required to avoid serious adverse maternal or fetal outcomes, and shared decision making may not be possible. The midwife must also be ready and able to manage acute emergent situations within the appropriate institutional and legal boundaries of midwifery practice until the complication is resolved or the consulting physician can take over.

In some cases, a collaborative management plan with the consulting physician will already be in place prior to the onset of labor. The development of a new intrapartum complication or deviation from normal labor progress will require the midwife to seek physician consultation or to establish a collaborative plan of care. Within this collaborative model of care, the midwife provides continuity of care and advocacy for the woman, and protects normal labor progress as the clinical situation allows. Additionally, the midwife contributes to medical decision making while promoting family-centered care and keeps the consulting physician informed of any changes in the woman's condition. The medical rationale for consultation, collaboration, and referral as well as the resultant personnel involved in a woman's care are always clearly documented in her health record.

In cases where referral of care is indicated, the midwife can maintain an active role in the supportive care of the woman and her family, seeking the most appropriate balance of medicine and midwifery in collaboration with the physician. This supportive role is ongoing throughout the woman's intrapartum clinical course and continues into the postpartum period as needed. Women who experience an unanticipated obstetric complication or emergency will greatly benefit from the continuity and emotional support provided by their midwife.

Preterm Labor/Birth

Preterm birth is one of the leading causes of neonatal mortality in the United States.[2] Infants born prior to 37 weeks' gestation also face an increased risk of morbidity, such as neurologic and developmental disabilities. The *Pregnancy-Related Conditions* chapter details risk assessment for preterm birth and the role of various prevention strategies. In this chapter, the focus is on the evaluation and management of women who have suspected or confirmed active preterm labor.

Evaluation of Women with Possible Preterm Labor

Anticipatory guidance about signs and symptoms of possible preterm labor (PTL) should be provided during prenatal care, and women should be encouraged to report any such signs or symptoms. Women who are experiencing painful or frequent uterine contractions or more than 6 contractions per hour, vaginal bleeding, or any loss of fluid prior to 37 weeks' gestation should be evaluated promptly for possible preterm labor.[3] Other less-specific symptoms, such as cramping, pelvic pressure, abdominal pain, back pain, and watery vaginal discharge, could also indicate preterm labor.

The differential diagnoses for spontaneous PTL includes physiologic causes such as Braxton Hicks contractions, dehydration, lax vaginal tone, and round ligament pain. Other, more serious etiologies, such as infection (intrauterine, renal, genital tract), abruption, trauma, or appendicitis, must be considered as well and assessed for when obtaining a history and conducting a physical examination. The evaluation procedure for preterm labor is outlined in **Table 29-1**.

Table 29-1	Evaluation for Preterm Labor

1. History and Chart Review

a. Confirm the gestational age using the most credible data available.
b. Review the woman's history for any preterm birth risk factors, especially for prior spontaneous preterm births or unexplained second-trimester loss.
c. If there is a history of prior preterm birth:
    i. Has the woman been receiving 17-hydroxy-progesterone injections?
    ii. Are serial cervical length measurements obtained via ultrasound or other cervical measurement instrument documented on prenatal record ?
d. Inquire about recent intercourse, strenuous physical activity, abdominal trauma, vaginal bleeding, loss of fluid, fever, nausea, vomiting, urinary tract infection symptoms, or abnormal vaginal discharge.

2. Physical Examination

a. Note the woman's demeanor, distress, and/or coping.
b. Assess temperature, pulse, respirations, and blood pressure.

c. Palpate the lower back and assess for CVAT tenderness to rule out independent or concomitant UTI.
d. Palpate the abdomen for rebound tenderness; note any guarding.
e. Palpate the uterus for fundal tenderness, contraction strength, and fetal position (Leopold's maneuvers).
f. Place the woman on continuous electronic fetal monitoring to assess fetal well-being and uterine activity.
g. Perform a sterile speculum examination:
    i. If there is pooling fluid, check nitrazine, check for ferning, or use other assessment method to rule out PPROM.
    ii. If there is no history of bleeding, vaginal examinations, or intercourse in the past 24 hours, obtain an fFN culture (see **Figure 29-1** for collection technique).
    iii. Obtain cultures for gonorrhea and chlamydia.
    iv. Obtain a specimen for wet mount to assess for bacterial vaginosis or trichomoniasis infection.
    v. Obtain a sample from the proximal third of the vagina and through the rectal sphincter for GBS culture.
h. If there is no evidence of PPROM, perform a digital cervical examination to assess dilatation, effacement, station, and presenting part.
i. Laboratory tests:
    i. Clean catch urinalysis (with microscopic analysis) and urine culture
    ii. Complete blood count
    iii. Other labs as clinically indicated
j. Ultrasonography
    i. Perform transvaginal assessment of cervical length (depending on institutional protocols and availability).
    ii. If ultrasound is performed, confirm the presenting part.

Abbreviations: CVAT, costovertebral angle tenderness; fFN, fetal fibronectin; GBS, group B *Streptococcus*; PPROM, premature prelabor rupture of membranes; UTI, urinary tract infection.

Fetal Fibronectin

Contraction frequency is a poor predictor of subsequent preterm birth. When evaluating women for possible preterm labor, diagnostic accuracy may be improved with use of fetal fibronectin and measurement of cervical length by vaginal ultrasound.[3]

When evaluating women for possible preterm labor, diagnostic accuracy may be improved with use of fetal fibronectin and measurement of cervical length by vaginal ultrasound.[4-6] *Fetal fibronectin* (fFN) is a biochemical marker that may aid in management of women with symptoms of preterm labor. This extracellular glycoprotein, which is found in the amniochorionic membrane, serves as an adhesive binder between the membranes and the decidua. It is normally present in cervicovaginal secretions prior to 20 weeks' gestation and again after 37 weeks' gestation as cervical remodeling takes place in preparation for normal full-term labor. Between 24 and 34 weeks, the presence of fFN is atypical and could indicate inflammation or uterine activity—both of which are precursors to preterm birth.[4]

Unfortunately, studies have found that fFN has a poor positive predictive value (PPV). The reported PPV among women who present with preterm labor symptoms varies from 13.9% to 25.9%.[5,6] Conversely, fFN has a high negative predictability (97.6%) for indicating that birth will not occur within 7 days.[6] In practical terms, this means that fFN should not be used as a screening test.[6] A positive fFN test in symptomatic women has limited clinical utility and

should not be used alone to determine management. However, a negative fFN test can be helpful in avoiding unnecessary treatment in symptomatic women, especially in equivocal situations such as a woman with contractions and no cervical change. Women with symptoms of preterm labor between 24 and 34 weeks' gestation with a negative fFN culture (or assay) have only a 1% to 2% chance of giving birth within the next 7 to 14 days.[6]

Fetal fibronectin is tested by sampling vaginal secretions; this sample must be collected prior to any other vaginal examinations. fFN sampling is contraindicated if there is evidence of ruptured membranes. The accuracy of fFN testing is decreased in the presence of lubricants, blood, recent intercourse, or cervical manipulation within the previous 24 hours. A sample of vaginal secretions should not be collected for fFN testing in these situations or if the cervix is more than 3 cm dilated or 80% effaced.

Typically, the fFN sample is collected at the beginning of the examination of a woman presenting with preterm labor symptoms and set aside until the results of a digital cervical examination and possibly a transvaginal ultrasound cervical length measurement are available. The culture can then be sent or discarded depending on the clinical situation. The fFN collection technique is outlined in **Figure 29-1**.

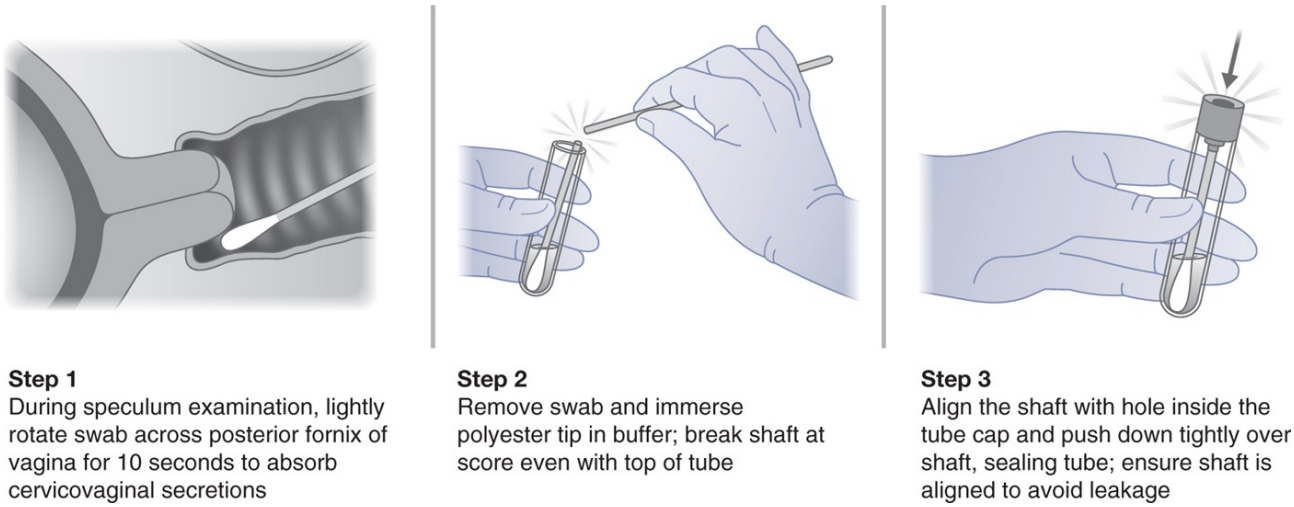


Figure 29-1 Fetal fibronectin specimen collection procedure.

Courtesy of HOLOGIC, Inc., and affiliates.

Cervical Length Measurement

As discussed in the *Pregnancy-Related Conditions* chapter, transvaginal ultrasound cervical length measurement can be used as a screening tool for asymptomatic women. A cervical length of 25 mm or less after 16 weeks' gestation and before 24 weeks' gestation is associated with an increased risk of preterm birth.[7] In women who are more than 24 weeks' gestation, cervical length assessment can be a helpful adjunct to fFN testing. Some researchers now advocate combining fFN and cervical length in standardized preterm labor algorithms.[4,8,9]

In these suggested protocols, women presenting with preterm labor symptoms between 24 and 34 weeks' gestation would have an fFN collected (if not contraindicated) during their initial speculum examination, followed by a cervical length measurement. The results of the

cervical length measurement determine whether the fFN test was needed. The fFN does not aid diagnosis if the cervical length is very short (< 20 mm) or very long (> 30 mm) and in those cases, the sample collected may be discarded. For example, a cervical length of 30 mm or more is considered normal and has a negative predictive value that preterm birth will not occur that is 96% to 100%.[8] The fFN culture could then be discarded and the woman discharged home pending any other clinical problems that need to be addressed. If the cervical length is between 20 and 29 mm, however, the fFN is sent for analysis. A negative fFN would again be reassuring and indicates preterm birth is unlikely. In contrast, a positive fFN in this situation or a cervical length of less than 20 mm would warrant admission and initiation of preterm labor treatment.

The cut-off value for cervical length varies between 20 mm and 30 mm. Because the positive predictive value of both fFN and cervical length measurements is inexact, management should nor rely on these measurements alone when a woman has acute symptoms. Although preterm labor evaluation algorithms appear to reduce false-positive diagnoses and decrease unnecessary hospital admissions, the availability of cervical length ultrasound is limited in many areas and its performance requires specialized training and credentialing.

It can be challenging to formulate a plan for women who have some symptoms of preterm labor but no cervical dilation. The March of Dimes has provided recommendations for this situation that may prevent unnecessary hospitalization or interventions.[3] If gestational age is less than 34 weeks, the March of Dimes recommends fFN and/or cervical length (CL) measurement by transvaginal ultrasound. If fFN or CL measurement is not done, or if the woman is at 34 gestational weeks or more, a repeat assessment of cervical dilation by digital cervical examination 2 hours after the initial assessment should be done, ideally by the same examiner. If the fFN is negative or the CL measurement is 25 mm or more, or if there is no change in the cervix detected by digital cervical examination, the March of Dimes recommends the woman be discharged home.[3]

Treatment of Preterm Labor

For women in active preterm labor between 34 and 37 weeks' gestation, the midwife should consult with a physician regarding a collaborative management plan, depending on individual practice guidelines and institutional resources. If the woman is less than 34 weeks' gestation and her initial cervical examination is more than 3 cm dilated or 80% effaced, preterm labor treatment should be initiated and the woman referred for immediate medical management. Many smaller institutions will transfer women in preterm labor at less than 36 weeks' gestation to a tertiary center, because tertiary centers have specialized neonatal care available. Midwives in all practice settings should be aware of the current preterm labor treatment recommendations and initiate these interventions as the clinical situation requires. Any other abnormalities encountered by the midwife during the initial data collection should be managed and treated according to individual practice guidelines.

In the past, strategies such as bedrest, home contraction monitoring, maintenance tocolysis, hydration, periodontal care, and abstinence from intercourse were used to prevent preterm labor and birth. Evidence has not found these methods to be effective in reducing the preterm

birth rate; thus, they are no longer used as specific preventive or treatment therapies.[9]

There is also extensive evidence regarding the use of tocolytic medications to prolong pregnancy. Several different agents have been studied over the past 50 years, but none has effectively reduced the preterm birth rate or decreased neonatal mortality and morbidity (**Table 29-2**).[9,10] It is important to remember that tocolysis is contraindicated if the woman has any condition that requires labor such as intrauterine fetal demise, nonreassuring fetal status, severe preeclampsia, chorioamnionitis, lethal fetal anomaly, maternal bleeding with hemodynamic instability, or any maternal contraindication to use one of the tocolytic agents.[9]

Table 29-2 Tocolytic Medications Used to Treat Preterm Labor

Mechanism of Action	Dose	Common Maternal Side Effects	Fetal Effects	Contraindications/Cautions
Nifedipine (Procardia)				
Calcium channel blocker that directly blocks calcium ion influx through cell membrane and release of intracellular calcium from sarcoplasmic reticulum. This inhibits myometrial contraction	Optimal dose has not been established Common regimen is loading dose of 20–30 mg orally given in divided doses Then 10–20 mg orally every 3–8 hours	Peripheral vasodilator, may induce transient nausea, flushing, headache, palpitations, hypotension, dizziness, tachycardia	Secondary to maternal hypotension	Contraindications: Preload-dependent cardiac disorder, left ventricular dysfunction, congestive heart failure or hemodynamic instability Do not use concurrently with terbutaline or magnesium sulfate
Indomethacin (Indocin)				
Cyclooxygenase (COX) inhibitor reduces prostaglandin production by COX	Loading dose: 50–100 mg orally or 50 mg rectally Then 25–50 mg orally every 6 hours, for 48 hours	Gastrointestinal: nausea, vomiting, reflux, gastritis Platelet dysfunction	Premature closure of ductus arteriosus Oligohydramnios	Contraindicated for women with platelet dysfunction, bleeding diathesis, hepatic dysfunction, gastrointestinal ulcerative disease, and asthma if sensitive to aspirin Not recommended for more than 48 hours of continuous use Not recommended for women at ≥ 32 weeks' gestation
Magnesium sulfate				
Mechanism of action unknown but probably competes with calcium at cell membrane which reduces calcium available for myometrial contraction	Loading dose: 4–6 g intravenously (IV) over 20–30 minutes Then 2 g/hour by continuous IV infusion (pump required)	Flushing, nausea, blurred vision, headache, lethargy, muscle weakness, hypotension Rare effects: pulmonary edema, respiratory or cardiac arrest	Neuroprotective, Decreased fetal heart rate variability Decreased neonatal tone	Contraindicated for women with impaired renal function, myasthenia gravis, cardiac conduction defects Do not use concurrently with nifedipine Toxicity: loss of patellar reflexes, decreased urine output < 30 mL/hr, respiratory rate < 12/min Toxicity risks increase with serum creatinine > 1.0 mg/dL
Terbutaline (Brethine)				
Beta-agonist binds to beta-2 adrenergic receptors that cause a chain reaction resulting in depletion of intracellular calcium thereby blocking myometrial receptors. Receptors can become desensitized with prolonged use thereby diminishing effectiveness.	Varying doses used Common regimen is 0.25 mg subcutaneous (SQ) injection every 20 to 30 minutes up to 4 doses Then 0.25 mg SQ every 3-4 hrs. May be administered by continuous intravenous infusion	Tachycardia, peripheral vasodilation, hypotension, bronchial relaxation, pulmonary edema, hyperglycemia, myocardial infarction.	Tachycardia, neonatal hypoglycemia	Contraindications: tachycardia sensitive cardiac disease, poorly controlled hypertension, poorly controlled diabetes. Use with caution in women with hemorrhage as tachycardia and hypotension can complicate symptoms of hemorrhage The FDA has a black box warning that terbutaline should not be used for tocolysis for more than 72 hours secondary to the risk for maternal cardiac problems and recommends against use of oral terbutaline secondary to lack of proven effectiveness. However ACOG states terbutaline can be used for short term inpatient use

Abbreviations: ACOG, American College of Obstetricians and Gynecologists; FDA, U.S. Food and Drug Administration.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 171: management of preterm labor. *Obstet Gynecol.* 2016;128:931-933[9]; Nijman TA, Vliet EO, Koullali B, Mol BW, Oudijk MA. Antepartum and intrapartum interventions to prevent preterm birth. *Semin Fetal Neonatal Med.* 2016;21:121-128[10]

Tocolytics

All tocolytic agents slow uterine contractions to some degree, but at best they will delay birth for only 2 to 7 days.[9,10] In the past, magnesium sulfate and beta-mimetics (terbutaline [Brethine]) were the most commonly used tocolytic medications. More recently, research has shown that calcium-channel blockers (nifedipine [Procardia]) and prostaglandin synthetase inhibitors (indomethacin [Indocin]) have the best clinical efficacy and a lower incidence of

toxicity and maternal side effects when compared to magnesium sulfate and terbutaline.[9] As a result, terbutaline is no longer recommended for acute tocolysis, and magnesium sulfate should be reserved for clinical situations in which nifedipine and indomethacin are contraindicated or fetal/newborn neuroprotection is sought.[9]

Regardless of the agent chosen for tocolysis, the primary goal is to delay birth long enough to administer a full course of corticosteroids to facilitate fetal lung development and arrange for transfer of the woman to a tertiary center if needed. These two interventions have demonstrated dramatic improvements in neonatal outcomes and are the mainstay of preterm labor management.

Corticosteroids for Fetal Lung Maturity

The administration of corticosteroids to women in preterm labor between 24 and 34 weeks' gestation has been conclusively shown to improve fetal lung maturity and decrease other morbidities associated with prematurity.[9] Either betamethasone (Celestone) 12 mg intramuscularly (IM) given in two doses 24 hours apart or dexamethasone (Decadron) 6 mg IM given in four doses 12 hours apart can be used.[9] Once the woman has received a complete course of steroids, tocolysis is discontinued. If she does not give birth, the full steroid course can be repeated once as a "rescue dose" if more than 2 weeks has passed, the gestational age is less than 33 weeks, and the woman has a risk of birth within 7 days.[9] Some evidence suggests that a "rescue dose" may also be beneficial when the time period since the last course of steroids is 7–14 days.[9] Newer research suggests that administration of steroids in the late preterm period may also be beneficial.[11] Therefore, in women with a gestational age of 34 0/7 to 36 6/7 weeks at risk of preterm birth within 7 days who have not yet received steroids, this treatment should be considered.[9]

Group B Streptococcus Prophylaxis

Another important intervention in the treatment of preterm labor is the administration of antibiotics to prevent group B *Streptococcus* (GBS) disease. Preterm infants are especially susceptible to GBS sepsis, and the woman experiencing preterm labor will likely have unknown colonization status because the standard screening culture is not performed until 35 to 36 weeks' gestation. All women in preterm labor prior to 37 weeks with unknown GBS status should receive antibiotic prophylaxis.[12] The Centers for Disease Control and Prevention (CDC) website maintains updated GBS treatment guidelines and algorithms for GBS prevention strategies.[12]

Magnesium Sulfate for Neuroprotection

In addition to having some tocolytic properties, magnesium sulfate reduces the risk of cerebral palsy in infants born prior to 32 weeks' gestation.[9] Many institutions now include administration of magnesium sulfate as part of preterm labor management. Although the exact mechanisms of fetal neuroprotection are unknown, it is hypothesized that the magnesium sulfate

improves fetal cerebral blood flow and safeguards the fetus against hypoxic stress. Given that prematurity and low birth weight are the most significant risk factors for cerebral palsy, fetal neuroprotection has the potential to greatly improve the neurodevelopmental future of children born prior to 32 weeks' gestation.

Institutions that use magnesium sulfate for fetal neuroprotection should establish written guidelines and dosing protocols for this therapy.[7] These guidelines should also include an informed consent process and shared decision-making discussion with the woman and her family regarding the risks and potential benefits of magnesium sulfate in preventing cerebral palsy. It is important for the midwife to be aware of these guidelines and to appreciate the distinction of using magnesium sulfate for this indication *separately* from tocolysis. More importantly, the full dosing protocol for neuroprophylaxis differs from the magnesium sulfate doses used in the past for tocolysis and the dosing regimen currently used for treatment of preeclampsia/eclampsia.

Follow-Up

Women admitted for treatment for preterm labor will typically remain in the hospital for at least 24 hours. Once the full course of steroids is completed, tocolysis is usually discontinued and expectant management is resumed if the woman has not given birth. Women with advanced cervical dilation may need prolonged inpatient care and monitoring, especially if they are at a periviable, extremely preterm, or very preterm gestational age. The midwife can maintain an active supportive role in the woman's care during hospitalization and discuss the continued management plan with the physician as the clinical scenario changes. Prior to the woman's discharge from the hospital, the midwife may be involved in reassessment for preterm birth risk factors and initiation of any necessary treatments or interventions.

Outpatient monitoring will include weekly prenatal visits for the remainder for the pregnancy with either the midwife or the physician, or both, according to the plan of care. The woman should also be counseled about any signs of preterm labor and advised to call if any symptoms are noted. If symptoms do occur, the woman should be reassessed promptly in the hospital and the appropriate treatments repeated if needed.

Post-Term and Late Term Pregnancy

Concerns over the increased risks associated with giving birth during late term and post-term gestational weeks, particularly perinatal mortality, have prompted investigation of interventions to improve outcomes. The primary intervention studied has been induction of labor. Research has sought to determine whether routine induction improves outcomes and at which gestational age, and to identify any associated risks, especially an increased rate of cesarean births.

Expectant Management Versus Induction of Labor

Three meta-analyses comparing routine induction and expectant management for late term and post-term pregnancy have been conducted and produced slightly different results.[13-15] The authors of a 2012 Cochrane Review concluded that routine induction at 41 weeks results in a lower perinatal mortality rate (0.03% or 1/3285 birth vs. 0.33% or 11/3238 births, respectively) without an increase in cesarean births.[13] In the meta-analysis by Wennerholm et al., the authors found a lower incidence of cesarean birth and meconium aspiration syndrome with induction of labor at 41 weeks as compared to expectant management, and no difference in the perinatal mortality rate with the two management options.[14] Sanchez-Ramos et al. reported a lower rate of cesarean births and perinatal mortality with induction at 41 weeks as compared to expectant management, although the difference in the rates of perinatal mortality did not reach statistical significance.[15]

The risk of cesarean birth associated with induction of labor for late term or post-term gestation must be considered separately from the risk of cesarean associated with routine induction at any gestational age. The latter is discussed in the “Induction of Labor” section in this chapter. The evidence is clear that induction of labor for later term and post-term pregnancy does not increase the risk of cesarean, and may actually lower the incidence of cesarean birth.[13-15]

Based in this evidence, the American College of Obstetricians and Gynecologists (ACOG) supports considering induction between 41 0/7 and 41 6/7 weeks. Induction of labor is recommended at 42 0/7 weeks and indicated for all women who reach 42 6/7 weeks’ gestation based on the consensus of evidence that there is an increased risk of perinatal mortality and morbidity at that time.[16]

Expectant management of women with late term and post-term pregnancy includes antepartum fetal surveillance such as a nonstress test or a biophysical profile. Fetal surveillance has not been shown to decrease perinatal morbidity and mortality,[15] as studies in women who are post term have not included an unmonitored control group for ethical reasons. Regardless, starting antepartum fetal surveillance at 41 weeks’ gestation provides reassurance and is a commonly accepted practice.

Midwifery Management of Women at Post-Term Gestational Ages

When a pregnant woman approaches 41 0/7 weeks’ gestation, prenatal care must include a

discussion of management options for late term and post-term pregnancy. During this discussion, it is important to clearly separate the evidence on (1) the risks of late term and post-term pregnancy, and (2) the outcomes comparing induction of labor and expectant management with fetal surveillance as care options for late term and post-term pregnancy.

Principles of shared decision making can best guide the discussion which includes an explanation of the evidence showing increased risk after 41 weeks, and evidence on pros and cons of the different management options. These principles include the use of absolute risk as opposed to relative risk, and may include use of a visual aid such as a pictograph.[17] Relative risk versus absolute risk is important in this context. Perinatal mortality is a rare outcome, and the absolute risk of stillbirth and early neonatal death in uncomplicated pregnancies is small. The use of relative risk to explain this numeric evidence can introduce unintended bias.[17]

A discussion of the woman's values, goals, and preferences as they relate to the options for care is another principle of shared decision making and is a critical component of midwifery management of women with a late term and post-term pregnancy. Some women are anxious to end their pregnancies at the soonest possible time and will readily request induction of labor at 41 weeks. Some women find any increase in risk of perinatal mortality unacceptable and choose induction to mitigate this risk. Other women have a strong preference to avoid induction and may choose to await spontaneous labor.

If a woman declines induction at 42 0/7 weeks' gestation, consultation with a physician usually occurs for a continued plan of care. Careful fetal surveillance must be recommended, as well as ongoing discussion with the woman regarding the risks and benefits of induction versus awaiting spontaneous labor. Any consultation, results of fetal surveillance, and details of the discussion with the woman are documented in the health record.

Prelabor Rupture of Membranes

Prelabor rupture of membranes (PROM) is defined as rupture of the membranes prior to the onset of labor. This condition is also referred to as premature rupture of the membranes, although the use of the word "premature" may be confusing in this context because it is often used to refer to gestational age. When this event occurs in women who are less than 37 weeks' gestation, it is referred to as Premature prelabor rupture of membranes (PPROM). Prelabor rupture of the membranes at term gestation is a normal process associated with physiologic weakening of the amniotic membrane as labor nears. PPROM, in contrast, is thought to result from some type of underlying pathology, such as infection. The recommended management differs considerably for term and preterm PROM; thus, these conditions are considered separately here.

Evaluation for Ruptured Membranes

The diagnosis of PROM is clinical and based on evaluation of fluid present in the vagina. A history of a sudden gush of fluid that soaks underwear is a strong indication that PROM has occurred. Some women will report "feeling wet" or an intermittent gush of a small amount of fluid; in these cases, the diagnosis can be more difficult to make. Evaluation of the woman with possible ruptured membranes is reviewed in the *First Stage of Labor* chapter.

Premature Prelabor Rupture of Membranes

Management of PPROM and PROM at term can be either expectant or treated with induction of labor. In general, the fetus will benefit from a longer period in utero unless other complications are present. Unlike in women with term PROM, subclinical or clinical infection is often present in women who present with PPROM. A substantial number of women with preterm PROM have clinically diagnosed infection; 15% to 20% have chorioamnionitis and 15% to 20% have postpartum endometritis.[17]

The management of PPROM is based on weighing the risks of prematurity against the risks of infection with prolonged latency and delayed delivery. In general, the latency period with PPROM lasts an average of approximately 1 week with expectant management, but in some cases, it may last several weeks. Pregnant women with confirmed PPROM should be referred to a consulting physician for the continued plan of care. The management decision at the time of diagnosis is based on gestational age, presence of other complications such as infection or preterm labor, fetal well-being, fetal lung maturity, and access to a neonatal intensive care unit.

PPROM at Less Than 23 Weeks

In situations where PPROM occurs prior to 23 weeks' gestation, an individualized collaborative approach that includes the neonatal team is necessary to best determine the course of treatment. GBS prophylaxis, tocolysis, corticosteroids, and magnesium sulfate for fetal neuroprotection are not recommended if the fetus is at a previable gestational age.[18] Extensive counseling and shared decision making with the woman and her family should occur.

The midwife can fill a valuable role by listening and helping the woman understand all the complex information she will be given in this situation.

PPROM at 23 to 33 6/7 Weeks

In gestations between 23 and 33 6/7 weeks, expectant management is preferred if there are no clinical signs of infection, fetal compromise, or labor. Transfer of the woman to a tertiary center should be arranged if necessary. In addition, a single course of corticosteroids is administered to improve fetal lung maturity, and antibiotics are administered to the woman to prolong the latency period. The regimen for latency antibiotics varies among institutions but always includes an antibiotic that is appropriate for GBS prophylaxis.[12] The CDC guidelines for GBS prophylaxis, which can be found on the CDC website, detail evaluation and management of women with PPROM with regard to culturing for GBS, time period over which to administer antibiotics, and management if the woman does or does not go into labor. ACOG recommends against the use of tocolytics in this situation. Magnesium sulfate for fetal neuroprotection is controversial but should be considered if the gestational age is less than 32 0/7 weeks and birth is thought to be imminent within the next 12 to 24 hours. Digital cervical examinations are contraindicated, so as to reduce the risk of chorioamnionitis. A baseline cervical examination should not be performed.

PPROM After 34 Weeks

The gestational age at which infection risk outweighs the risks associated with prematurity is not clear. ACOG recommends induction of labor for all women with PPROM at 34 weeks' gestation or greater.[18] Administration of betamethasone to reduce respiratory morbidity in the newborn is now recommended between 34 0/7 and 36 6/7 weeks' gestation, including when PPROM occurs.[18] A collaborative management plan can be considered between the midwife and the physician in these situations, and will also depend on institutional and individual practice guidelines for midwifery management of women in preterm labor.

The Term PROM Controversy

The primary risk associated with term PROM is development of infection, which is termed either intra-amniotic infection or chorioamnionitis. Factors most strongly associated with an increased risk of infection are digital cervical examinations, maternal colonization with GBS, duration of rupture, duration of labor, and duration of latency period (i.e., the time between rupture of membranes and onset of labor).[19]

The two management options for women with term PROM are induction of labor and expectant management. Primary questions of concern are which management option results in lower infection rates, and whether one of these options is associated with more cesarean births. The 1996 TermPROM study is the most relevant contemporary study due to its randomized design and large sample size.[20] In this study, neonatal infection rates and cesarean birth rates were similar in women who underwent induction and those managed expectantly,

but there was a higher rate of maternal infection in those women in the expectant management group. Several study limitations likely affected the risk of maternal infection in the expectant management group including an atypical diagnosis of chorioamnionitis, more digital cervical examinations in the expectant management group, and no prenatal GBS screening and therefore no antibiotic prophylaxis during labor for GBS-positive women.[21]

ACOG recommends induction of labor as the optimal management approach, but recognizes expectant management as an acceptable option for women who decline induction.[22] The American College of Nurse-Midwives' (ACNM) Position Statement on Premature Rupture of Membranes at Term reaffirms the woman's rights to self-determination and supports the option of expected management for low-risk women.[23] **Table 29-3** summarizes these recommendations.

Table 29-3 Criteria for Expectant Management of Prelabor Rupture of Membranes

Full term (> 37 0/7 weeks)
Singleton pregnancy
Vertex presentation
Clear amniotic fluid
No fever
No infection including HIV; hepatitis B and C
Not a known group B *Streptococcus* carrier (GBS negative)
No evidence of risk for fetal acidemia in fetal heart rate pattern
No health or obstetric condition that requires immediate induction of labor

Based on American College of Nurse-Midwives position statement: premature rupture of membranes at term. ACNM Division of Standards and Practice, Clinical Practice Section 2008; reviewed 2012. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000233/PROM%20Mar%202012.p

Women who choose or require induction of labor should be admitted and a plan of care made, including decisions regarding cervical ripening and/or oxytocin infusion. Physician consultation may be indicated depending on institutional guidelines and the individual clinical scenario. If induction is chosen, it is important to allow adequate time for the latent phase of labor to avoid cesarean birth for failed induction. Multiple cervical examinations are a primary risk factor for infection, so digital cervical examinations should be minimized during induction.[18,19]

If expectant management is chosen, the management plan should include where the woman will await onset of labor and for how long, and how maternal and fetal well-being will be monitored during this time. There is little evidence on which to base the decision of where the woman should remain until labor starts,[17] and having her return home for a predetermined time may be considered. A woman who returns home for expectant management should be advised to monitor her temperature (with a thermometer) every 2 to 4 hours as well as contraction frequency and fetal movement. It is important to also counsel the woman to avoid inserting anything into her vagina. The woman should be advised to call immediately if she has a temperature of 38°C or higher, decreased fetal movement, signs of active labor, and either

meconium-stained or foul-smelling amniotic fluid. Plans for follow-up evaluation should also be made.

In the TermPROM study, the median latency duration was 33 hours and 95% give birth within 95–107 hours after rupture.[20] The optimal maximum duration of expectant management is not known. The TermPROM study allowed up to 96 hours before induction was performed.[20] Women choosing expectant management may elect to have their labor induced at any time.

Women with ruptured membranes must be monitored for developing infection regardless of the management option chosen. Fetal or maternal tachycardia, fever, uterine tenderness, and foul-smelling fluid are all signs of possible chorioamnionitis. Any of these signs or symptoms requires admission, physician consultation, and prompt initiation of the appropriate treatment interventions. To avoid infection, digital cervical examinations should be minimized during induced or spontaneous labor.[18,19]

Intrauterine Infection

Intrauterine infection or inflammation refers to infection of the amniotic sac, the amnion, and the chorion.[24,25] *Chorioamnionitis* is the historical term, but refers to a heterogeneous set of conditions characterized by infection and/or inflammation, which have differing degrees of severity. Therefore, the terms *intrauterine infection*, *intrauterine inflammation*, or *intrauterine infection and inflammation* (Triple I) have been proposed as replacements. The term *intra-amniotic infection*, which has also been proposed as a replacement for chorioamnionitis, is used in this chapter.

A common cause of intra-amniotic infection is ascending bacteria from the woman's lower genital tract. Organisms such as group B *Streptococcus*, *Escherichia coli*, *Ureaplasma urealyticum*, *Fusobacterium* species, and *Mycoplasma hominis* have all been implicated as causative pathogens.[26] Because the amniotic membrane provides some protection against fetal infection, prolonged rupture of membranes and long labor are risk factors for intra-amniotic infection. Manipulative vaginal or intrauterine procedures and frequent digital cervical examinations are also associated with an increased risk of intra-amniotic infection, especially once the membranes have ruptured.[24] Many of these factors are interrelated and, therefore, their independent effects have not been fully established. For example, an increased number of cervical examinations during labor is associated with longer labors and longer duration of membrane rupture, both of which are also associated with an increased risk of infection.[24,27]

The definitive diagnosis of intra-amniotic infection can be made only after the birth through histological examination of the placenta and membranes.[24,25] Maternal fever during labor is the primary clinical marker of intra-amniotic infection and is associated with 95% of cases.[26] However, fever can also be caused by maternal dehydration, overheated room temperature, and possibly prolonged time in the shower or tub. Furthermore, maternal fever is associated with epidural analgesia. Given these ambiguities, a National Institutes of Health Expert Panel Workshop held in 2015 evaluated the research on intra-amniotic infection and released new guidelines for diagnosis and management of this condition (**Table 29-4**).[25]

Table 29-4 Diagnostic Criteria for Intrauterine Infection, Intrauterine Inflammation, or Both

Diagnosis	Diagnostic Criteria
Isolated maternal fever	Oral temperature of 38–38.9°C that persists when the temperature measurement is repeated after 30 minutes
Suspected intrauterine infection, inflammation, or both (Triple I, intra-amniotic infection)	Oral maternal temperature of ≥ 39°C[a] without other symptoms *or* Maternal intrapartum fever and one or more of the following: Fetal tachycardia: FHR > 160 bpm Maternal leukocytosis: WBC count > 15,000/mm^3 if corticosteroids are not given Purulent discharge from the cervical os
Confirmed intra-amniotic infection	All of the above plus: Amniocentesis-proven infection via positive Gram stain and

	low glucose or positive amniotic fluid culture
	Placental histopathology with diagnostic features of infection

Abbreviations: FHR, fetal heart rate; WBC, white blood count.

[a] The National Institutes of Health Expert Panel identified an oral temperature of 39°C or greater without other symptoms as being an isolated maternal fever, but the American College of Obstetricians and Gynecologists included this finding as part of the criteria for suspected intra-amniotic infection.

Based on American College of Obstetricians and Gynecologists. Committee Opinion No. 712: intrapartum management of intraamniotic infection. *Obstet Gynecol.* 2017;130:e95-e101[24]; Higgins RD, Saade G, Polin RA, et al. Evaluation and management of women and newborns with a maternal diagnosis of chorioamnionitis. *Obstet Gynecol.* 2016;127:426-436.[25]

These definitions distinguish between suspected versus confirmed intra-amniotic infection and provide standardized temperature criteria for isolated maternal fever. The Expert Panel workshop criteria included women with temperature 39°C or greater but no other clinical risk factors in the isolated maternal fever group, whereas ACOG recommends that these women be treated as though they have suspected intra-amniotic infection.[24]

Some risk factors for intra-amniotic infection are modifiable. Thus, preventive measures such as promoting hydration during labor and avoiding digital cervical examinations are important components of midwifery practice. If the maternal temperature begins to rise, an intravenous fluid bolus can be given initially and the temperature rechecked in an hour.

Women with isolated maternal fever or suspected intra-amniotic infection are offered antibiotics in labor, and physician consultation or notification is indicated as well. Although no randomized trials have compared the various antibiotic regimens, broad-spectrum antibiotics such as ampicillin or penicillin, used in combination with gentamicin (Garamycin), are the most commonly recommended treatment for suspected infection. Administered intravenously until the woman gives birth, these antibiotics have consistently demonstrated the highest efficacy against the two leading causes of neonatal sepsis—namely, group B *Streptococcus* and *E. coli*. **Figure 29-2** shows the recommended dosing regimens, including alternatives for women with penicillin or ampicillin allergy.[24–26,28]

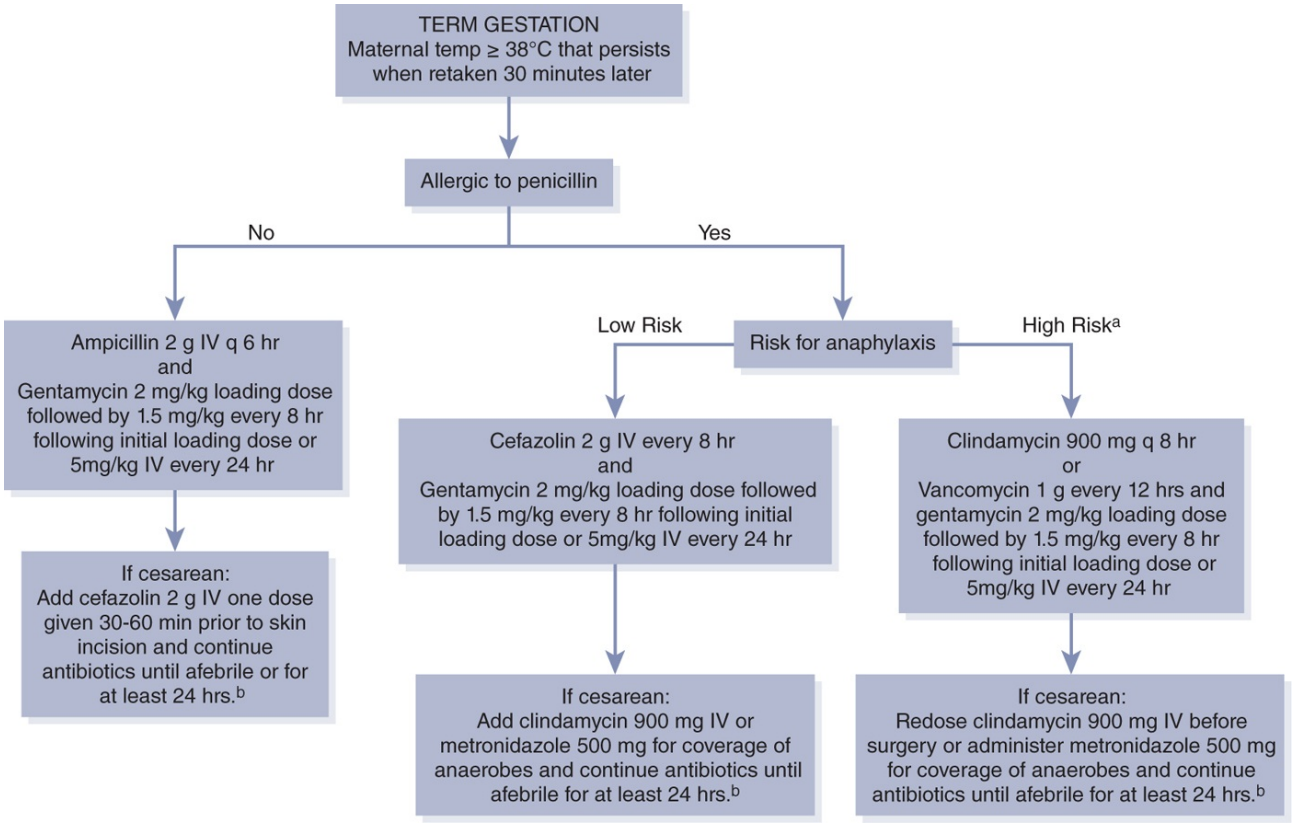


Figure 29-2 Treatments for isolated maternal fever and suspected intra-amniotic infection.

Abbreviations: g, gram; hr, hour; IV, intravenous; mg, milligram; min, minutes; q, every.

[a] High risk for anaphylaxis includes history of angioedema, respiratory distress, anaphylaxis, or urticaria (hives) after exposure to penicillin.

[b] Some authors suggest one dose following birth is sufficient. Thus, the duration of antibiotic administration after birth is based on institutional guidelines.

Based on American College of Obstetricians and Gynecologists. Committee Opinion No. 712: intrapartum management of intraamniotic infection. *Obstet Gynecol.* 2017;130:e95-e101[24]; Higgins RD, Saade G, Polin RA, et al. Evaluation and management of women and newborns with a maternal diagnosis of chorioamnionitis. *Obstet Gynecol.* 2016;127:426-436[25]; Fahey JO. Clinical management of intra-amniotic infection and chorioamnionitis: a review of the literature. *J Midwifery Womens Health*. 2008;53:227-235[26]; Fishman SG, Gelber SE. Evidence for the clinical management of chorioamnionitis. *Semin Fetal Neonatal Med.* 2012;17:46-50.[28]

Women who are being given penicillin or ampicillin for GBS prophylaxis are not sufficiently treated for intra-amniotic infection. If a woman who is receiving penicillin for GBS prophylaxis develops a fever during labor, a broad-spectrum antibiotic that is effective against anaerobic bacteria must be initiated.

Oral acetaminophen (Tylenol) 975 mg should also be given. Reducing the woman's temperature has been shown to improve fetal acid–base balance.[26] The woman’s condition and labor progress should be monitored closely. Oxytocin should be used if labor is not progressing normally. Intra-amniotic infection is not an indication for cesarean birth. If a cesarean birth is performed, the woman may need additional anaerobic antibiotic coverage with either cefazolin (Ancef) or clindamycin to prevent postpartum endometritis. Once birth occurs, the placenta is sent to the pathology laboratory for histologic evaluation and the infant

closely monitored for any signs of sepsis or infection. Intravenous antibiotics are typically continued for at least one additional dose after the delivery, although many institutions will extend postpartum dosing until the woman has remained afebrile for at least 24 hours.[28]

Induction of Labor

Induction of labor refers to stimulation of uterine contractions through artificial means. Methods for inducing labor include both hormonal and nonhormonal approaches. The decision to induce labor involves assessing the risks associated with continuing the pregnancy versus the risks associated with labor induction for both the woman and the fetus. Induction of labor can be elective or medically indicated. Examples of evidence-based medical indications for an induction of labor include gestational hypertension, preeclampsia/eclampsia, fetal growth restriction, cholestasis of pregnancy, diabetes mellitus, fetal demise, intra-amniotic infection, oligohydramnios, and nonreassuring fetal status.[29] Other medical indications include prelabor rupture of membranes and post-term pregnancy. Induction of labor should occur only in a hospital and is contraindicated in any situation that precludes vaginal birth, such as placenta previa, vasa previa, transverse lie, umbilical cord prolapse, previous myomectomy entering the endometrial cavity, previous classical uterine incision, active genital herpes infection, or presence of a Category III fetal heart rate (FHR) tracing.[30]

Elective induction (i.e., no medical indication for induction) is controversial. Factors such as maternal or provider convenience, history of fast labors, maternal distance from the hospital, relief of physiologic pregnancy discomforts, avoidance of certain calendar dates or holidays, institutional staffing concerns, and patient satisfaction have all been cited as reasons for elective induction. Risks associated with induction of labor include uterine tachysystole, fetal intolerance of labor, postpartum uterine atony, and possibly an increased risk for cesarean section. Adverse outcomes include increased incidence of neonatal morbidity from iatrogenic prematurity if the estimated gestational age is incorrect and increased costs.[30]

The relationship between cesarean birth and induction of labor is complex. Induction for certain indications, such as late term and post-term pregnancy[13] and prelabor rupture of membranes at term,[21] is not associated with an increased risk of cesarean birth. In the early 2000s, a considerable body of evidence developed demonstrating that the risk of cesarean section is substantially increased with induction in nulliparous women, with most researchers finding the risk to be at least doubled.[30,31] These studies compared labor outcomes in women who were induced versus labor outcomes in women who experienced spontaneous labor at the same gestational age. Some researchers have noted that the proper comparison group for women who are induced is actually women at the same gestational age who are managed expectantly.[32] Several recent studies, including a systematic review that used this comparison group, demonstrated a decreased rate of cesarean births in women whose labors were induced when compared to women who were managed expectantly.[33-36] More recently, a small randomized controlled trial (RCT) found that induction was associated with a higher rate of cesarean section (30.5% vs 17.7%; relative risk [RR], 1.72; 95% confidence interval [CI], 0.96–3.06) in nulliparous women at 39 weeks' gestation whose cervix was not favorable.[37] Although this result was not statistically significant, it may be clinically significant and suggests further research is needed to better parse the individual clinical characteristics that may or may not increase the risk of cesarean section following induction of labor. It is likely that the relationship between induction and cesarean birth is affected by multiple factors, such

as nulliparity, obesity, lack of cervical ripening, gestational age, and labor management practices.[34]

A positive relationship between elective induction of labor and an increased rate of iatrogenic prematurity is clear, and the practice of elective induction prior to 39 weeks' gestation has come under close scrutiny as the adverse effects associated with late preterm birth have become clearer. Strict criteria for gestational age dating are now in place, and elective induction prior to 39 weeks' gestation is contraindicated.[29] Some institutions have adopted stringent guidelines for elective induction at term that also require a favorable Bishop's score, a signed consent form, and a preinduction checklist for providers.[38,39] The Bishop's score is a numeric assessment of four different characteristics of the cervix and fetal station; it is used to predict the success of induction.[40,41] The currently used version of the Bishop's score is shown in **Table 29-5**.[29,40,41] The ACNM Position Statement on Induction of Labor (2016) recommends against the practice of elective induction of labor; midwives in clinical practice will find valuable information in this document.[42]

Table 29-5 **The Modified Bishop Scoring System**

Point Value	0	1	2	3
Cervical position	Posterior	Midline	Anterior	
Cervical consistency	Firm	Medium	Soft	
Cervical length[a] (cm)	> 4	2–4	1–2	< 1
Cervical dilation (cm)	0	1–2	3–4	> 5
Fetal station[b]	–3	–2	–1/0	+1/+2

[a] The original cervical length categories were cervical effacement of 0–30%, 40–50%, 60–70%, and greater than 80%. Either the original percent effacement or centimeters of cervical length may be used.
[b] Station is based on a –3 to +3 range.

Based on Bishop EH. Pelvic scoring for elective induction. *Obstet Gynecol.* 1964;24:266-268[40]; Lange AP, Secher NJ, Westergaard JG, Skovgård I. Prelabor evaluation of inducibility. *Obstet Gynecol.* 1982;60(2):137-147[41]; American College of Obstetricians and Gynecologists. Practice Bulletin No. 107: induction of labor. *Obstet Gynecol.* 2009;114:386-397. [Reaffirmed 2016].[29]

Prior to the initiation of any cervical ripening or induction methods, the woman should be counseled about the medical indication for induction as well as the risks and benefits to her and her fetus that are associated with induction and expectant management. In addition, a collaborative management plan between the midwife and the consulting physician regarding the plan of care for cervical ripening and induction of labor is required. **Table 29-6** summarizes key midwifery considerations when caring for women who require an induction of labor and/or cervical ripening.

Table 29-6 **Clinical Considerations Prior to Induction of Labor and/or Cervical Ripening**

1. Physician consultation should occur prior to the initiation of cervical ripening or oxytocin induction. In some cases, a collaborative management plan is required.
2. The medical indications for induction should be discussed with the woman and her support person(s). The

risks and benefits of each proposed agent (or technique) should be thoroughly reviewed. The woman's preferences should be taken into consideration. This discussion should be documented in her health record.

3. The pre-induction (or cervical ripening) database should include the following information:
   a. Documentation of the gestational age and criteria used to establish the final estimated due date.
   b. Bishop's score.
   c. Indications for induction or cervical ripening.
   d. No contraindications for induction.
   e. Clinical pelvimetry.
   f. Confirmation of a cephalic presentation.
   g. Confirmation of Category I fetal heart rate pattern. Induction may proceed with oxytocin if a Category II fetal heart rate pattern is present, following consultation with a physician.
4. The physician should be notified if the woman declines a medically indicated induction of labor.
5. Caution should be exercised when prescribing or recommending alternative methods of cervical ripening or induction of labor not supported in the scientific literature.
6. During an induction, practices that encourage the normal process of labor (e.g., ambulation with telemetry, oral hydration) should be encouraged.
7. Water immersion in the shower or tub (with waterproof fetal telemetry monitoring) may be considered during induction of labor with oxytocin.

Cervical Ripening Techniques

As pregnancy approaches full term, the cervix gradually begins to soften, efface, and move anteriorly in the vaginal vault. This process, which is commonly referred to as "ripening," occurs as a result of normal remodeling of the collagen fibers and other glycoprotein connective tissues within the cervix. Cervical ripening occurs more readily in multiparous women, and the process may begin in the days to weeks before the onset of labor.

The degree of ripening can be quantified using the modified Bishop's score, which takes into consideration all of the factors associated with cervical remodeling (**Table 29-5**). This scoring system indicates whether the woman's cervix is "favorable" or "unfavorable" with regard to predicting the success of labor induction and is the best predictor of induction success known to date.

For example, women who have a modified Bishop's score higher than 8 are considered to have a favorable cervix and a high probability for vaginal birth with induction of labor.[29] By comparison, women with a Bishop's score of 6 or lower have an unfavorable cervix, such that cervical ripening may improve their labor induction success rates. Cervical ripening agents can advance the Bishop's score by acting directly on the glycoproteins within the cervix (hormonal) or through mechanical dilation of the cervix. Because cervical dilation is considered to be the component of the Bishop's score that is most predictive of induction success, mechanical methods are often used concurrently either with hormonal ripening or during oxytocin induction. No cervical ripening method should be employed before 40 weeks' gestation unless there is a medical indication for induction.

Foley Balloon Catheter

The most common method of mechanical dilation is use of a Foley balloon catheter, although hydroscopic vaginal dilators (laminaria) are still used in some locations. Using aseptic technique, a Foley catheter with a 30-cc balloon is inserted directly into the cervix and advanced beyond the internal os. The balloon is then inflated to the full 30 cc with sterile water or saline, and the end of the catheter taped to the woman's leg, applying mild tension. Insertion can be done with direct visualization of the cervix using a speculum or during a digital examination. The balloon falls out on its own when the cervix dilates to 3 cm, which typically occurs within 6 to 12 hours. The insertion procedure may cause some discomfort for the woman and lead to persistent lower abdominal cramping. Accidental rupture of the membranes or cervical bleeding can also occur. The risks, benefits, and expected side effects should be explained to the woman prior to the insertion of a balloon catheter and documented with a procedure note in the woman's medical record.

Prostaglandin Agents for Cervical Ripening

The two hormonal agents currently used for cervical ripening indications are prostaglandin E_2 (dinoprostone [Prepidil, Cervidil]) and the synthetic prostaglandin E_1 analogue (misoprostol [Cytotec]). The current preparations, dosing regimens, and clinical considerations for these agents are outlined in **Table 29-7**.[43-45] A considerable amount of research has compared hormonal agents alone or in combination with mechanical dilation. This body of evidence has conclusively demonstrated that cervical ripening prior to initiation of oxytocin shortens the time of induction onset to birth and may increase the vaginal birth rate. To date, no preferred strategy for cervical ripening has emerged.[43] Vaginal administration of misoprostol is the intervention most likely to result in birth within 24 hours but is associated with the highest incidence of tachysystole; by comparison, use of a Foley catheter for cervical ripening is associated with the lowest rate of tachysystole. Oral misoprostol has the lowest rate of cesarean birth.[44]

Table 29-7 **Hormonal Cervical Ripening Agents**[a]

Drug: Generic (Brand)	Dose and Route	Onset of Action (min)	Peak Plasma Level (min)	Duration of Action (hr)	Mean Time to Sustained Uterine Activity (min)	Clinical Considerations
Prostaglandin E_1						
Misoprostol (Cytotec)	25–50 mcg orally every 3–6 hours	12 ± 3	20–30	2	90	Contraindicated in women with a scarred uterus Inpatient only with continuous fetal heart rate monitoring Vaginal doses of 50 mcg are associated with higher incidence of uterine tachysystole and are not recommended Oxytocin may be started 4 hours after last dose
Misoprostol (Cytotec)	25 mcg every 3–6 hours posterior vaginal fornix	20.9 ± 5.3	60–80	4.5–5	106	
Prostaglandin E_2						
Dinoprostone (Prepidil)	0.5 mg in 2.5 mL syringe; endocervical gel	Rapid onset	60–120	Variable	127	Inpatient only with continuous fetal heart rate monitoring Left in place for up to 12 hours Remove insert if more than 5 contractions/10 minutes (tachysystole) Oxytocin may be started 30–60 minutes after removal
Dinoprostone (Cervidil)	10 mg 0.3 mg/hour vaginal	Rapid onset	Unknown	Variable	Unknown	May be repeated every 6 hours × 3 doses Can be used for outpatient ripening in selected women Monitor fetal heart rate for 1–2 hours after each dose Oxytocin may be started 6–12 hours after last dose

[a] Cervical ripening agents are contraindicated if there is an abnormal FHR tracing or more than 3 contractions in a 10-minute window.

Based on American College of Obstetricians and Gynecologists. Practice Bulletin No. 107: induction of labor. *Obstet Gynecol.* 2009;114:386-397. [Reaffirmed 2016][29]; Yount S, Lassiter N. The pharmacology of prostaglandins for induction of labor. *J Midwifery Womens Health.* 2013;58:133-144.[43]

Many variables—such as provider preference, availability, and institutional guidelines—will determine which agents or methods are used. All hormonal agents can cause excessive uterine activity (tachysystole) or induce labor, so assessment of fetal well-being is an important prerequisite before hormonal ripening is started. The midwife should monitor the woman's response to cervical ripening and remain vigilant for signs of tachysystole or fetal intolerance to uterine activity.

Misoprostol (Cytotec)

Misoprostol is available as a 100- or 200-microgram (mcg) tablet. This agent is approved by

the U.S. Food and Drug Administration (FDA) and marketed for managing the symptoms of peptic ulcer disease. The tablets are not formulated for vaginal use, and misoprostol is not FDA-approved for cervical ripening. Nevertheless, it is effective for cervical ripening and is commonly used on an off-label basis for this indication. Misoprostol is contraindicated for women who have had a prior cesarean section or uterine surgery.

Misoprostol may be administered either orally or vaginally. Due to the potential dosing inaccuracies from splitting up the tablets, the uterine response can be somewhat unpredictable and unreliable, especially with vaginal dosing. As a precaution, the dose is not usually administered if the woman experiences more than 3 contractions in a 10-minute period.

Terbutaline should always be readily available to treat tachysystole that is associated with abnormal or indeterminate FHR patterns. Interestingly, although caution is warranted, episodes of FHR decelerations that occur in response to misoprostol-induced tachysystole have not been associated with adverse neonatal outcomes in the studies that assessed the effectiveness of misoprostol for cervical ripening.

Induction of Labor Techniques

Membrane Sweeping

Membrane sweeping is a practice often performed in the outpatient setting for women at term who desire an intervention to speed the onset of labor. Membrane sweeping is not an induction agent per se, but has been shown to shorten the time interval to onset of labor. This procedure is reviewed in detail in the *Pregnancy-Related Conditions* chapter.

Amniotomy

Planned artificial rupture of the membranes (AROM) may be used as a method of labor induction either alone or concurrently with oxytocin. Studies have shown amniotomy to be more effective when the cervix is favorable, but individual response to amniotomy can be unpredictable and this technique can result in long intervals before the onset of regular uterine contractions.[29] There is a potential risk of infection from a prolonged labor if amniotomy is performed when the cervix is unfavorable. When amniotomy is combined with oxytocin, the induction-to-birth interval is decreased; however, the best timing for AROM has not been clearly established. In addition, once the membranes have been ruptured, the woman is committed to giving birth and no longer has the possibility of further cervical ripening or reverting to a period of expectant management if a trial of oxytocin fails to initiate labor. For women with cervical dilation of 4 cm or less, early amniotomy is not associated with adverse maternal or neonatal outcomes and may shorten labor by 2 hours or more.[45]

The primary risk associated with amniotomy is umbilical cord compression—or, more emergently, a prolapsed umbilical cord. Variable decelerations are commonly seen when the umbilical cord is compressed; the management of variable decelerations is discussed in the *Fetal Assessment During Labor* chapter. Umbilical cord prolapse is an obstetric emergency regardless of whether the membranes ruptured spontaneously or following amniotomy and is the primary reason why amniotomy should not be performed if the fetal head is not engaged in

the pelvis. See the “Obstetric Emergencies” section of this chapter for detailed management guidelines for a prolapsed umbilical cord.

Induction of Labor with Oxytocin (Pitocin)

Oxytocin is a small, nine-amino-acid peptide hormone produced in the hypothalamus and secreted from the posterior pituitary gland. This hormone is similar to vasopressin and has a direct antidiuretic effect on the kidney. Most importantly, oxytocin is a potent uterotonic agent that causes uterine contractions. Although pituitary secretion appears to be the primary source of oxytocin, this hormone is also synthesized and secreted by the placenta and fetus. Synthetic oxytocin, which is chemically identical to endogenous oxytocin, is one of the most commonly prescribed medications in midwifery practice today.

When this medication is used appropriately, the clinical benefits are undeniable, especially for treating postpartum hemorrhage. However, oxytocin is associated with adverse fetal and maternal outcomes and frequently implicated in obstetric litigation cases.[46] As a result, oxytocin is now considered a “high alert” medication, which mandates compliance with safety measures to reduce the increased risks of error that may result in maternal or neonatal morbidities. Some of the safeguards currently in place include standardized intravenous concentrations, mandatory use of an infusion pump, continuous FHR monitoring, and lower-dosing protocols.[46,47]

Oxytocin exerts its effect via agonist action on oxytocin receptors that are present in the uterine myometrium. These receptors become active or “upregulate” more than 100-fold during early labor. The uterotonic effect mediated by receptor function is theorized to be the reason for the marked interindividual variability in responses to this drug. The oxytocin receptors also downregulate or become desensitized following prolonged exposure to oxytocin, which may partially explain why women who are exposed to long-duration oxytocin therapy for labor induction are at increased risk for postpartum hemorrhage.[48,49]

The onset of action of synthetic oxytocin is 3 to 4 minutes, and the half-life is approximately 15 minutes. The uterine response reaches a steady state after 30 to 40 minutes.[49] The individual response to a particular dose is both variable and dose dependent: As the infusion rate increases, the frequency and intensity of contractions follow.

Oxytocin Dosing Regimens. The variability in the individual response to oxytocin requires that the medication be started at a small dose and titrated until the desired contraction response is met. Various dosing regimens have been proposed, including those based on the starting dose and on the frequency at which the dose is increased and the amount of increase. Regimens are generally referred to as “low dose” or “high dose,” but these terms are not well defined. Generally, the higher-dose regimens include a starting dose of 6 mU/min that is increased by 3–6 mU/min every 15–40 minutes. In contrast, the low-dose protocols are based on the pharmacokinetics of oxytocin and use a starting dose of 0.4–2 mU/minute, which is increased by 1–2 mU/minute every 40 minutes. High-dose regimens are associated with more uterine tachysystole, a shorter interval between starting oxytocin and adequate labor, and shorter duration of labor. The evidence regarding cesarean birth rates with high-dose versus

low-dose regimens has been inconsistent, although most studies have not found that high-dose protocols increase the cesarean rate.[49-51] The influence of provider behavior on cesarean rates with oxytocin augmentation is not known.

Despite a lack of standard protocols, the trend is to use low-dose regimens that call for waiting at least 30 to 40 minutes before increasing the dose, to allow for evaluation of the full effects of the prior dose. This time frame allows the oxytocin administered to reach a steady-state plasma concentration and reduces the chance of dysfunctional uterine activity or tachysystole. Moreover, during the first stage of labor, there may be a small baseline concentration of endogenous oxytocin in the woman's circulation, which may combine with the intravenous oxytocin infusion to produce a cumulative effect on uterine contractions.[45,49,52]

Side Effects and Adverse Effects of Oxytocin. Oxytocin is contraindicated for women who have contraindications to induction of labor. This agent has an antidiuretic effect, so it must be used with caution in women who need fluid restriction or who are at increased risk for pulmonary edema. Oxytocin can cause hypotension, tachycardia, and transient myocardial ischemia that results in EKG changes if given as a bolus dose.

The most commonly encountered side effect of oxytocin is uterine tachysystole. Tachysystole is defined as more than 5 contractions in 10 minutes averaged over a 30-minute window.[53] Due to the short half-life of oxytocin, decreasing or stopping an oxytocin infusion will rapidly slow the frequency of contractions. In many cases, uterine tachysystole does not result in obvious fetal compromise but still requires an oxytocin dose adjustment to allow for adequate fetal oxygenation in between contractions. Titration of the infusion is highly individualized, with some finesse required to find a balance between the desired effect and avoidance of uterine tachysystole. Women receiving oxytocin also require an assessment of maternal and fetal status every 15 minutes during the active phase of labor and every 5 minutes during second-stage labor.[47,52] This important safeguard ensures prompt recognition and treatment of tachysystole and abnormal FHR patterns.

Regardless of the contraction pattern, any sign of fetal compromise requires immediate discontinuation of the oxytocin infusion. Additional interventions include lateral repositioning of the woman, a 500-mL intravenous fluid bolus of lactated Ringer's solution, and administration of oxygen. A single dose of terbutaline 0.25 mg can be administered subcutaneously if these initial interventions fail to ameliorate the uterine contraction pattern. The midwife should notify the consulting physician and remain at the bedside with the woman until fetal well-being is ensured, in which case the oxytocin infusion can be resumed after 15 to 30 minutes or when indicated. The reader is referred to the *Fetal Assessment During Labor* chapter for suggested tachysystole management guidelines.

In some cases, induction with oxytocin takes longer than anticipated, especially if cervical ripening was clinically contraindicated or was ineffective in improving the Bishop's score. Desensitization ("downregulation") of the oxytocin receptor sites on the uterus from continuous exposure to oxytocin over a prolonged period of time may occur.[49] When this happens, oxytocin becomes less effective and further increases in the dose merely add to the risk of tachysystole and inefficient uterine contraction activity without necessarily dilating the cervix.

Prolonged use of oxytocin can also increase the risk of postpartum hemorrhage from uterine atony for the same reasons. Thus, it is important to be alert for this possibility and consider active management of third-stage labor to minimize the risk for postpartum hemorrhage in women who have been exposed to exogenous oxytocin for several hours (see the *Third Stage of Labor* chapter).

In addition to postpartum hemorrhage, the use of oxytocin is associated with an increased risk of uterine rupture. Uterine rupture in a woman who does not have a scar in the uterus is a rare event in the United States (< 1 incident per 10,000 pregnant women).[54] By comparison, in low-resource areas with limited access to cesarean birth, the incidence of uterine rupture is more common, especially among women with higher parity experiencing an obstructed labor. In developed countries, uterine rupture is almost exclusively associated with women attempting a trial of labor after cesarean (TOLAC). Although oxytocin for induction or labor augmentation is not contraindicated for women who are attempting TOLAC, higher doses significantly increase the risks of uterine rupture and should be avoided.

If the woman's condition is stable and she is not yet in active labor, it is reasonable to discontinue the oxytocin infusion after 10 to 12 hours. This conservative approach will correct any receptor downregulation and enable the woman to rest. It will also reduce the risk of postpartum uterine atony from a prolonged infusion. Moreover, this approach allows time for additional cervical ripening overnight if needed. The oxytocin can then be restarted the next day and the induction plan will proceed accordingly. For some women, achieving a vaginal birth from a labor induction requires considerable patience on the part of the caregivers. The criteria for a diagnosis of "failed induction" are not clearly defined in the literature, and this possibility should be considered only after the woman reaches the active phase of labor.[29]

One last issue with oxytocin is the conflicting goals and miscommunication that sometimes occur between the nurses at the bedside managing the infusion and the ordering providers.[46] Often this conflict revolves around the dosing intervals of the oxytocin infusion. One possible solution is to implement mutually agreed-upon checklist-based protocols for oxytocin administration.[46,47,52] These checklists focus on the fetal and uterine response to oxytocin instead of specifying a specific dosing regimen, allowing time for normal labor progress rather than more oxytocin. Also included are other safety measures such as appropriate staffing ratios and standard order sets for treating tachysystole. Prompt initiation of interventions without additional orders from the provider is an essential component of best practice recommendations for oxytocin use.[52]

Other Methods of Induction

The use of more natural techniques to stimulate labor or facilitate cervical ripening is of interest for many women. In many cases, these methods have been endorsed in an attempt to help women avoid an induction of labor with oxytocin.

Nipple Stimulation

Researchers have evaluated the safety and effectiveness of nipple or breast stimulation as a

method to stimulate or induce labor.[55] Stimulation of the nipples is known to induce the release of endogenous oxytocin from the pituitary gland, which in turn causes uterine contractions similarly to administration of synthetic oxytocin. In fact, nipple stimulation protocols have been used as an alternative to oxytocin administration for performing a contraction stress test. Various techniques have been employed, such as rolling one or both nipples between the fingers or using a manual or electric breast pump.

A systematic review of the available studies on nipple stimulation reported that low-risk women with a favorable cervix who used this technique were likely to be in labor within 72 hours.[55] Other significant findings were a decreased incidence of postpartum hemorrhage and no episodes of uterine tachysystole or abnormal FHR changes. One remaining concern is the effect of nipple stimulation and endogenous oxytocin on placental perfusion. Because the existing evidence has not specifically examined nipple stimulation for women who are at risk for fetal acidemia, its use is discouraged with this population.[55]

Herbal Preparations

Several herbal preparations, such as castor oil, evening primrose oil, raspberry leaf, and blue or black cohosh, have been used for the purposes of stimulating labor. Midwives who recommend herbal preparations believe these methods are safe, are inexpensive, and help women avoid medical induction and prolonged pregnancy.[56] They can also be self-administered by the woman at home or used in a birth center where oxytocin and prostaglandin use is contraindicated. Information regarding herbal methods of labor induction is often shared informally among colleagues and likely based on anecdotal experiences, folklore, and traditional practices. There is wide variation among midwives in how and when these preparations are prescribed.

A review of the literature regarding herbal methods of labor induction reported an overall lack of scientific evidence regarding these treatments' efficacy or clinical benefit to women.[57] In case reports, blue cohosh has been associated with perinatal stroke and acute myocardial infarction; it should not be used.[58] Currently, there is no scientific literature that documents the safety or efficacy of black cohosh. Until scientific studies can be conducted to refute these concerns, blue and black cohosh, either together or in combination with other substances, should not be used in clinical practice. Evening primrose oil and raspberry leaf, in contrast, have demonstrated no evidence of harm over many years of use. Whether they actually cause contractions or promote cervical ripening is not conclusively seen in the evidence, but their use during pregnancy is considered to be safe.[58]

The use of castor oil can be traced back to Egyptian times, and this product is still commonly used today to induce or stimulate labor. Although the exact mechanism of action is not completely understood, ingestion of castor oil produces a powerful cathartic effect that in turn induces uterine contractions. Some women have reported rapid tumultuous labors after ingesting castor oil, and use of this preparation may be associated with an increased risk of meconium-stained amniotic fluid. The evidence from several studies remains inconclusive regarding the effectiveness of castor oil. Although castor oil ingestion is safe for women during pregnancy, midwives who elect to use it for labor induction should be aware of the

inconsistent evidence as well as the lack of standard dosing protocols.

In summary, caution should be exercised when prescribing or recommending alternative methods of cervical ripening or induction of labor not supported in the scientific literature. The midwife should also inform the woman of the known risks, benefits, and safety concerns prior to prescribing or endorsing the use of any herbal preparations.

Labor Abnormalities

The classic abnormalities of labor progress are prolonged latent phase, slow-slope active phase, active-phase arrest, arrest of descent, prolonged second stage, and arrest of descent in the second stage. Second-stage abnormalities are presented in detail in the *Second Stage of Labor and Birth* chapter. Evaluation of labor progress is an ongoing intrapartum assessment, although the relationship between labor duration and maternal and neonatal outcomes is not well understood.[59] Obstructed labor resulting from true cephalopelvic disproportion is a real phenomenon, and neglected obstructed labor is a significant contributor to maternal and neonatal morbidity and mortality in low-resource settings.[60] Primary contributors to this problem are undernourishment in childhood resulting in a small pelvis and lack of access to needed medical care during abnormal labors.

The situation in developed countries is quite different. It is not clear that commonly used frameworks for diagnosing and treating prolonged labor in the United States improve outcomes.[59] Nonprogressive labor is the most common reason for primary cesarean births and is indirectly responsible for many repeat cesareans, and most agree that this condition is overdiagnosed.[61,62]

Many terms are used to denote abnormally slow labor—for example, labor dystocia, dysfunction labor, and failure to progress. There is no consensus on the diagnostic criteria for these terms.[62] New research and expert opinions on the parameters of normal labor progress are reviewed in the *First Stage of Labor* chapter, and questions about the appropriate overall framework for care related to labor progress make this an evolving and complex topic.[63-71]

For many years, assessment of labor progress was based on the work of Friedman, who published original labor curve data in the 1950s.[63] In the Friedman framework, parameters for abnormal labor progress are as follows:[64]

- Prolonged latent phase:
  - Nulliparas: greater than 20 hours
  - Multiparas: greater than 14 hours
- Protracted active phase:
  - Nulliparas: less than 1.2 cm/hr dilation
  - Multiparas: less than 1.5 cm/hr dilation
- Active-phase arrest: no progress in 2 hours

More recent research based on two large data sets, the Collaborative Perinatal Project (CPP) from 1959–1966[65] and the Consortium on Safe Labor (CSL) from 2002–2008,[66] have challenged Friedman's criteria, suggesting that labor progress for contemporary women is actually much slower for both nulliparous and multiparous women. Another key finding is that many women are not in the active phase of labor until their cervices are 6 cm dilated. Currently proposed evidence-based definitions for abnormal labor progress based on the CSL database are as follows:[61,66]

- Nulliparas: less than 0.5 to 0.7 cm/hr dilation; multiparas: 0.5–1.3 cm/hr dilation, with faster dilation as labor progresses
- No application of active labor diagnosis until 6 cm dilation

The differences between the Friedman curve and newer analyses by Zhang et al.[65,66] can be attributed to several factors. Some have proposed that women today are older, heavier, and subject to different intrapartum interventions compared to the women included in Friedman's population studies. Furthermore, Friedman's work was based on direct observation of women in labor, whereas Zhang et al. applied a different statistical analysis to previously collected data. Also debated is optimal timing and type of interventions, including cesarean birth. In the past, recommendations were based on Friedman criterion for protracted and arrested labor. Oxytocin augmentation and amniotomy were often used at this point, with the consensus being that a cesarean was indicated if no additional progress occurred in 2 hours with adequate labor.[67]

There are no currently agreed-upon protocols indicating when interventions should begin. The authors of the consensus document published by ACOG and the Society for Maternal-Fetal Medicine state, "When the first stage of labor is protracted or arrested, oxytocin is commonly recommended."[61] This document also asserts that slow but progressive labor is not an indication for a cesarean section.[61] In addition, it recommends that before a cesarean section for nonprogressive labor is undertaken, the woman should be at 6 cm of cervical dilatation or more, have ruptured membranes, and have no cervical change over 4 hours with adequate uterine activity *or* no cervical change with at least 6 hours of oxytocin with inadequate uterine activity.[61] Neal et al. offer more specific guidance, suggesting that "at more than 5 cm dilatation, oxytocin augmentation may be considered any time there is a more than a 4 h delay in cervical change."[59]

Midwifery Management of Abnormally Slow Labor

Prolonged Latent Phase

However, accurate measurement of the latent phase of labor is problematic, and the relationship between duration of the latent phase and outcomes is not clear.[68]

Management of prolonged latent labor can be particularly challenging. Delaying admission to the birth setting until the active phase of labor is optimal, because earlier admission is associated with an increased risk of a variety of interventions, such as oxytocin augmentation, epidural analgesia, and cesarean birth.[69]

The use of coping techniques at home will be sufficient for some women with long latent phases.[68,69] Among these strategies are the presence of a supportive companion, a warm bath or shower, fluid and food, comfort measures (e.g., massage), ambulation and rhythmic movement, music, and aromatherapy.[70] The woman should be encouraged to stay hydrated, nourished, and rested.

Medications that have a sedative effect are sometimes used to help women sleep.[70] There is a paucity of research on the effectiveness or safety of these agents for treating women during

labor. Some such as diphenhydramine (Benadryl) have a long history of use during pregnancy and appear to be quite safe. Others such as secobarbital (Seconal) have very long half-lives that could adversely affect the newborn so they are no longer in common use. Short-acting non-benzodiazepine sedatives such as zolpidem (Ambien) may be used for this purpose in some locales but no published studies that have assessed either safety or effectiveness have been published.

Qualitative evidence suggests that many women find coping with latent labor at home difficult and stressful, and desire admission earlier than recommended.[68] This is a quandary for midwives, especially when the latent phase is lengthy. Regular phone contact with the midwife, outpatient check-ins, reassurance of normalcy, realistic anticipatory guidance, and emotional support may help many women manage this part of labor at home.

If the woman becomes dehydrated or too sleep deprived secondary to a prolonged latent phase, she may need admission to the hospital. The two treatment options in such a case are (1) therapeutic rest to stop contractions and allow a period of rest or (2) uterotonic drugs to encourage organized effective contractions. It is best to base management decisions on maternal and/or fetal status and maternal preferences, rather than duration of the latent phase.

Therapeutic rest typically involves a single dose of morphine sulfate administered intramuscularly (or intravenously), which is sometimes combined with promethazine (Phenergan) or hydroxyzine (Vistaril).[70] A variety of dosing strategies are used, all of which have a primary goal of inducing heavy sedation or sleep. Commonly encountered maternal side effects include respiratory depression, euphoria, sedation, dizziness, and nausea and vomiting. Neonatal respiratory depression is also a potential concern, especially if labor progresses rapidly if morphine is administered close to the time of birth. In many cases, women managed with therapeutic rest will sleep for several hours and awaken in active labor.

Slow Labor in the Active Phase

A delay in cervical dilation may be a normal variation of a particular woman's labor. Analysis of the CSL database found significant individual variability in the time interval in dilating from one centimeter to the next during the active phase of labor.[66] In addition, no progress for 2 hours, especially before 7 cm dilation, is a common occurrence.[71] Management of slow labor should begin with thorough data collection. Maternal and fetal well-being must be confirmed first, then all factors that might contribute to slow labor progress must be assessed, including physical, psychological, and environmental factors (**Table 29-8**).

Table 29-8	Assessment for Slow Labor Progress

1. Observe the woman for signs of exhaustion, decreased coping, or dehydration.
2. Explore the possibility of an underlying maternal fear or anxiety related to labor or the pending birth to determine ways to improve the woman's coping ability.
3. Assess adequacy of labor support and the woman's environment for factors that may impede labor progress.
4. Check maternal vital signs for fever or tachycardia.
5. Palpate the contractions for frequency and intensity, and note any trends or changes.

6. Reassess estimated fetal weight and fetal lie, presentation, position and attitude with Leopold's maneuvers.
7. Assess fetal well-being with intermittent auscultation or continuous electronic fetal monitoring.
8. Review the labor progress up until this point.
9. If the membranes are not ruptured, perform a cervical examination and carefully assess dilation, effacement, station, and position of the fetal head. Note any asynclitism, molding, or caput.
10. During the cervical examination, perform clinical pelvimetry; note any obvious narrowing of the pelvic diameters.
11. If the membranes are ruptured, perform this examination only if clinically indicated.

Abbreviations: TOLAC, trial of labor after cesarean; VBAC, vaginal birth after cesarean.

Continued expectant management is reasonable if there are no clinical indications to expedite birth. Strategies to stimulate or augment labor should be presented to the woman, and the risks and benefits of each option reviewed. Depending on the woman's preferences, the least invasive strategies should be attempted first. For example, if the woman is rested and coping well, ambulation or position changes should be encouraged.

Theoretically labor progress may slow or stall secondary to acute stress, and some authors have postulated that fear or anxiety can interfere with labor progress. The catecholamines that are released during the stress response—that is, norepinephrine and epinephrine—normally have an inhibitory or tocolytic effect on uterine contractility. If labor, fatigue, and pain cause an acute stress response, a diminution of uterine contractility may occur. A few small studies have correlated plasma epinephrine levels and uterine contractility, with the results supporting this hypothesis.[72] Nevertheless, it is not known if beta-adrenergic receptors for endogenous catecholamines are significantly upregulated in the uterus during term labor, and the research that supports this theory is not robust. Thus, the relationship between significant fear or anxiety and slow labor progress remains somewhat theoretical. Strategies that promote a sense of emotional well-being and safety are certainly good options if labor progress is slow. Continuous labor support—especially the therapeutic presence of the midwife, decreasing unnecessary stimulation from caregivers, low lighting, and privacy—may help decrease the physiologic stress response.

Relief of pain is another important component of managing slow labor. Unrelieved pain is also associated with increased levels of circulating catecholamines. Hydrotherapy can be very effective in decreasing pain and increasing comfort. Inability to cope may indicate the need for medication and, for some women, epidural anesthesia. While this strategy may increase the likelihood of other interventions being used to stimulate labor, ensuring adequate relief of pain is always a priority.

Malpositions such as occiput posterior position, and other normal variations in fetopelvic relationships such as asynclitism are common causes of slow labor. Watchful waiting and position changes that increase pelvic diameters are suggested approaches. Most fetuses in an occiput transverse or occiput posterior position will rotate to occiput anterior prior to birth. Persistent fetal malpositions are addressed in the *Second Stage of Labor and Birth* chapter.

Amniotomy can also stimulate or augment labor when slowed progress is encountered. This procedure is not associated with a shortening of the duration of labor in women who start labor spontaneously and progress normally.[73] In contrast, in women whose labor is induced and

those who are experiencing slow progress, amniotomy with oxytocin augmentation is associated with a shorter duration of labor.[74]

A final strategy for managing slow labor is oxytocin augmentation. The same cautions and clinical considerations exist regardless of whether oxytocin is used for augmentation or induction of labor. Insertion of an intrauterine pressure catheter is not mandatory unless external monitoring is unable to detect contraction frequency. The consulting physician should be informed of the ongoing plan of care during labor augmentation with oxytocin and the woman's labor progress and fetal response monitored closely.

Often there are many possible appropriate strategies for slow labor. Shared decision making should be used, as the woman's preferences are a key aspect of clinical decision making in this situation.

Active-Phase Arrest

Active-phase arrest is diagnosed when a woman is at a cervical dilation of 6 cm or more with ruptured membranes and no cervical change for 4 hours or more in the presence of adequate uterine contractions (> 200 Montevideo units) or no cervical change in 6 hours or more in the presence of inadequate contractions.[61] When active-phase arrest is diagnosed, physician referral is required, as the treatment is cesarean birth. Possible etiologies of active phase arrest are cephalopelvic disproportion or deep transverse arrest wherein the fetus in a vertex presentation cannot rotate from transverse to occiput anterior or occiput posterior. Cephalopelvic disproportion can be secondary to maternal pelvic architecture or fetal size or malposition.

Trial of Labor After Cesarean

For the past several decades, women presenting with a history of one or more previous cesareans have faced a series of changing opinions regarding the best mode of birth during subsequent pregnancies. Prior to the early 1980s, the predominant philosophy was "once a cesarean, always a cesarean." Early reports of successful vaginal birth after cesarean (VBAC), however, began to challenge this long-standing practice. In 1980, the National Institutes of Health (NIH) published a landmark statement endorsing a trial of labor for women who had one previous lower transverse cesarean birth.[75] Over the next 10 years, VBAC continued to grow in popularity, with several large studies reporting a 60% to 80% success rate for women who underwent a trial of labor after cesarean (TOLAC).[76] These studies also demonstrated that planned repeat cesarean delivery (PRCD) was associated with more maternal morbidity than a TOLAC and subsequent VBAC.

The NIH had hoped promoting a trial of labor would reduce rising cesarean birth rates, along with their associated costs in terms of morbidity and economic expense. This strategy appeared to be successful, and by 1996, the rate of cesarean births in the United States reached an all-time contemporary low of 20.7%.[76] As more women underwent TOLAC, reports of uterine rupture during a trial of labor emerged, leading to increased safety concerns. In 1996, a large clinical trial was published that examined a composite measure of serious maternal morbidity including uterine rupture, hysterectomy, and operative injury. The absolute risk of this measure was found to be 1.6% in women who underwent TOLAC and 0.8% in women who had a PRCD.[77] The risk of these complications was found to be much higher in the 40% of women in the TOLAC group who had an unplanned repeat cesarean birth.

This study, which was widely publicized, reported the relative risk of a doubling of serious maternal complications with TOLAC versus PRCD. Almost immediately, enthusiasm for TOLAC began to wane and VBAC rates steadily fell. Concern about the risk of uterine rupture and its associated liability resulted in ACOG guidelines that recommended the immediate in-house availability of an obstetrician and an anesthesiologist to perform an emergent cesarean birth if needed.[78] Many hospitals and providers subsequently stopped offering TOLAC services, radically altering women's childbirth choices. By 2007, cesarean birth rates had risen to 32.8%; in turn, VBAC rates plummeted to 8.9%.[79] While many factors have been cited for the cesarean epidemic, decreased access to available TOLAC services, particularly in rural areas that do not have immediate in-house anesthesia availability, is a frequently cited cause of this unfortunate trend.

Concerns about the rising primary cesarean birth rates and the associated morbidities, particularly the increase in the incidence of placenta previa and placenta accreta in subsequent pregnancies led the NIH to revisit the comparative safety of having a VBAC versus PRCD. In 2010, the NIH Consensus Development Conference Statement on this topic was published; it concluded that women with a previous low transverse cesarean birth can be safely offered a trial of labor when no other contraindications to vaginal birth exist.[79] The NIH statement also encouraged obstetric providers to improve access to TOLAC services and support women's

preferences when possible. ACNM published a position statement and an updated clinical practice bulletin that supports the NIH's consensus report.[80] In 2017, ACOG published revised guidelines that recommend women who are candidates for TOLAC can be safely offered TOLAC in Level 1 hospital settings.[81] These settings do not require immediate in-house anesthesia but do have the ability to initiate an emergency cesarean in life-threatening situations.[81]

Risks of TOLAC and PRCD

The comparative risks of TOLAC versus PRCD are presented in the *Pregnancy-Related Conditions* chapter, reflecting the reality that discussion of these issues begins during prenatal care. The most serious and catastrophic risk for a woman who elects TOLAC is uterine rupture. Generally speaking, the term *uterine rupture* describes an anatomic separation of the uterine muscle.[76,79,81] This outcome can be further differentiated between uterine scar dehiscence and full separation of all uterine layers. Occult scar dehiscence does not include the serosa layer and is an incidental, asymptomatic finding with no adverse maternal or fetal consequences. A complete uterine rupture, in contrast, is a symptomatic, life-threatening emergency. Currently, the risk of uterine rupture is cited as 0.4% to 0.7% for women attempting TOLAC, compared to 0.03% in women who opt for PRCD.[79] Uterine rupture is associated with a 14% to 33% rate of hysterectomy and a 6% intrapartum fetal death rate (or 2.9 fetal deaths per 10,000 women who have a TOLAC).[79]

PRCD has risks for both the women during the current pregnancy and any subsequent pregnancy. There is an increased risk of abnormal placentation (placenta previa, accreta, and abruption) with a history of cesarean birth, and this risk increases with each cesarean birth. For example, after one cesarean birth, the risk of placenta previa is 9 per 1000, but this risk increases to 17 per 1000 after two cesareans.[82]

A history of previous classical or "T" uterine incision, previous uterine rupture, or any type of fundal uterine surgery (i.e., myomectomy), significantly increases the risk for uterine rupture.[81] Women presenting for prenatal care with any of these risk factors are not candidates for TOLAC; instead, it is recommended that they undergo a PRCD. The optimal gestation age for PRCD is 39 weeks.[82] In addition, these women require prompt evaluation in the hospital with continuous fetal monitoring and physician consultation should regular contractions occur before the scheduled date of surgery.

Factors such as a history of two or more previous (low transverse) cesarean births, or previous preterm cesarean birth, increase the risks of uterine rupture to varying degrees. A review of the existing evidence found weak associations for these factors and concluded that evidence for most of the factors is limited.[81] Consequently, women with these obstetric histories can now safely be offered a TOLAC if desired and after a discussion about her individualized risks. These women should receive prenatal counseling from a physician given they have conditions that may increase the risk of uterine rupture to some extent.

Induction or Augmentation of Labor During TOLAC

Abnormal labor progress is associated with a small increased risk of uterine rupture.[75,81] Similarly some studies have shown a slightly increased risk of uterine rupture when oxytocin is used for augmentation of labor in women who have a TOLAC.[75] The increased risk of uterine rupture associated with oxytocin augmentation is minimal, however, and does not rule out its use when medically indicated.[75,81] Because the upper limits for oxytocin dosing with TOLAC have not been established, a conservative approach is preferred, accompanied by close monitoring of fetal well-being, uterine activity, and labor progress.

Similarly, the risk of uterine rupture is higher in women whose labor is induced with oxytocin, but the absolute risks are still low.[81] Notably, induced labor is less likely to result in a successful VBAC, especially if the woman has an unfavorable cervix.[75] Therefore induction of labor should be considered only when medically indicated and only in hospital settings where physician consultation for a cesarean birth is available.

The use of cervical ripening agents for women who desire TOLAC is more controversial. Prostaglandins E_1 (misoprostol/Cytotec) and E_2 (Cervidil and Prepidil) are commonly used to facilitate cervical remodeling and have been shown to improve labor induction success rates. Interpretation of the evidence is made difficult by inconsistent study results and design limitations such as grouping of different types of prostaglandins together, use of birth certificate data (which are known to have poor accuracy), and underpowered for detection of relevant outcomes.[81] Even so, based on several small studies showing an increased rate of uterine rupture following use of misoprostol for cervical ripening in women who had a prior cesarean, this medication is now considered contraindicated.[81] Due to limited evidence regarding the effects of prostaglandin E_2 in women who have a uterine scar, use of this medication is discouraged.[81] Mechanical methods (i.e., cervical Foley balloon) have not been associated with increased risks of uterine rupture and can be used for cervical ripening.[81]

Intrapartum Management for Women During TOLAC

Table 29-9 outlines intrapartum management guidelines for women attempting TOLAC. When a woman with a history of previous cesarean birth presents for a labor evaluation, the prenatal record should be reviewed, along with any previously discussed labor management options. The woman's continued desire for TOLAC should be reaffirmed and the informed consent documentation reviewed. The intrapartum database should also include a thorough review of her obstetric history, paying particular attention to the indications for and circumstances surrounding the previous cesarean birth. Equally important is confirming the type of uterine incision (if known) and ascertaining whether the woman has undergone any other uterine surgeries since her last birth. Lastly, a consulting physician should be notified if there is any contraindication to VBAC or if the woman is opting for PRCD.

Table 29-9 Intrapartum Management Guidelines for Women Attempting Trial of Labor After Cesarean

1. A consulting physician should be notified when a woman desiring TOLAC is admitted in labor.
2. Intravenous access (i.e., heparin or saline lock) may be required according to institutional policies.

3. Clear liquid intake is not contraindicated; however, some institutional policies may restrict this intake to ice chips or sips of water.
4. More frequent fetal heart rate monitoring is required—either continuous electronic monitoring or intermittent monitoring per high-risk guidelines (i.e., every 15 minutes in active labor and every 5 minutes in second-stage labor). A telemetry monitor can be used to encourage freedom of movement.
5. The woman should be monitored closely for indications of uterine rupture. The most common initial sign is either recurrent fetal heart rate decelerations that become progressively deeper or an abrupt fetal heart rate bradycardia. Other signs include changes or cessation of uterine contractions, vaginal bleeding, or a sudden loss of fetal station.
6. Epidural anesthesia is not contraindicated and will not mask the most common signs and symptoms of uterine rupture.
7. Labor progress should be closely monitored for signs of abnormally slow progress. Oxytocin should be used with caution, and only after other nonpharmacologic methods to promote labor progress have been attempted. Physician consultation should be obtained prior to initiating labor augmentation with oxytocin.
8. High-dose oxytocin protocols are contraindicated.
9. Intrauterine pressure catheters have not been shown to accurately depict an impending uterine rupture and should not be used solely for that purpose.
10. Hydrotherapy (i.e., tub or shower) is not contraindicated as long as waterproof fetal monitoring is available, although it is frequently restricted according to individual policies.
11. There are no contraindications to alternative positions for birth as long as fetal assessment guidelines are followed.
12. Management of third-stage labor is the same following VBAC as it is following a normal vaginal birth. The midwife and woman can opt for either expectant or active management of the third stage. There is no need to explore the uterine scar for signs of asymptomatic dehiscence after the delivery of the placenta.
13. Women with a previous uterine scar have an increased risk of abnormal placentation (e.g., placenta accreta). A physician should be consulted for a retained placenta prior to any attempts for manual removal.

Midwifery management for women in labor who desire TOLAC is similar to the care of any laboring woman with a few exceptions, including the recommendation for continuous FHR and uterine contraction monitoring and possibly a requirement for intravenous access. In addition, women who have experienced a previous unplanned cesarean birth often present with intense feelings regarding their past birth. They may have psychological milestones to overcome at the point in labor in which the events leading to the cesarean birth occurred. The midwifery model of care supports practices that promote normal labor and birth and are beneficial for women who desire TOLAC. All interventions, therefore, should be evaluated for safety and interference with normal labor progress and mechanisms.

Intrapartum Management for Women with Coexisting Medical Conditions

Two common medical conditions that can have adverse effects for a pregnant woman and her fetus are diabetes and hypertension. When caring for a woman with diabetes or hypertension, a collaborative plan of care between the midwife and a consulting obstetrician should be in place. As these women enter labor, the balance of management authority will greatly depend on individual policies, the evolving clinical situation, and the experience of the midwife. It is important for midwives to have knowledge of what to expect for these women and to recognize that the intrapartum clinical course could change suddenly, necessitating immediate referral of care to the obstetrician. Ongoing and clear communication between the physician and the midwife is paramount throughout labor to ensure the best outcome for the mother and her infant. When midwives collaborate in the care of women with coexisting medical complications, the midwife often monitors and manages labor progress, while the physician monitors and manages interventions required to minimize adverse effects of the medical condition. For example, if the woman has diabetes, the midwife may care for her during labor and birth, while the physician oversees administration of insulin and other tests to monitor diabetes.

Many other medical conditions can adversely affect pregnancy, including epilepsy, autoimmune disorders, and heart conditions. Every possible condition cannot be addressed here, but management is always contingent on knowledge of the physiology of the disorder, diagnosis, and the effects on pregnancy. The midwife also needs to know when to obtain a consultation and/or transfer care of the woman. In this section, diabetes and hypertension are used as examples of the midwifery management process when caring for a woman who has a coexisting medical condition.

Women with Diabetes

Approximately 6% to 7% of pregnancies in the United States occur in women with diabetes, with 90% of the affected women having gestational diabetes.[83] For women with well-controlled, pregestational diabetes, induction of labor is recommended between 39 and 40 weeks' gestation.[83] The optimal timing for birth for women with well-controlled gestational diabetes is not clear.[83] A plan for fetal surveillance and timing of birth should be developed in collaboration with the woman and consulting obstetrician.

Women with either pregestational or gestational diabetes have an increased risk of shoulder dystocia during delivery. This trend reflects the increased risk of macrosomia and a tendency for the infants of diabetic mothers to have larger shoulder diameters at a given weight.[84] Prediction of macrosomia via ultrasound or clinical palpation is quite inexact. Induction of labor is not recommended for suspected macrosomia. Nevertheless, the option of cesarean birth is offered to women with diabetes who have a fetus with an estimated fetal weight of 4500 grams or more.[84] This practice is based on evidence showing a higher risk of brachial plexus injury in the infants born to women in this cohort. **Appendix 29A** reviews the management of shoulder dystocia. Fetal lung maturity may be delayed in the infants of women

with diabetes, and these seemingly full-term infants can exhibit respiratory distress and other related complications immediately following birth.

During labor, monitoring the woman's blood glucose and maintaining glycemic control have been shown to reduce the incidence of neonatal hypoglycemia.[83] For women who are euglycemic with dietary changes only, it may not be necessary to monitor glucose levels during labor. Women who are using insulin or oral glycemic agents, however, will require finger-stick glucometer testing of their blood glucose approximately every 2 hours. Any evidence of intrapartum hyperglycemia should be treated promptly with intravenous insulin, balanced with dextrose 5% in lactated Ringer's solution. The goal is to maintain the woman's blood glucose between 80 and 110 mg/dL.[83] Institutional variations on these guidelines may exist, and the midwife should develop the most appropriate plan of care with a consulting physician. When a woman in labor has an insulin infusion, she is best cared for collaboratively by a physician who manages the diabetes and a midwife who manages her labor progress.

Careful assessment of fetal size by Leopold's maneuvers is performed when the woman is admitted, and labor progress is monitored closely. Continuous fetal monitoring is required if an insulin infusion is administered. The midwife should be prepared for the possibility of shoulder dystocia at the time of birth and have a low threshold for requesting the presence of the consulting obstetrician during the second stage of labor. The pediatric team should also be notified, especially if the woman has poor glycemic control or fetal lung immaturity is a possibility.

Women with a Hypertensive Disorder

The diagnostic criteria for hypertensive disorders in pregnancy are described in the *Medical Complications in Pregnancy* chapter. In all cases, these diagnoses may become medical indications for delivery, but the timing will vary according to the severity of the woman's condition and the gestational age of her fetus. Although some fetal risks are associated with these disorders, maternal mortality and morbidity are the primary concern; treatment focuses on preventing end-organ damage in the woman. It is most important for clinicians to recognize that hypertension and preeclampsia exist on a continuum that is often unpredictable. For this reason, women admitted with either of these diagnoses require vigilant monitoring during labor for subtle changes or a sudden acceleration of worsening symptoms. The midwife works closely with the collaborating physician to develop the most appropriate plan of care, especially if the woman is diagnosed with preeclampsia.

Intrapartum considerations include an ongoing assessment of the woman for the presence of headache, visual changes, or epigastric pain. Any headache should be taken seriously and reported to the consulting physician, especially if unrelieved with oral acetaminophen. Visual changes such as scotomata (loss of visual field) or scintillating spots and epigastric or right upper quadrant pain are also worrisome symptoms that require prompt evaluation and consultation with the physician. Careful monitoring of fluid balance is essential as well. Although women with preeclampsia may have intravascular volume depletion, they are at risk for pulmonary edema secondary to the need for intravenous fluids in the presence of impaired kidney function. **Table 29-10** outlines specific assessments that should be included in the plan

of care during labor, and **Table 29-11** describes how to determine the presence or absence of clonus.

Table 29-10 Labor Assessment for Women with Preeclampsia

1. Hourly blood pressure monitoring[a]:
   a. Any result higher than or equal to 160 mm Hg systolic or 110 mm Hg diastolic requires immediate physician consultation and administration of an intravenous antihypertensive medication.[85]
2. Lung auscultation and/or pulse oximetry:
   a. Any shortness of breath, abnormal breath sounds (crackles, wheezes), or other signs of pulmonary edema require the immediate presence of a physician.
   b. Obtain pulse oximetry if the woman has any shortness of breath.
3. Deep tendon reflexes:
   a. Hyperreflexia or the presence of clonus requires physician consultation. (See **Table 29-11** for information on assessment of clonus.)
4. Serum creatinine, AST, complete blood count (hemoglobin, hematocrit, and platelets):
   a. Obtain on admission and every 8 hours per practice/institutional guidelines. Report abnormalities to a consulting physician.
5. Strict intake/output:
   a. Measure urine output at least every 2 to 4 hours. If the urine output is averaging 30 mL/hour or less, notify the physician.
   b. Carefully monitor intravenous and oral fluids.
6. If magnesium sulfate is infusing:
   a. Perform hourly assessments of respiratory rate, deep tendon reflexes, and urine output. The infusion should be discontinued and a physician notified immediately for any signs of magnesium toxicity (respiratory rate less than 12 breaths/minute, absent or diminished deep tendon reflexes, or urine output less than 30 mL/hour).
   b. The antidote for magnesium toxicity is calcium gluconate 10 mL of a 10% solution administered intravenously over 3 minutes.

Abbreviation: AST, aspartate aminotransferase.

[a] Manual blood pressure measurements are preferred, as they are the most accurate.

Table 29-11 Assessing for Clonus

What Is Clonus?
Clonus is involuntary, rapid, repetitive, rhythmical contractions and relaxations of a muscle when it is sharply stretched and the stretch is maintained in either flexion or extension. Clonus can be elicited at a number of sites; in obstetrics, most commonly the ankle is used.
What Causes Clonus?
The presence of clonus is associated with a hyperreflexive state and could indicate abnormal central nervous system (CNS) excitability. It is always associated with abnormal (+4) deep tendon reflexes and may be a precursor to an eclamptic seizure.
Assessment for Clonus

1. Position the woman so her knee is partially flexed. Support this position with one hand underneath the bend in her knee.
2. With the other hand grasping her foot, sharply dorsiflex the foot and maintain pressure to keep it in dorsiflexion.
3. Beats of clonus will be seen and felt as the muscle contractions and relaxations cause rhythmical alternation between dorsiflexion and plantar flexion. The muscles being stretched are the same as for the ankle jerk reflex —the gastrocnemius and soleus muscles.

Severe hypertension and eclampsia are two of the most serious consequences of preeclampsia. The National Partnership for Maternal Safety has a consensus bundle (set of evidence-based guidelines) of recommendations for recognizing early warning signs (triggers) of these conditions and treating severe hypertension (**Table 29-12**).[85,86] Other evidence-based protocols are listed in the Resources section at the end of this chapter.

Table 29-12 Treatment of Severe Hypertension

Drug: Generic (Brand)	Dose	Contraindications[a]
Hydralazine (Apresoline)	5–10 mg IV every 20 minutes Maximum total dose: 20 mg in first hour	Tachycardia
Labetalol (Normodyne, Trandate)	20 mg IV bolus Followed by 40 mg if not effective in 10 minutes Followed by 80 mg if not effective in 10 minutes Maximum total dose: 300 mg in first hour	Asthma, congestive heart failure
Nifedipine	10–20 mg orally every 30 minutes Maximum total dose 50 mg in first hour	Tachycardia

Abbreviation: IV, intravenous.

Note: If blood pressure is ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic, verify within 15 minutes. If confirmed by a second reading, administer a hypertensive within 30–60 minutes.

[a] Contraindications may be relative contraindications depending on the clinical condition.

Based on American College of Obstetricians and Gynecologists. *Hypertension in Pregnancy.* Washington, DC: American College of Obstetricians and Gynecologists; 2013[85]; Bernstein PS, Martin JN Jr, Barton JR, et al. National Partnership for Maternal Safety: consensus bundle on severe hypertension during pregnancy and the postpartum period. *Obstet Gynecol.* 2017;130(2):347-357.[86]

Magnesium sulfate remains the standard of care for the prevention and treatment of eclampsia for women who have preeclampsia with severe features.[85,86] Magnesium sulfate is administered as a bolus of 4–6 gm in 100 mL over 20 minutes, followed by an intravenous infusion of 2 gm per hour throughout labor and for 24 hours post birth. When preeclampsia occurs without severe features, magnesium sulfate therapy is not recommended.[85,86] However, progression of preeclampsia can occur suddenly, and the woman must be monitored very closely for any such changes.

Deep tendon reflexes are part of both the initial evaluation and ongoing assessments. Reflexes are evaluated on a scale of 0 to 4+:

0: absent; no response
1+: decreased; diminished; sluggish
2+: normal; average
3+: brisk
4+: very brisk; hyperactive (usually associated with clonus)

Reflexes designated as 1+ are low-normal reflexes; 3+ reflexes are more brisk than the average reflex response and indicate the possible but not absolute presence of disease; 0 and 4+ reflexes are definitely abnormal and indicate disease requiring physician consultation. Normally, deep tendon reflexes are evaluated as 1+ or 2+. Hyperreflexia (i.e., 3+ or more) is one indication that a seizure is imminent; thus, hyperreflexia is an important piece of information to obtain when a woman presents with hypertension. Conversely, a sign of magnesium toxicity is absent or depressed deep tendon reflexes. Therefore, when magnesium sulfate is infusing, deep tendon reflexes are assessed hourly.

Obstetric Emergencies

Midwives in clinical practice will inevitably encounter an obstetric emergency. Fortunately, most obstetric emergencies are rare occurrences, and in some cases, they can be anticipated. Regardless, midwives are responsible for the care of women during the intrapartum period, which requires a thorough understanding of how to manage emergent events during labor and birth. Prompt recognition and immediate action are imperative until the situation is resolved or the consulting physician can assume management. The following sections review the clinical management for several intrapartum emergencies, including intrapartum hemorrhage, shoulder dystocia, prolapsed umbilical cord, twin gestation, and breech birth.

Recent studies of maternal morbidity and mortality suggest that 40% to 50% of maternal deaths are preventable.[87] Delays in recognition and treatment have prompted the development of early warning signs and rapid response teams to help practitioners more effectively anticipate and respond to obstetric emergencies. Maternal early warning triggers that require immediate attention are listed in **Table 29-13**. All maternity care providers should know these signs and the plans for intervention. Resources for learning more about maternal early warning systems are listed at the end of this chapter.

Table 29-13 Maternal Early Warning Criteria

Sign or Symptom	Definition
Blood pressure	Systolic: < 90 or > 160 mm Hg
	Diastolic: > 100 mm Hg
Heart rate	< 50 or > 120 beats/minute
Respiratory rate	< 10 or > 30 breaths/minute
Maternal symptoms	Agitation, confusion, unresponsiveness
	If preeclampsia: nonremitting headache or shortness of breath
Oxygen saturation at room air at sea level	< 95%
Oliguria	< 35 mL/hr for > 2 hours

Modified with permission from Mhyre JM, D'Oria R, Hameed AB, et al. The maternal early warning criteria: a proposal from the National Partnership for Maternal Safety. *Obstet Gynecol.* 2014;124(4):782-786.[87]

Intrapartum Hemorrhage

The differential diagnoses for vaginal bleeding in the second or third trimester include placenta previa, placental abruption, uterine rupture, and vasa previa. The midwife's role in the care of a woman who is bleeding is to initiate stabilization measures until the collaborating physician can take over the primary management. Anesthesia specialists, the operating room team, and the neonatal/pediatric team should also be mobilized and on standby for an emergent cesarean birth and possible neonatal resuscitation. If the woman is hemodynamically unstable or the fetus is in jeopardy, birth will be expedited, regardless of gestational age.

Initial evaluation of hemodynamic status includes assessment of vital signs, mental status,

and skin color (pallor), as well as a visual evaluation of blood loss. The consulting physician is called for an immediate bedside consultation. If there is evidence of significant blood loss on the woman's clothes or body with any signs of hemodynamic compromise, two large-bore intravenous lines should be started. The woman should be given oxygen and placed on continuous electronic fetal monitoring. Laboratory evaluation includes a complete blood count, coagulation studies, blood type, antibody screen, and cross-match for a minimum of 4 units of blood if the woman is actively bleeding. In addition, a Kleihauer–Betke test should be obtained on all women who are Rh negative to quantify the degree of fetomaternal transfusion to guide the dosing of Rh-immune globulin (RhoGAM).

No digital examinations should be performed when a woman presents with vaginal bleeding. Prenatal ultrasound records should be obtained and reviewed as quickly as possible. If previous ultrasound studies have confirmed a normal placental location, placenta previa can be ruled out, but other possibilities such as placental abruption or vasa previa should be considered.

Placenta Previa

Placenta previa may cause a small amount of vaginal bleeding, or it may result in acute hemorrhage. In the majority of cases, women with placenta previa will present with an initial episode of painless vaginal bleeding. During this first episode, the bleeding usually stops and the woman and fetus are stable. Expectant management in the hospital is initiated in such a case. During this hospitalization, the woman is monitored for signs of labor and, depending on the fetus's gestational age, steroids may be administered to enhance fetal lung maturity and/or magnesium sulfate administered for fetal neuroprotection.

In case of placenta previa, the woman's care is managed by a consulting physician. The midwife may continue in a collaborative role to provide emotional support and anticipatory guidance. Preparation for breastfeeding and parenting as well as family adjustments to hospitalization are an important midwifery component of the woman's plan of care.

Placental Abruption (Abruptio Placentae)

One of the leading causes of obstetric hemorrhage in the second and third trimesters is placental abruption, defined as a premature separation of a normally implanted placenta.[88] Placental abruption occurs in approximately 1% of all pregnancies and is associated with significant perinatal mortality and morbidity.[89,90]

In most cases of abruption, bleeding occurs between the membranes and the decidua, and passes through the cervix into the vagina. Less commonly, the abruption is concealed, such that the blood accumulates behind the placenta with no obvious vaginal bleeding. In such a case, the blood may penetrate through the uterine decidua and escape into the peritoneum. This phenomenon is also termed a Couvelaire uterus.

An abruption can be either total (involving the entire placenta) or partial (involving only a portion of the placenta) (**Figure 29-3**). A total abruption is a catastrophic obstetric event, often leading to fetal death and significant maternal morbidity.

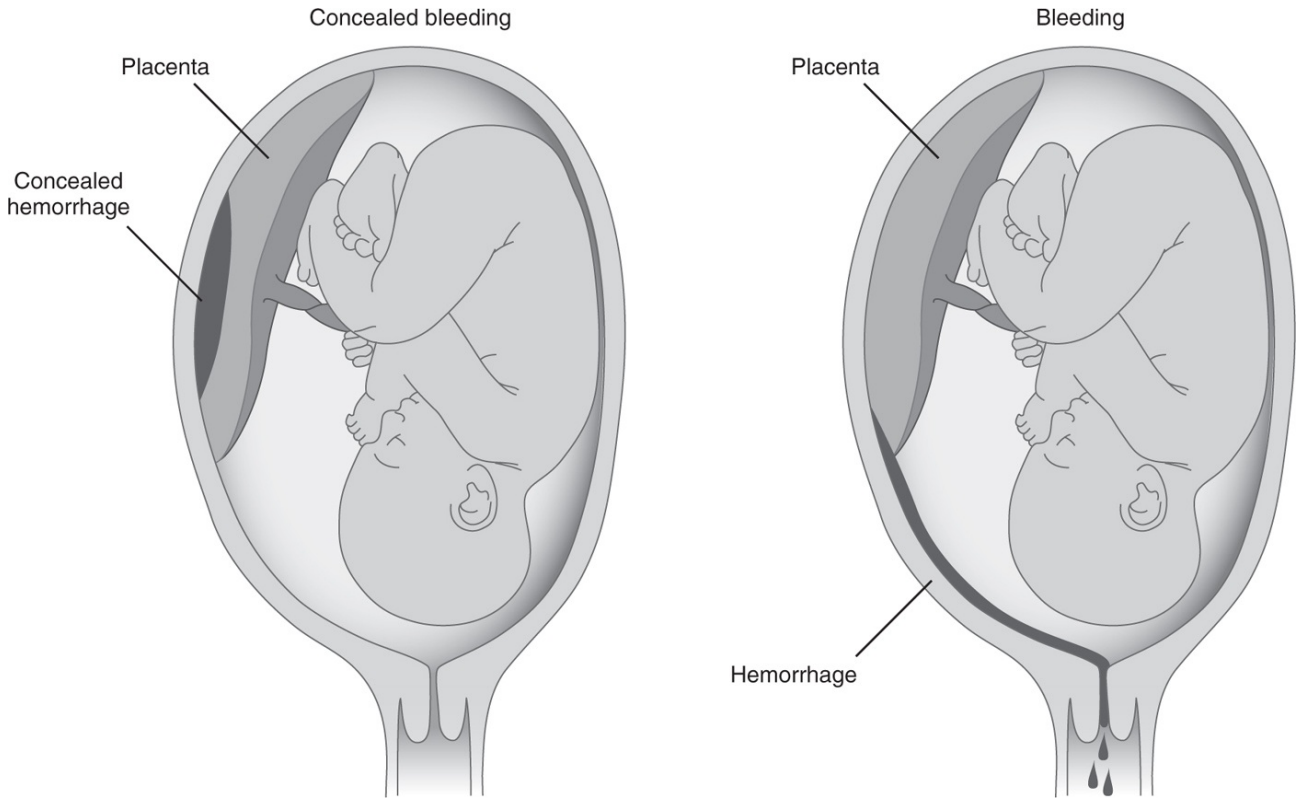


Figure 29-3 Placental abruption.

Although the specific etiology of abruption is unknown, several factors associated with it have been identified (**Table 29-14**). The most common of these risk factors is hypertensive disorders, especially chronic hypertension with superimposed preeclampsia.[89,90] A history of previous abruption, maternal smoking, and cocaine use also significantly increase the risks of placental abruption. In addition, any maternal trauma (e.g., fall, abdominal injury, intimate-partner violence, or motor vehicle accident) warrants an evaluation in the hospital to rule out an abruption. Women who are at more than 24 weeks' gestation should have continuous fetal monitoring for a minimum of 4 hours following trauma to the abdomen. If more than 6 contractions occur in any given hour, the monitoring should be continued for 24 hours.

Table 29-14	Risk Factors for Placental Abruption
Maternal Risks	
Advanced maternal age	
Cocaine use	
Smoking during pregnancy	
Obstetric Risk Factors	
Chorioamnionitis	
Fetal growth restriction	
History of placental abruption	
Hypertensive disorders (chronic, gestational, preeclampsia)	
Multifetal gestation	

Premature rupture of membranes
Polyhydramnios
Thrombophilias
Acute Etiology
Trauma (fall, abdominal injury, motor vehicle accident)
External cephalic version for breech presentation

Based on Oyelese Y, Ananth CV. Placental abruption. *Obstet Gynecol*. 2006:108:1005-1016.[89]; Hladky K, Yankowitz J, Hansen WF. Placental abruption. *Obstet Gynecol Surv*. 2002;57(5):299-305.[90]

The clinical presentation of an abruption depends on the degree of separation and blood loss. Maternal symptoms can vary widely, from mild generalized back discomfort or abdominal cramping to the classic symptoms of vaginal bleeding and severe abdominal pain. Early signs of an abruption can mimic those of labor, and the woman may report periodic mild contractions with what may be perceived as bloody show or bloody amniotic fluid. Low backache may be the only symptom if the placenta is posterior and there is a concealed abruption. The woman may report decreased fetal movement or even a recent surge in fetal activity.

On examination, the woman's demeanor or distress may be out of proportion to the objective clinical signs. The maternal vital signs may be normal with no obvious signs of hemodynamic compromise. The abdomen may be mildly tender with palpable contractions, or it may feel firm, rigid, and diffusely painful. A pelvic examination may reveal minimal to no bleeding, but this will not correlate with the amount of blood loss from a concealed hemorrhage. In most cases, frequent, low-amplitude contractions are seen on the external fetal monitor. Fetal status will also greatly depend on the degree of separation and blood loss. Thus, a variety of FHR patterns have been associated with a placental abruption, including recurrent late or variable decelerations, loss of FHR variability, bradycardia, or sinusoidal pattern.[89] Fetal demise is associated with more than a 50% placental separation; rarely does this degree of abruption occur without other overt symptoms.[89]

Ultrasonography is rarely helpful in confirming the diagnosis of placental abruption or in determining the degree of separation. Research has found that the sonographic appearance of placental separation varies considerably depending on how much time has passed from the acute bleeding event.[89] Ultrasound evaluation of a pregnant woman who presents with vaginal bleeding, however, will identify the placental location and the presence of a placenta previa. Although some degree of separation may be seen with placenta previa, it is considered a distinctly separate diagnosis and would, in effect, rule out placental abruption as the cause of bleeding.

When a woman presents with symptoms of abruption, she should immediately be assessed for signs of hemodynamic compromise and the fetus assessed for signs that suggest an increased risk for fetal acidemia. The continued management will depend in part on the gestational age and in part on the condition of the woman and fetus. The consulting physician should be notified immediately of the midwife's clinical assessment and evaluation and seek to determine the most appropriate plan of care. The midwife should also initiate the maternal/fetal stabilization measures and laboratory assessments discussed in the previous

section.

Although management may vary depending on the degree of maternal/fetal compromise, a diagnosis of placental abruption near term will warrant an expedited birth. In many cases, the placental abruption will stimulate vigorous contractions, with labor then rapidly progressing on its own. The midwife can collaboratively manage this situation with a consulting physician and conduct the birth as planned. During labor, the fetus should be monitored continuously and any signs of fetal compromise promptly reported to the physician. Even with a mild abruption, the situation could deteriorate rapidly, and the woman is at significant risk for coagulopathy and hypovolemic shock. In addition to blood transfusions, the woman may need aggressive replacement of coagulation factors. After the birth, the placenta should be sent to pathology and the woman closely monitored collaboratively with a physician until her condition stabilizes.

Prolapsed Umbilical Cord

There are three types of umbilical cord prolapse: occult, funic, and overt. An *occult* cord prolapse occurs when the umbilical cord is compressed alongside the presenting part in the lower uterine segment or inner cervix. The cord may not be palpable during a cervical examination but will often cause variable decelerations or FHR bradycardia similar to that seen with umbilical cord compression. In a *funic* umbilical cord prolapse, the cord is palpable in front of the presenting part. In an *overt* umbilical cord prolapse, the cord slips completely out of the uterus through the cervix and may be protruding at the vaginal introitus. Variant FHR patterns may be present depending on the degree of cord compression from the presenting part.

When a frank or complete umbilical cord prolapse is encountered, the woman will need an emergent cesarean birth and immediate interventions to improve fetal oxygenation. Risk factors and emergency management of a prolapsed umbilical cord are outlined in **Appendix 29B**.

Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is a rare obstetrical emergency that may occur from the onset of an apparently uncomplicated labor to within 30 minutes of placental expulsion. The incidence in the United States is 5.1% in 100,000 births.[91] AFE is characterized by sudden cardiorespiratory arrest that might include hypotension, dyspnea, and cyanosis.[92] Blood pressure falls rapidly and peripheral S_PO_2 is less than 90%. New uniform diagnostic criteria specify that there was no fever during labor and that disseminated intravascular coagulation (DIC) signs and symptoms become apparent before coagulopathy could be attributed to acute blood loss or shock-related consumptive coagulopathy.[92]

Clinically, a woman with AFE has sudden respiratory distress with simultaneous cardiovascular system deterioration. The cause of AFE was previously thought to be mechanical obstruction of pulmonary vasculature by fetal cells in amniotic fluid that had sudden inflow to maternal circulation. Pulmonary vasospasm documented during autopsies indicates an anaphylactoid reaction to fetal cells, therefore, AFE is now thought of as an immune-inflammatory reaction.[91] Differential diagnoses include occult hemorrhage, septic shock, pulmonary thromboembolism, and anaphylaxis. An anesthetic accident is a

consideration if symptoms occur during cesarean surgery.[92]

The responsibility of the midwife present when symptoms occur is to summon emergency medical assistance, initiate cardio-pulmonary resuscitation if needed and to provide help within the response team. The woman with AFE will require intensive care including cardiopulmonary support and correction of coagulopathy. The mortality rate, once thought to approximate 100%, is now calculated to range from 11% to 44% using population-based studies.[93] The midwife will have an important role in providing support to the woman during recovery and assisting her transition to motherhood.

Shoulder Dystocia

Shoulder dystocia is defined as a birth requiring extra obstetric maneuvers after gentle downward traction on the fetal head has failed to release the shoulders.[94,95] Shoulder dystocia is believed to be caused by impaction of the fetal anterior shoulder on the symphysis pubis, or less commonly the fetal posterior shoulder on the sacral promontory. Although there is no standard description of shoulder dystocia, failure to deliver the fetal shoulders by the usual methods, prolonged head-to-body delivery time, and need for ancillary maneuvers are suggested criteria.[94] The true incidence of shoulder dystocia is not known due to variations in research design, lack of consensus on its definition, and differences in populations studied.[94,95] A recent review of literature from 1985–2012 found the incidence of shoulder dystocia overall is 1.4% in the United States.[94] The incidence in women who do not have diabetes is 0.6%, and that in women with diabetes is 1.9%.[94]

Risk Factors for Shoulder Dystocia

Antepartum risk factors for shoulder dystocia include macrosomia, pregestational and gestational diabetes, previous shoulder dystocia, maternal obesity, excessive weight gain during pregnancy, and multiparity.[96] The combination of two or more of these factors significantly increases the risk of shoulder dystocia.[96] Macrosomia is an especially significant risk factor. The incidence of shoulder dystocia is thought to rise with increasing fetal size, especially among infants born to women with diabetes.[95] The principal intrapartum risk factors for shoulder dystocia are a precipitous second stage of labor and operative vaginal birth. Rapid descent of the fetus during second-stage labor may not allow enough time for normal internal rotational maneuvers and compression of the shoulders toward the thorax, which results in impaction of the anterior shoulder behind the pubic bone. Midwives managing the second stage of labor for women with any of these risk factors should have a low threshold for requesting the immediate availability of the consulting obstetrician.

Prediction and Prevention Strategies

It is impossible to accurately predict shoulder dystocia. Known risk factors have a very low positive predictive value,[96] in part due to the lack of an accurate means of diagnosing macrosomia prenatally. Also, the great majority of macrosomic infants do not have shoulder

dystocia, and 48% of shoulder dystocias occur with infants who are not macrosomic. Cesarean birth prevents shoulder dystocia and most neonatal morbidity associated with an impacted shoulder. Nevertheless, planned cesarean birth for suspected macrosomia is not recommended due to the increased maternal morbidity, economic cost, and unreliable predictive accuracy of macrosomic infants, with two exceptions: (1) women with diabetes whose fetus has an estimated fetal weight of more than 4500 grams and (2) women without diabetes whose fetus has an estimated fetal weight of more than 5000 grams.[95]

Complications of Shoulder Dystocia

Even when managed appropriately, shoulder dystocia can result in neonatal injuries, such as a fractured humerus or clavicle and either temporary or permanent damage to the infant's brachial plexus (Erb's palsy). Prolonged fetal asphyxia during a difficult shoulder dystocia can also result in neonatal encephalopathy, seizures, and infant death. Maternal complications may include excessive blood loss (e.g., from postpartum hemorrhage) and perineal, vaginal, or rectal lacerations. Last-resort maneuvers such as cephalic replacement or abdominal rescue via cesarean birth significantly increase the risks for both maternal morbidity and perinatal mortality.

The neonatal consequences of shoulder dystocia are at the root of many medical lawsuits. For this reason, brachial plexus injury in particular has received considerable attention in the obstetric literature. This injury is thought to occur from forceful stretching or hyperextension of one side of the neck, which then results in avulsion or rupture of one or all of the brachial plexus nerves. Lateral traction applied to the infant's neck while attempting to deliver the anterior shoulder is the likely cause. While research has attempted to quantify the degree of force needed to cause a brachial plexus injury, this injury can also occur prior to birth and may be unavoidable in certain circumstances.[94]

Management of Shoulder Dystocia

The detailed management of shoulder dystocia can be found in Appendix 29A. For many years, the management of shoulder dystocia followed a specific protocol that began with external manipulation combined with maternal pushing efforts, which was often repeated several times before internal rotational maneuvers were attempted. Current evidence suggests shoulder dystocia management should focus on internal maneuvers such as rotation and delivery of the posterior arm, without maternal pushing efforts or placing any traction on the infant's neck.[97-100] This "hands-off" approach may decrease brachial plexus injury.[97-100]

Two recent commentaries argue in favor of a hands-off approach with all births.[100,101] These authors advocate expulsion of the shoulders during a normal birth by maternal effort only, waiting for the next contraction after birth of the head if necessary. It is theorized that this approach may allow for more complete internal rotation of the shoulders, possibly preventing some cases of shoulder dystocia.[100] It also avoids the lateral traction that may, in fact, be the cause of some cases of brachial plexus injury, even when the traction is not excessive.[101]

All professional organization recommendations agree that fundal pressure should never be

used, and that episiotomy should be performed only if extra room is needed for internal maneuvers.[95] ACOG recommends McRoberts' maneuver as a reasonable first step, while stating that there is no evidence of efficacy or safety that favors one release maneuver over another.[95] The Royal College of Obstetricians and Gynaecologists (RCOG) recommends against the use of any lateral traction to facilitate delivery of the shoulders, even in the absence of shoulder dystocia, recommending axial traction instead (i.e., in line with the fetal spine).[102] Maternal pushing efforts should not occur until the impacted shoulder is released.[102] The Society of Obstetricians and Gynaecologists of Canada (SOGC) recommends against maternal pushing efforts and any traction that causes severe neck traction.[103] Although there is not yet consensus on the issue of maternal pushing efforts and lateral traction to release an impacted shoulder, midwives should consider the theoretical concerns of these practices when managing a shoulder dystocia.

Another issue to consider is the use of the hands and knees position as a strategy to resolve a shoulder dystocia. Both RCOG and SOGC include this position in their shoulder dystocia algorithms after McRoberts' maneuver, rotational maneuvers, and delivery of the posterior arm, all with the mother lying on her back.[103,104] The rationale for this strategy is apparently assumed unfamiliarity with birth in this position, though midwives tend to have a greater familiarity with women giving birth on hands and knees. In addition, numerous anecdotal reports cite the woman's movement to this position itself relieving the impacted shoulder. If the woman is mobile, use of this strategy earlier than indicated in the RCOG and SOGC algorithms is reasonable.

Shoulder Dystocia Simulation

Given that the incidence of shoulder dystocia is relatively low and risk factors are not predictive, obtaining competency in management of this complication is challenging. While popular mnemonics are helpful memorization tools, evidence suggests simulation and team training are more likely to improve skills and confidence in managing a shoulder dystocia.[105-107] Research on simulation specifically related to shoulder dystocia has also shown that the use of high-fidelity manikins is preferred, especially when trying to learn and practice rotational maneuvers.[105-107] "High fidelity" refers to the ability of the equipment, the environment, and the learners to simulate and act as if the learning scenario is a real-life situation.[108]

Another advantage of simulation is the ability to train groups of learners to work together as a multidisciplinary team for resolving a specific clinical emergency, such as shoulder dystocia. At the end of the scenario, participants have an opportunity to debrief on the experience under the guidance of a skilled simulation coordinator. Teaching points are highlighted and the team is given constructive feedback related to the original learning objectives for the session. Studies have shown both improvements in confidence and skills when simulation is used for shoulder dystocia training.[105-107] Even though high-fidelity simulation centers may not be immediately available to all students and providers, the benefits of this type of learning experience in improving critical thinking and skills with shoulder dystocia management cannot be overstated.

Twin Gestation

In recent years, the incidence of twins and higher-order multifetal gestations has increased primarily due to the availability of assisted reproductive technologies. Currently, twin gestations occur in approximately 3% of all pregnancies in the United States and account for 17% of preterm births. Twins are associated with a sevenfold increased neonatal death rate compared to singleton gestations.[109] In addition, higher rates of congenital anomalies, fetal growth restriction, and maternal complications such as gestational hypertension, preeclampsia, and diabetes are found among women with multifetal gestations.[109] Lastly, intrapartum complications such as malpresentation, prolapsed umbilical cord, placental separation, and postpartum hemorrhage occur more frequently in twin gestations.[110]

When referring to twin gestations, it is critical to know the chronicity (one or two placentas) and the amnionicity (one or two amniotic sacs)—that is, the twins will be either dichorionic–diamniotic (di-di), monochorionic–diamniotic (mono-di), or monochorionic–monoamniotic (mono-mono). Chorionicity is explained further in the *Pregnancy-Related Conditions* chapter.

In most midwifery practice settings, women with a multifetal gestation require physician referral, especially if an intervening amniotic membrane has not been identified (mono-mono). For either mono-di or di-di twins, a collaborative management plan with the consulting physician can be considered (per practice guidelines) with strict criteria for referral of care. The woman will need ongoing surveillance for both maternal and fetal complications throughout the remainder of her pregnancy. An individualized plan of care regarding the optimal timing and mode of birth with the appropriate informed consent discussion will also be needed.

Mode of Birth

The mode of birth depends on the fetal presentation. Therefore, as the pregnancy progresses, the presentation and lie of each fetus will be carefully assessed and documented in the woman's prenatal record. Women with diamniotic twins in a vertex–vertex presentation are ideal candidates for a vaginal birth.[109,111] This option should be discussed with the woman well before the onset of labor, and the risks and benefits of vaginal birth versus elective cesarean section thoroughly reviewed. Current evidence does not show that cesarean birth improves perinatal outcomes in vertex–vertex presentations, and vaginal birth should be encouraged for all vertex–vertex presenting twins.[109,111]

Women at or beyond 32 weeks' gestation with diamniotic twins, with a vertex presentation of the first twin and a breech or transverse presentation of the second twin, are also candidates for vaginal birth if a provider skilled in internal podalic version and vaginal breech birth is present.[109,111] When the presenting fetus is breech, cesarean birth is recommended.[109,111]

Upon admission in labor, the presentation and lie of each fetus should be reassessed with ultrasound. The midwife should notify the consulting physician, and a collaborative management plan should be established if the woman desires a trial of labor. Intrapartum management guidelines for managing a woman with a twin gestation are found in **Appendix 29C**. Midwives may be required to assist a woman with a twin gestation or breech

presentation during birth if a physician is not able to be present when the birth occurs; thus, these skills are periodically reviewed as needed.

Fetal Malpresentations

The three fetal presentations possible are cephalic, shoulder, and breech. Cephalic presentations include vertex, sinciput (forehead), brow, and face, based on the degree of the flexion or extension of the fetal head.

- Vertex presentation: The occiput is the presenting part. If the fetus is in an occiput-anterior position, the smallest anterior-posterior and transverse diameters of the fetal head are presenting through the birth canal. This is the optimal position for vaginal birth. Variations include occiput posterior which is associated with prolonged labor, particularly prolonged second stage, and deep transverse arrest which is associated with arrest of dilation or arrest of descent in either the first or second stage of labor. In addition if the fetus is asynclitic, presenting diameters are enlarged and labor progress may be slower.
- Sinciput presentation: The fetal head is partially extended. The fetus in this position usually converts to vertex as the fetal head flexes toward the chin.
- Brow presentation: If the fetal head remains in the military position between full extension and full flexion, the brow becomes the presenting part. This presentation is the least common. The fetus in a brow presentation can convert to an occiput or face presentation but if the fetal head remains in this position, cesarean will be required because the mento-vertex diameter (13 cms) is the largest of the fetal head diameters and vaginal birth is precluded.
- Face presentation: If the fetal head is completely extended so the fetal occiput is in contact with the fetal back, the face will present. If the woman has an adequate pelvis, this fetus can rotate to a mentoanterior position and vaginal birth will ensue. If the fetus rotates to a mentoposterior position, cesarean section will be needed.

A shoulder presentation is possible for a fetus in a transverse lie. A cesarean birth is required if the fetus does not convert to a cephalic or breech presentation by the time labor starts.

Breech Birth

Even with careful assessment techniques, some women will present in labor with an undiagnosed breech presentation; thus, a breech presentation may be first diagnosed during a vaginal examination. Palpation of fetal small parts, usually the feet, or the buttocks may be encountered, although sometimes it is very difficult to distinguish the fleshy part of the breech from the soft-tissue caput on the fetal vertex. In some cases, the breech presentation is not discovered until the woman is well into active labor when fresh meconium stool is seen coming from the vagina. Once a breech presentation is confirmed, the woman should be informed and the consulting physician notified immediately. If the birth is imminent or a cesarean birth is not immediately available, the midwife should be prepared to perform a vaginal breech birth. **Appendix 29D** reviews the management steps for a vaginal breech birth. All midwives caring for laboring women should be thoroughly familiar with these techniques.

Prior to the late 1990s, vaginal breech birth was a common occurrence and an important component of obstetric training for both midwives and physicians. The publication of the Term Breech Trial in 2000, however, dramatically changed practice patterns regarding vaginal breech births.[112] This multicenter, randomized study originally reported that elective cesarean section for breech presentation significantly decreased perinatal morbidity and mortality. Almost immediately, women with a breech presentation were no longer given the option for vaginal birth. Closer examination of the definitions and methodology used in the Term Breech Trial has since challenged the reliability and validity of these findings.[113] Several protocol violations have been identified, and subsequent studies have reported conflicting results with regard to perinatal outcomes based on mode of birth for the fetus in a breech presentation.[112-115] In brief, current evidence suggests vaginal breech birth is associated with an increased risk of some adverse neonatal outcomes such as perinatal mortality and low Apgar scores, but studies completed to date have not had sufficient power to accurately identify the incidence of these outcomes. Maternal morbidity is higher in women who undergo cesarean section, and follow-up of children at age 2 years also reveals an increased incidence of medical problems within the cohort of children born via cesarean section.[115]

Unfortunately, these updated findings have failed to completely reverse the clinical changes brought about by the Term Breech Trial. The current medico-legal environment has continued to influence practice, with the result that women with a breech presentation today are still often advised to have an elective cesarean birth. More importantly, since the Term Breech Trial was published, expertise in vaginal breech birth and clinical learning opportunities have diminished. As a result, vaginal breech birth is now included in simulation training curricula along with other high-risk obstetric skills such as shoulder dystocia management. Midwives are encouraged to participate in these training experiences whenever possible.

Some women with a breech presentation will decline surgery or seek out a provider who will perform a planned vaginal breech birth. Current guidelines for these circumstances have been established, and institutions that can safely offer this service are encouraged to develop protocols for intrapartum management.[114] The option for planned vaginal breech birth is based on the availability (and willingness) of an obstetrician experienced in this mode of birth. A thorough shared decision-making discussion with the woman is also mandatory. Some sources

have outlined specific selection criteria shown to improve success rates, such as sonographically determined estimated fetal weight between 2500 and 4000 grams, frank or complete breech, no fetal anomalies, adequate maternal pelvis, adequate amniotic fluid, and flexion of the fetal head (documented by ultrasound).[114]

Special Clinical Situations

Birth is often thought of as the beginning of a person's life—but it can also be the end. Although birth is a joyous occasion for most women, stillbirth and the birth of a newborn with a critical illness or lethal anomaly are among the most profound traumas that a family can experience. These clinical scenarios can be traumatic for all involved.

Intrapartum Care of a Woman with a Stillbirth

The majority of women who are identified as having a stillbirth will opt for an in-hospital birth. The following discussion addresses this situation.

Hospital staff should be informed of the diagnosis before the woman's arrival and have a labor room arranged for her that will ensure privacy and liberal visitation from family members. Many labor and birth units have mechanisms in place that discreetly communicate a fetal demise (or lethal anomaly) to the entire staff (e.g., a special door magnet). It is especially important that the woman is not accidentally approached by a staff member who is unaware of her diagnosis and assumes she is a "normal" labor patient expecting a healthy newborn.

The midwife's role in the care of a woman with a stillbirth or a fetus with a lethal anomaly will vary according to individual practice guidelines and the specific clinical circumstances. A physician should be consulted to establish the most appropriate plan of care, which may in some cases require referral for medical management. The midwife's continued presence and availability, however, can provide emotional support and comfort for the woman and her family, especially in the immediate postpartum period. Midwives caring for women in these situations will have their own emotions and sadness to deal with. The priority is always to focus on the woman's immediate needs during labor and birth.

Once the woman is admitted, the midwife should thoroughly review the prenatal chart and all documentation of previous discussions regarding the proposed plan of care. A complete blood count and coagulation studies (especially platelets and fibrinogen) are obtained on admission to provide a baseline assessment of clotting factors due to the potential risk of disseminated intravascular coagulation (usually seen with prolonged fetal retention of . 2 weeks).[116,117] If an induction of labor is planned, a physician should be consulted to determine the most appropriate method of induction and cervical ripening. The specific agents and protocols used will depend on the woman's diagnosis, fetal gestational age, past obstetric history, and current Bishop's score.[116] This plan should be reviewed again with the woman and her family, with clear explanations of what to expect as labor and birth progress. The woman may need information repeated and should be given opportunities to express her feelings and ask questions. Every effort must be made to honor the woman's choices and requests.

Most labor and birth units have specific protocols and checklists in place for bereaved parents that include referrals to social services, the availability of a chaplain, assistance with burial plans, and collection of photographs, footprints, and other keepsakes from the baby after the birth has occurred. These services are offered to the woman at the appropriate times over the course of hospitalization. Religious and cultural choices are supported and respected (e.g., desire to baptize the infant).

The woman and her support persons will also be informed about the availability of detailed fetal evaluation after the birth. This evaluation includes a standard autopsy, collection of blood or tissue samples, and a pathology evaluation of the placenta and umbilical cord.[116] Less invasive options may also be available such as photographs, X-ray imaging, and ultrasonography.[116] It can be gently emphasized that this information may be useful to her and her family in planning future pregnancies. Tissue or amniotic fluid samples for fetal karyotype, in particular, may diagnose a syndrome or chromosomal abnormality without the results from a full autopsy.[116] If she declines autopsy or tissue sample collection, the placenta and umbilical cord should still be sent to pathology after the birth.

Parents may have difficulty making firm decisions, especially with regard to seeing or holding the infant at birth. If the woman has previously stated that she does not want to see the infant after the birth, the nursing staff should be prepared to take the infant to another location after the birth occurs. Women may change their minds about this point, however, and the midwife should remain open to all possibilities.

The birth itself is managed the same as with any other vaginal birth, except that there may be softening or overriding of the fetal skull bones or anomalies not previously identified. In addition, there is an increased risk of shoulder dystocia. Caution should be used when applying even gentle traction to any part of the fetus during the birth; instead, it is best to rely mostly on the woman's expulsive efforts. The umbilical cord should be clamped and cut as close to the abdomen as possible and the infant gently wrapped in a towel. The placenta should be delivered in the usual fashion and any lacerations repaired.

After the birth, the infant, umbilical cord, and placenta should be carefully examined for any recognizable abnormalities. Any deviations from normal (e.g., a true knot in the umbilical cord) should be shown to the woman and her partner. Sometimes seeing an obvious cause of death is reassuring for grieving families who are searching for answers. Unfortunately, the majority of intrauterine deaths cannot be attributed to a known cause, so it is important not to offer a potential diagnosis without clear evidence.

The woman and her family can be encouraged to view, touch, and hold the infant and allowed as much time as needed. Often photographs are taken and keepsakes (e.g., a lock of hair and footprints) are obtained and given to the parents. Once they are ready to say goodbye, the infant can be brought to the morgue per hospital policy.

Life-Threatening or Lethal Fetal Diagnosis

Due to improvements in diagnostic technology, more parents have advanced knowledge of a life-threatening fetal diagnosis prior to birth. In some cases, this early diagnosis may enable prenatal transfer of the woman to a tertiary center and consultation with neonatal specialists capable of interventions that will improve the infant's survival. In other cases, the woman and her partner have the task of deciding whether to continue the pregnancy to term. Depending on the gestational age at the time of diagnosis and individual state laws, elective termination of the pregnancy beyond the second trimester or a preterm induction of labor may not be an option. This section focuses on pregnant women with a known lethal fetal anomaly who present at full-term gestations either in spontaneous labor or for a planned induction.

In the majority of cases, the woman and her family will likely be receiving care from a multidisciplinary team that includes neonatology, maternal–fetal medicine, genetics specialists, and social services. In some settings, a perinatal palliative care plan has been established, and many of the decisions facing grieving parents during labor and birth have been discussed with the woman and her partner. Perinatal palliative care is an emerging field that moves beyond traditional bereavement support and is initiated at the time of diagnosis rather than during labor and birth.[118] This holistic approach incorporates the principles of bereavement care by integrating the woman's choices and understanding with realistic goals for care of the baby after birth, including perinatal hospice care. Ensuring quality of life, reducing pain and suffering, and honoring the infant's life and death are at the core of this framework.[118]

Once the woman is admitted to the labor unit, a collaborative plan of care is required with a consulting physician. Other care team providers (e.g., neonatology, pediatrics, chaplaincy) are notified of the impending birth. Depending on the type of anomaly, the gestational age, the expected perinatal outcome, and maternal medical conditions, physician management may be necessary.

The intrapartum management in this situation is similar to the care of a woman with a stillbirth. Regardless of the amount of prenatal counseling and support, the woman may exhibit a wide range of emotional responses during labor. All pain relief options should be available if requested. In addition, as second-stage labor nears, she may experience more difficulty coping and become quite conflicted about pushing. The birth represents facing the confirmation that the infant is going to die, and "holding on" subconsciously prevents this outcome from happening. Providing gentle encouragement, support, and patience will allow the woman to work through any emotional tethering and facilitate her sense of control over the situation.

If the neonatal team is planning to examine the newborn after birth, it may be less distracting for the woman to have them wait outside the room until after the birth occurs. Having a perinatal palliative care plan in place will facilitate the seamless transition from birth to end-of-life care.[118]

Conclusion

Complications during labor and birth can occur at any time, and knowledge of these conditions is essential for midwifery practice. Understanding the condition, its natural course, the effects on the woman and her fetus, and the degree of consultation, collaboration, or referral required are the basic competencies needed for all obstetric complications. In addition, because emergencies are unpredictable, management of the most common types will enable midwives to care for women safely. All women who experience a complication intrapartally will greatly benefit from the continuity, education, and support that a midwife provides.

Resources

Organization	Description	Webpage
Hypertension and Preeclampsia Toolkits		
American College of Obstetricians and Gynecologists (ACOG)	Resources for diagnosis and management of hypertensive disorders of pregnancy. Includes recent publications and guidelines.	https://www.acog.org/Womens-Health/Preeclampsia-and-Hypertension-in-Pregnancy
California Maternal Quality Care Collaborative (CMQCC)	Preeclampsia toolkit. Includes guidelines, algorithms, and resources for caring for women with preeclampsia.	https://www.cmqcc.org/resources-tool-kits/toolkits/preeclampsia-toolkit
Maternal Early Obstetric Warning System (MEOWS) and Maternal Early Warning Criteria (MERC)		
American College of Obstetricians and Gynecologists (ACOG)	Maternal early warning system.	https://www.acog.org/-/media/Districts/District-II/Public/SMI/v2/MaternalEarlyWarningSystem.pdf?dmc=1&ts=20171023T1836554977
Council on Patient Safety	Maternal safety bundles.	https://safehealthcareforeverywoman.org/wp-content/uploads/2017/02/MEWS-Protocol.pdf
Preterm Birth		
National Institutes of Health (NIH)	Preterm Labor and Birth: For Researchers and Health Care Providers. Website has a collection of links to research, guidelines, and professional organizations involved in preterm labor and birth.	https://www.nichd.nih.gov/health/topics/preterm/resources/Pages/providers.aspx
Obstetric Emergencies		
American College of Obstetricians and Gynecologists (ACOG)	Preparing for Clinical Emergencies in Obstetrics and Gynecology: Committee Opinion No. 590.	https://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Patient-Safety-and-Quality-Improvement/Preparing-for-Clinical-Emergencies-in-Obstetrics-and-Gynecology

	2014 (reaffirmed 2016). This document lists triggers and recommends tools for managing obstetric emergencies based on the Modified Early Obstetric Warning System from the United Kingdom.	
Apps		
American College of Obstetricians and Gynecologists (ACOG)	Indicated Delivery Calculator. This app summarizes all ACOG professional guidelines and best evidence for gestational age at which induction is indicated for multiple obstetric and medical complications.	https://www.acog.org/About-ACOG/ACOG-Departments/ACOG-Rounds/September-2017/Early-Indicated-Delivery-ACOG-Has-an-App-for-That

References

1. Gee RE, Corry MP. Patient engagement and shared decision making in maternity care. *Obstet Gynecol.* 2012;120:995-99
2. Frey HA, Klebanoff MA. The epidemiology, etiology and costs of preterm birth. *Semin Fetal Neonatal Med*. 2016;21:68-73.
3. Hedriana H, Byrne J, Campbell Bliss M, et al. *March of Dimes Preterm Labor Assessment Toolkit.* White Plains, NY: March of Dimes; 2013.
4. Wax JR, Cartin A, Pinette MG. Biophysical and biochemical screening for the risk of preterm labor: an update. *Clin Lab Med*. 2016;36:369-383.
5. Koulalli B, Oudijk MA, Nijman TA, Mol BW, Pajkrt E. Risk assessment and management to prevent preterm birth. *Semin Fetal Neonatal Med*. 2016;21:80-88.
6. Sanchez-Ramos L, Delke I, Zamora J, Kaunitz AM. Fetal fibronectin as a short-term predictor of preterm birth in symptomatic patients. *Obstet Gynecol.* 2009;114(3):631-640.
7. American College of Obstetricians and Gynecologists. Practice Bulletin No. 13: prediction and prevention of preterm birth *Obstet Gynecol.* 2012;120:964-973.
8. Society for Maternal-Fetal Medicine; McIntosh J, Feltovich H, Berghella V, Manuck T. The role of routine cervical length screening in selected high- and low-risk women for preterm birth prevention. Society for Maternal-Fetal Medicine Consult Series #40. 2016. *Am J Obstet Gynecol*. 2016;15(3):B2-B7.
9. American College of Obstetricians and Gynecologists. Practice Bulletin No. 171: management of preterm labor. *Obstet Gynecol.* 2016;128:931-933.
10. Nijman TA, Vliet EO, Koullali B, Mol BW, Oudijk MA. Antepartum and intrapartum interventions to prevent preterm birth *Semin Fetal Neonatal Med*. 2016;21:121-128.
11. Gyamfi-Bannerman EA, Thom EA, Blackwell SC, et al. Antenatal betamethasone for women at risk for later preterm delivery. *N Engl J Med*. 2016;374:1311-1320.
12. Centers for Disease Control and Prevention. Prevention of perinatal group B streptococcal disease: revised guidelines fror CDC 2010. *MMWR*. 2010;59(RR-10). Available at: https://www.cdc.gov/mmwr/pdf/rr/rr5910.pdf. Accessed October 20, 2017.
13. Gulmezoglu AM, Crowther CA, Middleton P, Heatley E. Induction of labor for improving birth outcomes for women at or beyond term. *Cochrane Database Syst Rev.* 2012;6:CD004945. doi:10.1002/14651858 .CD004945.pub3.
14. Wennerholm UB, Hagberg H, Brorsson B, Bergh C. Induction of labor versus expectant management for post-date pregnancy: is there sufficient evidence for a change in clinical practice? *Acta Obstet Gynecol Scand*. 2009;88:6-17.
15. Sanchez-Ramos LF, Oliver F, Delke I, Kaunitz AM. Labor induction versus expectant management for postterm pregnancies: a systematic review with meta-analysis. *Obstet Gynecol.* 2003;101:1312-1318.
16. American College of Obstetricians and Gynecologists. Practice Bulletin No. 146: management of late term and postterm pregnancies. *Obstet Gynecol.* 2014;124:182-192. [Reaffirmed 2016].
17. Edwards A, Elwyn G, Mulley AL. Explaining risks: turning numerical data into meaningful pictures. *Br Med J*. 2002;324:827-830.
18. Marowitz A. Evidence-based management of prelabor rupture of the membranes at term. In: Anderson B, Rooks J, Barroso R, eds. *Best Practices in Midwifery* (2nd ed.). New York, NY: Springer; 2017:411-427.
19. Marowitz A, Jordan R. Midwifery management of prelabor rupture of membranes at term. *J Midwifery Womens Health*. 2007;52:199-206.
20. Hannah ME, Ohlsson A, Farine D, et al. Induction of labor compared with expected management for prelabor rupture of the membranes at term. *N Engl J Med* 1996;334:1005-1010.
21. Middleton P, Shepherd E, Flenady V, McBain RD, Crowther CA. Planned early birth versus expectant management (waiting) for prelabour rupture of membranes at term (37 weeks or more). *Cochrane Database Syst Rev.* 2017;1:CD005302. doi:10.1002/14651858.CD005302.pub3.
22. American College of Obstetricians and Gynecologists. Practice Bulletin No. 188: premature rupture of membranes. *Obste Gynecol.* 2018;131(1):187-188.
23. American College of Nurse-Midwives position statement: premature rupture of membranes at term. ACNM Division of Standards and Practice, Clinical Practice Section; 2008 (reviewed 2012). Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000233/PROM%20Mar%20201
24. American College of Obstetricians and Gynecologists. Committee Opinion No. 712: intrapartum management of intraamniotic infection. *Obstet Gynecol.* 2017;130:e95-e101.
25. Higgins RD, Saade G, Polin RA, et al. Evaluation and management of women and newborns with a maternal diagnosis of chorioamnionitis. *Obstet Gynecol.* 2016;127:426-436.
26. Fahey JO. Clinical management of intra-amniotic infection and chorioamnionitis: a review of the literature. *J Midwifery Womens Health*. 2008;53:227-235.
27. Cahill AG, Duffy CR, Odibo AO, Roehl KA, Zhao Q, Macones GA. Number of cervical examinations and risk of

intrapartum maternal fever. *Obstet Gynecol.* 2012;119:1096-1101.
28. Fishman SG, Gelber SE. Evidence for the clinical management of chorioamnionitis. *Semin Fetal Neonatal Med*. 2012;17:46-50.
29. American College of Obstetricians and Gynecologists. Practice Bulletin No. 107: induction of labor. *Obstet Gynecol.* 2009;114:386-397. [Reaffirmed 2016].
30. Bonsack CF, Lathrop A, Blackburn M. Induction of labor: update and review. *J Midwifery Womens Health*. 2014;59:606-615.
31. Glantz J. Elective induction vs. spontaneous labor associations and outcomes. *J Reprod Med*. 2005;50:235-240.
32. Little SE, Caughey AB. Induction of labor and cesarean: what is the true relationship? *Clin Obstet Gynecol*. 2015;58(2):269-281.
33. Caughey AB, Sundaram V, Kaimal AJ, et al. Systematic review: elective induction of labor versus expectant management of pregnancy. *Ann Intern Med*. 2009;151:252-263.
34. Osmundsen S, Ou-Yang R, Grobman W. Elective induction compared with expectant management in nulliparous women with a favorable cervix. *Obstet Gynecol.* 2010;116:601-605.
35. Osmundsen S, Ou-Yang R, Grobman,W. Elective induction compared with expectant management in nulliparous women with an unfavorable cervix. *Obstet Gynecol.* 2011;117:583-587.
36. Wood S, Cooper S, Ross S. Does induction of labour increase the risk of caesarean section? A systematic review and meta-analysis of trials in women with intact membranes. *BJOG*. 2013;121:674-685.
37. Miller NR, Cypher RL, Foglia LM, Pates JA, Nielsen PE. Elective induction of labor compared with expectant management in nulliparous women at 39 weeks of gestation: a randomized controlled trial. *Obstet Gynecol*. 2015;126(6):1258-1264.
38. O'Rourke PT, Girrardi GJ, Balaskas TN, et al. Implementation of a system-wide policy for labor induction. *MCN*. 2011;36:305-311.
39. Fisch JM, English D, Pedaline S, Brooks K, Simhan HN. Labor induction process improvement: a patient quality-of-care initiative. *Obstet Gynecol*. 2009;113:797-803.
40. Bishop EH. Pelvic scoring for elective induction. *Obstet Gynecol.* 1964;24:266-268.
41. Lange AP, Secher NJ, Westergaard JG, Skovgård I. Prelabor evaluation of inducibility. *Obstet Gynecol*. 1982;60(2):137-147.
42. American College of Nurse-Midwives position statement: induction of labor. ACNM Division of Standards and Practice; 2016. Available at: http://www.midwife.org/index.asp?bid=59&cat=3&button=Search.
43. Yount S, Lassiter N. The pharmacology of prostaglandins for induction of labor. *J Midwifery Womens Health*. 2013;58:133-144.
44. Chen W, Zue J, Peprah MK, et al. A systematic review and network meta-analysis comparing the use of Foley catheters, misoprostol, and dinoprostone for cervical ripening in induction of labor. *BJOG*. 2016;123(3):346-354.
45. Macones GA, Cahill A, Stamilio DM, Odibo AO. The efficacy of early amniotomy in nulliparous labor induction: a randomized controlled trial. *Am J Obstet Gynecol.* 2012;207:403.e1-403.e5.
46. Clark SL, Simpson KR, Knox GE, Garite TJ. Oxytocin: new perspectives on an old drug. *Am J Obstet Gynecol.* 2009;200:35.e1-35.e6.
47. Simpson KR. Clinicians guide to the use of oxytocin for labor induction and augmentation. *J Midwifery Womens Health*. 2011;56:214-221.
48. Gimpl G, Fahrenholz F. Oxytocin receptor system: structure, function, and regulation. *Physiol Rev.* 2001;81(2):629-683.
49. Page K, McCool WM, Guidera M. Examination of the pharmacology of oxytocin and clinical guidelines for use in labor. *J Midwifery Womens Health*. 2017;62:425-433.
50. Wei S, Luo Z, Qi H, Xu H, Fraser WD. High-dose vs low-dose oxytocin for labor augmentation: a systematic review. *Am J Obstet Gynecol.* 2010;204:296-304.
51. Budden A, Chen LJY, Henry A. High-dose versus low-dose oxytocin infusion regimens for induction of labour at term. *Cochrane Database Syst Rev.* 2014;10:CD009701. doi:10.1002/14651858.CD009701.pub2.
52. Simpson KR, Miller L. Assessment and optimization of uterine activity during labor. *Clin Obstet Gynecol.* 2011;54:40-49.
53. Macones GA, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. *Obstet Gynecol.* 2008;112:661-666.
54. Dow M, Wax JR, Pinette MG, Blackstone J, Cartin A. Third-trimester uterine rupture without previous cesarean: a case series and review of the literature. *Am J Perinatol.* 2009;26:739-744.
55. Kavanagh J, Kelly AJ, Thomas J. Breast stimulation for cervical ripening and induction of labour. *Cochrane Database Syst Rev* 2005;3:CD003392. doi:10.1002/14651858.CD003392.pub2.
56. McFarlin BL, Gibson MH, O'Rear J, Harman P. A national survey of herbal preparation use by nurse-midwives for labor stimulation. *J Midwifery Womens Health*. 1999;44:205-216.
57. Hall HG, McKenna LG, Griffiths DL. Complementary and alternative medicine for inductions of labor. *Women Birth*.

2012;25:142-148.
58. Dugoua JJ, Perri D, Seely D, Mills E, Koren G. Safety and efficacy of blue cohosh (*Caulophyllum thalictroides*) during pregnancy and lactation. *Can J Clin Pharm.* 2008;15:e66-e73.
59. Neal JL, Lowe, NK. Physiologic partograph to improve birth safety and outcomes among nulliparous women with spontaneous labor onset. *Med Hypotheses*. 2012;78:319-326.
60. Neilson J, Lavender T, Quenby S, Wray S. Obstructed labour: reducing maternal death and disability during pregnancy. *Br Med Bull.* 2003;67:191-204.
61. American College of Obstetricians and Gynecologists, Society for Maternal-Fetal Medicine. Obstetric Care Consensus No 1: safe prevention of the primary cesarean delivery. *Obstet Gynecol.* 2014;123:693-711.
62. Neal JL, Ryan SL, Lowe NK, Buxton M, Holley SL, Wilson-Liverman AM. Labor dystocia: use of related nomenclature. *Midwifery Womens Health* 2015;60:485-498.
63. Friedman EA. Primigravid labor: a graphicostatistical analysis. *Obstet Gynecol.*1955;6:567-589.
64. Friedman EA. *Labor: Clinical Evaluation and Management* (2nd ed.). New York, NY: Appleton-Century-Crofts; 1978
65. Zhang J, Troendle J, Mikolajczyk R, Sundaram R, Beaver J, Fraser W. The natural history of the normal first stage of labo *Obstet Gynecol.* 2010;115:705-710.
66. Zhang J, Landy H, Branch DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcome. *Obstet Gynecol*. 2010;116:1281-1287.
67. American College of Obstetricians and Gynecologists. Practice Bulletin No. 49: dystocia and augmentation of labor. *Obste Gynecol.* 2003;102:1445-1454.
68. Marowitz A. Caring for women in early labor: can we delay admission and meet women's needs? *J Midwifery Womens Health*. 2014;59:645-650.
69. Neal JL, Lowe, NK, Phillipi JC, et al. Likelihood of cesarean delivery after applying leading active labor diagnostic guidelines. *Birth*. 2017;44(2):128-136.
70. Greulich B, Tarrant B. The latent phase of labor: diagnosis and management. *J Midwifery Womens Health*. 2007;52:190-198.
71. Neal JL, Lowe NK, Ahijevych KL, Patrick TE, Cabbage LA, Corwin EJ. "Active labor" duration and dilation rates among low-risk, nulliparous women with spontaneous labor onset: a systematic review. *J Midwifery Womens Health*. 2010;55:308-318.
72. Lowe N, Corwin E. Proposed biological linkages between stress, obesity, and inefficient uterine contractility during labor in humans. *Med Hypothesis.* 2011;76:755-760.
73. Smyth RMD, Alldred SK, Markham C. Amniotomy for shortening spontaneous labour. *Cochrane Database Syst Rev.* 2013;1:CD006167. doi:10.1002/14651858.CD006167.pub3.
74. Wei S, Wo BL, Qi HP, et al. Early amniotomy and early oxytocin for prevention of, or therapy for, delay in first stage spontaneous labour compared with routine care. *Cochrane Database Syst Rev.* 2013;8:CD006794. doi:10.1002/14651858.CD006794.pub4.
75. National Institutes of Health. *Consensus Development Conference on Cesarean Childbirth*. Publication No. 82:2067. Bethesda, MD: National Institutes of Health, Department of Health and Human Services; 1981.
76. Guise JM, Denman MA, Emeis C, et al. Vaginal birth after cesarean: new insights on maternal and neonatal outcomes. *Obstet Gynecol.* 2010;115(6):1267-1278.
77. McMahon MJ, Luther ER, Bowes WA, Olshan AF. Comparison of a trial of labor with an elective second cesarean section. *N Engl J Med*. 1996;335(10): 689-695.
78. American College of Obstetricians and Gynecologists. *ACOG Practice Bulletin No. 5: Vaginal Birth After Previous Cesarean Delivery.* Washington, DC: American College of Obstetricians and Gynecologists; July 1999.
79. National Institutes of Health consensus development conference statement on vaginal birth after cesarean: new insights March 8–10, 2010. *Obstet Gynecol.* 2010;115(6):1279-1295.
80. American College of Nurse-Midwives. Caring for women desiring vaginal birth after cesarean: ACNM Clinical Bulletin No. 12. *J Midwifery Womens Health*. 2011;56:517-525.
81. Committee on Practice Bulletins—Obstetrics. Vaginal birth after previous cesarean delivery. Practice Bulletin No. 184. *Obstet Gynecol.* 2017;130:217-233.
82. Chiossi G, Lai Y, Landon MB, et al. Timing of delivery and adverse outcomes in term singleton repeat cesarean deliveries. *Obstet Gynecol.* 2013;121:561-569.
83. American College of Obstetricians and Gynecologists. Practice Bulletin No. 115: gestational diabetes mellitus. *Obstet Gynecol.* 2013;122(2 pt 1):406-416.
84. American College of Obstetricians and Gynecologists. Practice Bulletin No. 173: fetal macrosomia. *Obstet Gynecol.* 2016;128e:195-209.
85. American College of Obstetricians and Gynecologists. *Hypertension in Pregnancy.* Washington, DC: American College of Obstetricians and Gynecologists; 2013.
86. Bernstein PS, Martin JN Jr, Barton JR, et al. National Partnership for Maternal Safety: consensus bundle on severe

hypertension during pregnancy and the postpartum period. *Obstet Gynecol*. 2017;130(2):347-357.
87. Mhyre JM, D'Oria R, Hameed AB, et al. The maternal early warning criteria: a proposal from the National Partnership fo Maternal Safety. *Obstet Gynecol*. 2014;124(4):782-786.
88. Silver RM. Abnormal placentation. *Obstet Gynecol*. 2015;126:654-668.
89. Oyelese Y, Ananth CV. Placental abruption. *Obstet Gynecol*. 2006;108:1005-1016.
90. Hladky K, Yankowitz J, Hansen WF. Placental abruption. *Obstet Gynecol Surv*. 2002;57(5):299-305.
91. Tamura N, Farhana M, Oda T, Kanayama N. Amniotic fluid embolism: pathophysiology from the perspective of pathology. *J Obstet Gynaecol Res*. 2017;43(4):627-632.
92. Clark S, Romero R, Dildy G, et al. Proposed diagnostic criteria for the case definition of amniotic fluid embolism in research studies. *Am J Obstet Gynecol*. 2017;217(2):228-229.
93. Benson MF. Amniotic fluid embolism mortality rate. *J Obstet Gynaecol Res*. 2017;43(11):1714-1718.
94. Hansen A, Chauhan SP. Shoulder dystocia: definitions and incidence. *Semin Perinat*. 2014;38:184-188.
95. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 40: shoulder dystocia. *Obstet Gynecol*. 2002;100:1045-1050. [Reaffirmed 2015].
96. Mehta SH, Sokol RJ. Shoulder dystocia: risk factors, predictability and preventability. *Semin Perinat*. 2014;38:189-193.
97. Gurewitsch ED, Allen RH. Reducing the risk of shoulder dystocia and associated brachial plexus injury. *Obstet Gynecol Clin North Am*. 2011;38:247-269.
98. Inglis SR, Feier N, Chetiyaar JB, et al. Effects of shoulder dystocia training on the incidence of brachial plexus injury. *Am Obstet Gynecol*. 2011;204:322.e1-322.e6.
99. Hoffman MK, Bailit JL, Branch W, et al. A comparison of obstetric maneuvers for the acute management of shoulder dystocia. *Obstet Gynecol*. 2011;117:1272-1278.
100. Politi S, D'Emidio L, Cignini P, Giorlandino M, Giorlandino C. Shoulder dystocia: an evidence-based approach. *J Perinat Med*. 2010;4:35-42.
101. Kotaska A, Campbell K. Two-step delivery may avoid shoulder dystocia: head-to-body interval is less important than we think. *J Obstet Gynecol Can*. 2014;36:716-720.
102. Royal College of Obstetricians and Gynaecologists. *RCOG Green-Top Guideline No. 42*. London, UK: Royal College of Obstetricians and Gynaecologists; 2012.
103. Society of Obstetricians and Gynaecologists of Canada. Shoulder dystocia. In: *Advances in Labour and Risk Management Textbook*. *ALARM International Program Manual*. 22nd ed. Ottawa, Canada: Society of Obstetricians and Gynaecologists of Canada; 2016. Available at: https://alarm.cfpc.ca/wp-content/uploads/2015/05/22nd-Ed-Manual-Printable-Version.pdf. Accessed October 23, 2017.
104. Menticoglou S. Delivering shoulders and dealing with shoulder dystocia: should the standard of care change? *J Obstet Gynecol Can*. 2016;38:655-658.
105. Andrighetti TP, Knestrick JM, Marowitz A, Martin C, Enstrom JL. Shoulder dystocia and postpartum hemorrhage simulations: student confidence in managing these complications. *J Midwifery Womens Health*. 2012;57:55-60.
106. Crofts JF, Fox R, Ellis D, Winter C, Hinshaw K, Drawycott TJ. Observations from 450 shoulder dystocia simulations: lessons for skills training. *Obstet Gynecol*. 2008;112:906-912.
107. Fahey JO, Mighty HE. Shoulder dystocia: using simulation to train providers and teams. *J Perinat Neonat Nurs*. 2008;22:114-122.
108. Maran NT, Glavin RT. Low- to high-fidelity simulation: a continuum of medical education? *Med Educ*. 2003;37(suppl 1):22-28.
109. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 169: multifetal gestations: complicated twin, triplet, and higher order multifetal pregnancies. *Obstet Gynecol*. 2016;128:926-928.
110. Lee YM. Delivery of twins. *Semin Perinatol*. 2012;36:195-200.
111. Monson M, Silver RM. Multifetal gestation: mode of delivery. *Clin Obstet Gynecol*. 2015;58:690-702.
112. Hannah ME, Hannah WJ, Hewson SA, Hodnett ED, Saigal S, Willan AR. Planned cesarean section versus planned vaginal birth for breech presentation at term: a randomised multicentre trial. Term Breech Trial Collaborative Group. *Lancet*. 2000;356:1375-1383.
113. Lawson GW. The Term Breech Trial ten years on: primun non nocere? *Birth*. 2012;39:3-9.
114. American College of Obstetricians and Gynecologists. Committee Opinion No. 340: mode of term singleton breech delivery. *Obstet Gynecol*. 2006;108:235-237.
115. Hofmeyr GJ, Hannah M, Lawrie TA. Planned caesarean section for term breech delivery. *Cochrane Database Syst Rev*. 2015;7:CD000166. doi:10.1002/14651858.CD000166.pub2.
116. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 102: management of stillbirth. *Obstet Gynecol*. 2009;113:748-761.
117. Maslow AD, Breen TW, Sarna MC, Soni AK, Watkins J, Oriol NE. Prevalence of coagulation abnormalities associated with intrauterine fetal death. *Can J Anaesth*. 1996;43:1237-1243.
118. Kobler K, Limbo R. Making a case: creating a perinatal palliative care service using a perinatal bereavement model. *J*

Perinat Neonat Nurs. 2011;25:32-41.

APPENDIX

29A

Shoulder Dystocia

AMY MAROWITZ

The editors acknowledge Linda A. Hunter, who was the author of this appendix in the previous edition.

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Shoulder dystocia is an obstetric emergency that most midwives will inevitably encounter in clinical practice. Management is focused on systematic steps that are designed to minimize adverse outcomes, especially injury to the infant's brachial plexus. This appendix reviews the key points and management steps required for safe intrapartum practice in the event of a shoulder dystocia.

Clinically, signs of an impending shoulder dystocia occur while the woman is pushing as the infant's head slowly extends and emerges over the perineum. Instead of fully extending, the vertex partially retracts back into the vagina, a phenomenon commonly referred to as the "turtle sign" (**Figure 29A-1**). Restitution and external rotation do not occur, as the infant's shoulders have not followed the normal cardinal movements through the pelvis and are impacted in an anterior–posterior position. In cases of maternal obesity, it may appear as if the "turtle sign" is occurring when there is simply excess maternal soft tissue that impedes the extension and rotation of the infant's head. Abducting the hips, called McRoberts' maneuver, causes the symphysis to shift up over the anterior shoulder of the newborn, which usually allows enough room for normal birth mechanisms to occur (**Figure 29A-2**).



Figure 29A-1 Turtle sign. Note that the infant's chin cannot be seen because the head has retracted into the vagina.

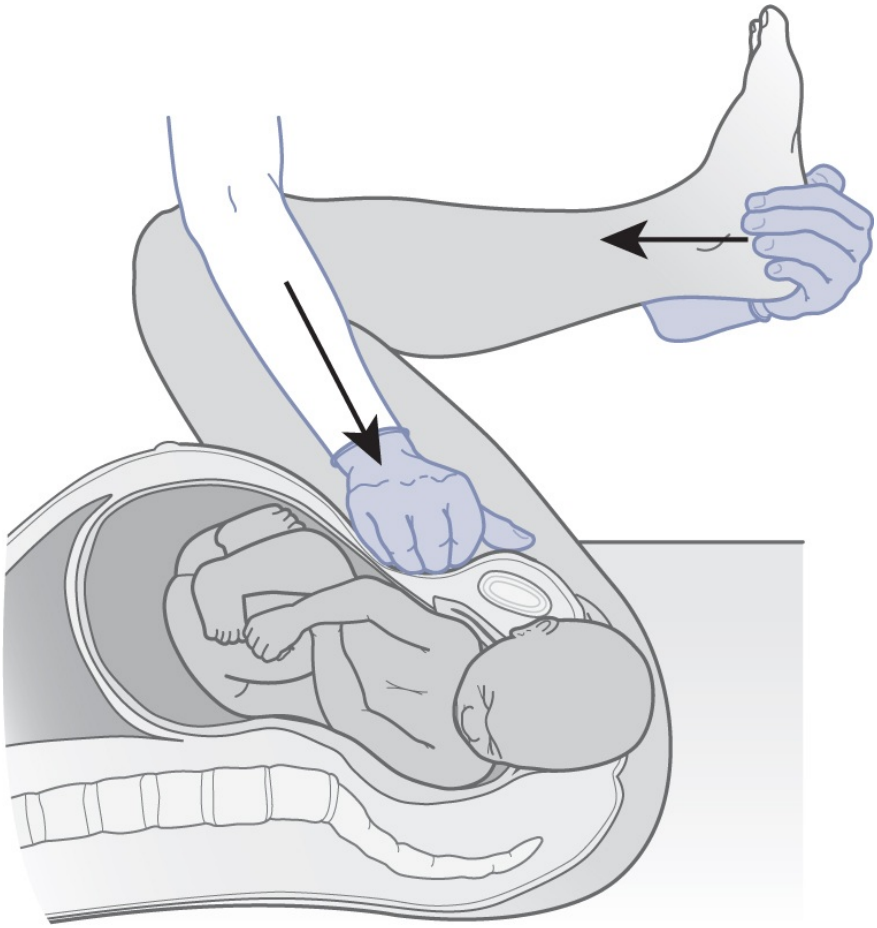


Figure 29A-2 McRoberts' maneuver and suprapubic pressure.

While accurate diagnosis of a shoulder dystocia is critical, it is equally important for the midwife to remain calm and fully assess the situation. After the head is delivered, waiting for the next contraction to allow for normal restitution and allowing the woman to push the infant out of the vagina unassisted may reduce the incidence of shoulder dystocia. If restitution and external rotation do not occur, it is equally important to first assess the location of the shoulders prior to initiating any maneuvers (other than repositioning the mother). Midwives who are attending births at home or in a birth center may need to enlist the assistance of the woman's partner and/or family if a shoulder dystocia is encountered. Even if the dystocia is resolved quickly, resuscitation assistance and ambulance transfer to the nearest hospital may still be needed for both the woman and her newborn.

After a shoulder dystocia occurs, a carefully written delivery note documenting the details of the birth and the sequence of events/maneuvers should be completed. Some institutions have specific delivery note templates or forms that are used following a shoulder dystocia. At a minimum, this note should include the position of the head at birth, which shoulder was anterior, time of the birth of the head, who was present or called, maneuvers that were done and in which order, time of the delivery of the body, newborn weight and Apgar scores, cord blood gas results if obtained, disposition of the newborn, and any motor deficit in the upper extremities noted on initial newborn examination; any maternal injuries should also be identified. In addition, using the term "traction" or trying to quantify the amount of force should

be avoided. From a medico-legal standpoint, it should be noted that fundal pressure was *not* used. The events should be explained clearly and simply with no attempts to editorialize or speculate.

Shoulder dystocia is a traumatizing event for all involved. It may be helpful for the midwife to organize a debriefing with the other staff and providers who were present as soon as possible to discuss what happened in a nonthreatening, supportive way. The midwife should also stay in close contact with the woman and her support persons. In the event of a serious shoulder dystocia, they may be facing devastating neonatal consequences and need ongoing support and follow-up. It is also essential that the midwife provides honest and accurate information to the woman regarding what happened during the birth without any blame or judgment.

Definitions of Shoulder Dystocia[1-4]

1. Impaction of the anterior shoulder behind the pubic bone
2. Impaction of the posterior shoulder on the sacral promontory
3. Prolonged head-to-body delivery time greater than 60 seconds
4. Need for ancillary maneuvers to deliver the shoulders

Incidence[1,2]

1. Varies in different studies due to variations in research design, lack of consensus on its definition, and differences in populations studied.
2. Contemporary review of literature: Incidence is approximately 1.4% of all births.
3. May be underreported due to variations in study design, definition, and population.
4. Incidence rises with increasing fetal weight.
5. Incidence is highest in women who have diabetes and a macrosomic fetus.

Risk Factors[5]

1. Prenatal risk factors
    a. Fetal macrosomia (more than 4000 grams)
    b. Maternal diabetes
    c. History of shoulder dystocia in a previous pregnancy
2. Intrapartum risk factors
    a. Precipitous second stage
        i. Rapid descent that does not allow enough time for normal rotational maneuvers
    b. Operative vaginal delivery (forceps or vacuum extraction)
    c. Prolonged second stage (increases the risk for operative delivery)

3. Associated risks (all of which increase incidence of macrosomia)
    a. Obesity
    b. Excessive weight gain
    c. Late term and post-term gestation

Key Points

1. Stay calm and clearly communicate to the woman and the team.
2. Do not panic and do not rush.
3. Be systematic and deliberate in moving through the procedures.
4. Keep hands off the infant's head as much as possible and minimize traction on the head.
5. These maneuvers are done when the woman is *not* pushing.
6. Do *not* let anyone perform suprapubic pressure until asked for by the person attending the birth.
7. Under *no* circumstances should anyone *ever* perform fundal pressure.
8. Try a maneuver once and then move on to another one; spend no more than 30 seconds on each maneuver.
9. Keep the team informed as each maneuver is performed.
10. Cut an episiotomy *only* if unable to get an entire hand into the vagina.
11. Start an intravenous line and obtain blood for a type and screen if this has not been done.
12. Continuously monitor the FHR and administer oxygen to the woman if needed.
13. Do not clamp or cut a nuchal cord during a shoulder dystocia; plan to deliver the infant with the somersault maneuver once the dystocia is resolved (see the *Hand Maneuvers for Birth* appendix in the *Second Stage of Labor and Birth* chapter).
14. Be prepared for a postpartum hemorrhage after the infant is born.
15. Be prepared for a full neonatal resuscitation. Cord blood gases should be sent if the shoulder dystocia was prolonged or if there is any sign of neonatal distress after the birth.
16. Neonatal asphyxia becomes a significant risk by 5 minutes. Rescue maneuvers (cephalic replacement or abdominal rescue) should be considered by 4 minutes. These will require transfer to the operating room and the availability of an obstetrician.

Management Steps

There is no clear superiority in terms of order of release maneuvers. Use of McRoberts' maneuver as a first step for women who give birth in a supine position is commonly recommended.[2-4] Steps 4, 5, 7, 8, 12, and 13 describe individual release maneuvers. The circumstances and midwife's judgment should guide the order of maneuvers when a shoulder dystocia occurs.

1. The moment that shoulder dystocia is suspected, have the woman *stop* pushing and

immediately assess the position of the fetal shoulders with a vaginal examination.

2. If the anterior shoulder is impacted, calmly and clearly state, "I have a shoulder dystocia."
3. Request help—additional nurses, the consulting physician, the neonatal team, and anesthesia should be called immediately.
4. Have the woman move to the edge of the bed and then into *McRoberts' position* (head down and knees/hips hyperflexed toward abdomen; **Figure 29A-3**). Hyperflexing the legs does not increase pelvic diameters but causes cephalic rotation of the symphysis so the symphysis slides up and over the anterior shoulder. Tell the woman not to push.

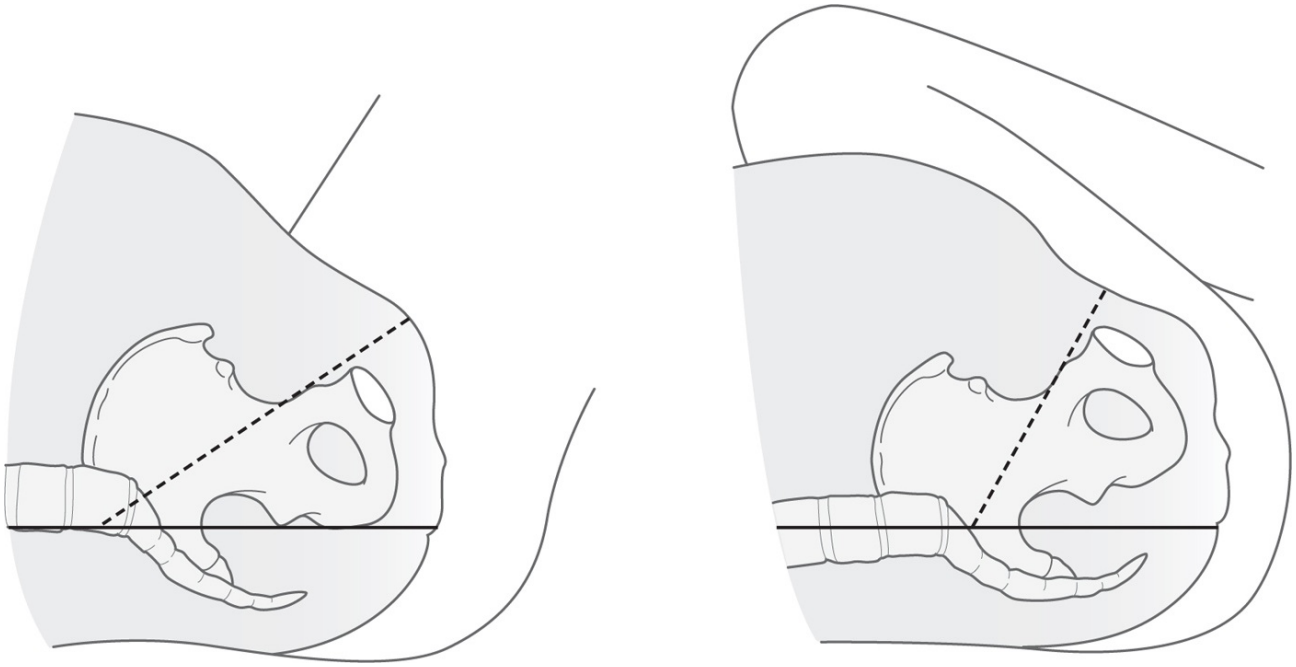


Figure 29A-3 Effect of McRoberts' maneuver on the symphysis. Notice that adduction of the legs toward the woman's abdomen moves the symphysis up so it slides over the fetal anterior shoulder.

5. Direct another person to perform *suprapubic pressure* at a slight angle in the direction you are trying to rotate the shoulder toward the fetal chest. Do this in between contractions first.
6. *The combination of McRoberts' maneuver and suprapubic pressure* will likely relieve the majority of shoulder dystocias. If it is not working, the fetus may be too large to fit in this alignment and will need rotational maneuvers.
7. *Rubin's maneuver*. Insert a hand (or as many fingers as possible) behind the anterior shoulder on the fetal back, and try to rotate the shoulder forward into an oblique position in the pelvis. This is also referred to as "collapsing the shoulders" and may help reduce the bisacromial shoulder diameter. The woman can attempt to push in coordination with your hand rotating the shoulder and the directed suprapubic pressure by an assistant. Use minimal and very gentle lateral traction (on the fetal shoulder—not the fetal head or neck). If this does not easily relieve the dystocia, repeated attempts will increase the force and the risk of brachial plexus injury and should not be continued.

8. *Delivery of the posterior arm.* Insert an entire hand in the posterior aspect of the vagina and follow the posterior arm to the infant's elbow. If the arm is extended, try to reach the forearm and bend the arm at the elbow until you can feel the infant's hand. Either grasp the hand or put gentle forward pressure on the elbow to sweep the arm across the chest and out of the vagina. There is an increased risk of humerus or clavicle fracture, but in the majority of cases, delivery of the posterior arm will relieve the dystocia. Because this maneuver is associated with the lowest risk of brachial plexus injury, some providers choose to try it before trying Rubin's maneuver.
9. Move gently, deliberately, and with purpose. Imagine you are inserting your hand into a circular can of potato chips or through a tight bangle bracelet. Continue to let the team know what you are doing (i.e., "I am going to try to deliver the posterior arm").
10. Cut an episiotomy *only* if more room is needed to insert your hand.
11. Continue to coach the woman not to push and keep her informed—her continued cooperation is critical.
12. *Gaskin maneuver*. Have the woman move onto her hands and knees. If the dystocia is not resolved with repositioning, attempt to deliver the posterior arm. If unsuccessful, rotational maneuvers can be attempted in this position or resumption of a supine McRoberts' position may be preferable
13. *Rotational maneuver.* With a hand firmly against either the posterior or anterior shoulder, push the shoulder clockwise or counterclockwise in an attempt to rotate it in a 180-degree arc. Continue to rotate the shoulders back and forth in whichever direction they will move until the impaction is dislodged and the infant is born. This maneuver may require both hands (one anterior and one posterior) pushing in opposite directions (**Figure 29A-4**).

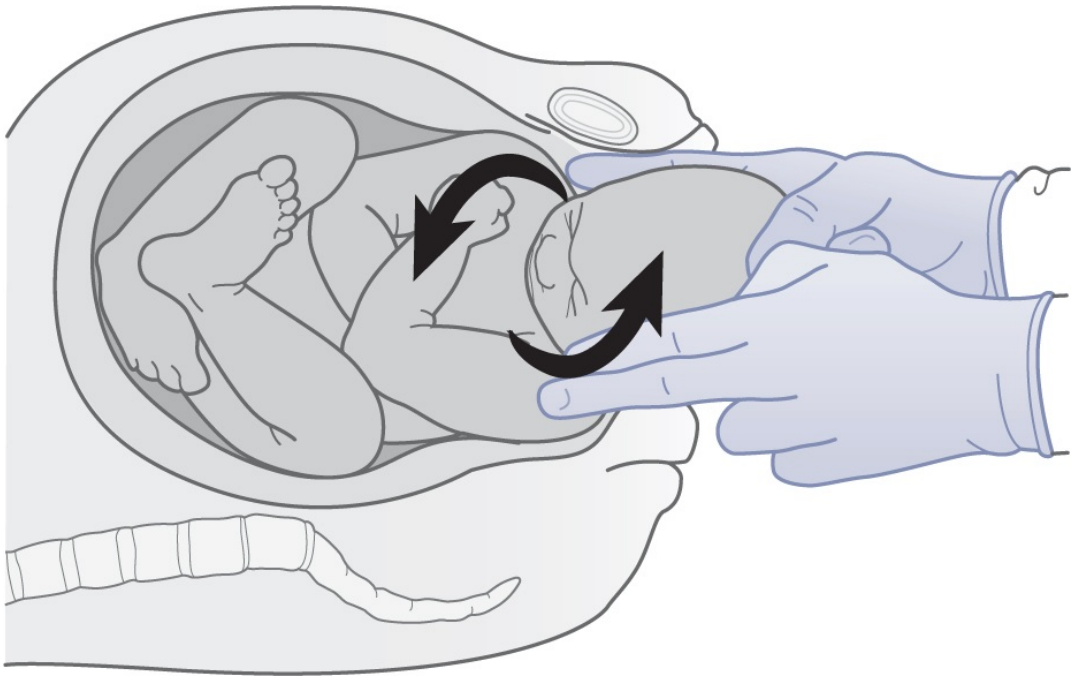


Figure 29A-4 Woods' screw (rotational) maneuver.

14. When the consulting physician or another provider enters the room, step aside and let that person try to facilitate the birth.
15. Intentional fracture of the clavicle may decrease the bisacromial diameter but may be difficult to perform in an emergent situation.
16. By 5 minutes, the risk of neonatal asphyxia starts to rise and rescue maneuvers must be considered. This will necessitate transfer to the physician and the availability of anesthesia and a full operating room team.
17. *Rescue maneuvers* include cephalic replacement (Zavanelli maneuver) or abdominal rescue, which both require a full operating room team and a physician capable of performing a cesarean delivery.

References

1. Hansen A, Chauhan SP. Shoulder dystocia: definitions and incidence. *Semin Perinat.* 2014;38:184-188.
2. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 40: shoulder dystocia. *Obstet Gynecol.* 2002;100:1045-1050. [Reaffirmed 2015].
3. Royal College of Obstetricians and Gynaecologists. *RCOG Green-Top Guideline No. 42*. London, UK: Royal College of Obstetricians and Gynaecologists; 2012.
4. Society of Obstetricians and Gynaecologists of Canada. Shoulder dystocia. In: *Advances in Labour and Risk Management Textbook. ALARM International Program Manual.* 22nd ed. Ottawa, Canada: Society of Obstetricians and Gynaecologists of Canada; 2016. Available at: https://alarm.cfpc.ca/wp-content/uploads/2015/05/22nd-Ed-Manual-Printable-Version.pdf. Accessed October 23, 2017.
5. Mehta SH, Sokol RJ. Shoulder dystocia: risk factors, predictability and preventability. *Semin Perinat.* 2014;38:189-193.

APPENDIX

29B

Emergency Interventions for Umbilical Cord Prolapse

AMY MAROWITZ

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An overt umbilical cord prolapse is an obstetric emergency that will require the woman to undergo an immediate cesarean section. Often the first sign of a cord prolapse is a sudden fetal bradycardia or recurrent variable decelerations that rapidly get more severe. Management of this acute situation requires prompt action on the part of the midwife in recognizing the prolapse and performing the appropriate lifesaving interventions while an operating room team and the consulting physician are mobilized. **Figure 29B-1** depicts the types of umbilical cord prolapse.

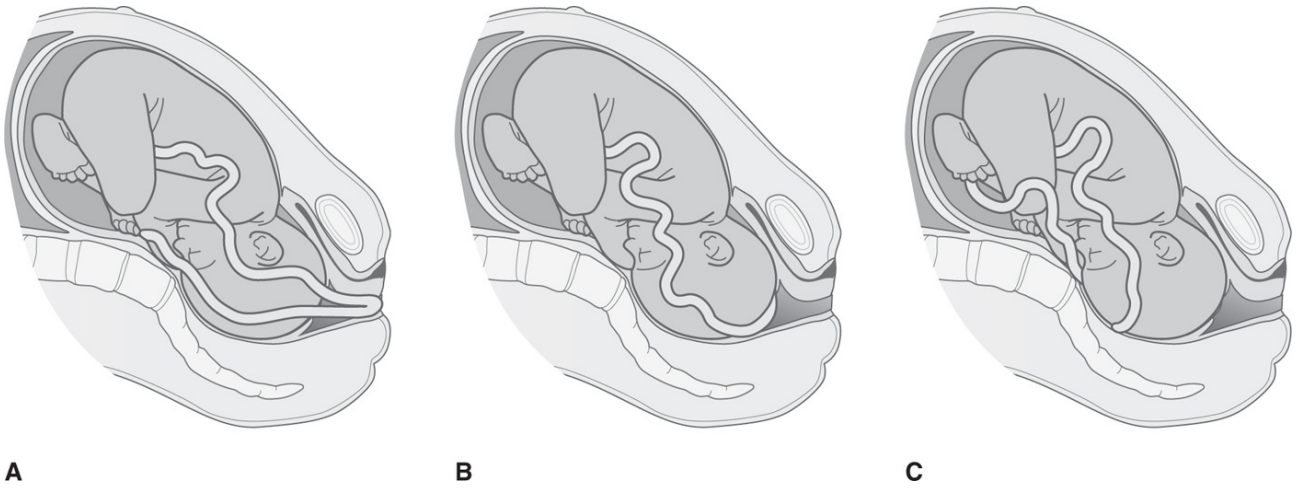


Figure 29B-1 Umbilical cord prolapse. **A.** Complete cord prolapse. **B.** Cord prolapsed in front of the fetal head. **C.** Occult (hidden) prolapse.

Definitions

1. *Overt:* The cord slips completely out of the cervix and is protruding in the vagina.
2. *Occult:* The cord is compressed alongside the presenting part in the lower uterine segment or inner cervix.
   a. Maternal repositioning or gentle upward pressure on the fetal head during a cervical examination can relieve an occult cord prolapse.
   b. If fetal heart rate (FHR) decelerations are present, intrauterine resuscitation measures should be initiated (see the *Fetal Assessment During Labor* chapter).
3. *Funic:* The cord is palpable in front of the presenting part.
   a. A funic presentation requires consultation with the collaborating physician to develop the most appropriate management plan.

Risk Factors

1. Prematurity
2. Multifetal gestation
3. Multiparity
4. Polyhydramnios
5. Fetal malpresentation
6. Rupture of membranes: intentional or spontaneous with an unengaged head

Management Steps

1. Call for help immediately and do not leave the woman.
2. Clearly state, “I have a prolapsed umbilical cord.”
3. Direct others to page a physician and notify the operating room team.
4. Inform the woman of what is happening and tell her she will need an emergency cesarean section.
5. Reposition the woman in the bed, tipped on her side (lateral Sims), or place her in knee–chest position.
6. Do *not* attempt to replace the cord in the uterus.
7. Put a sterile glove on and get into or on the bed, insert a hand into the vagina and firmly push up on the presenting part to relieve any compression on the cord. *Do not remove your hand or let up on the presenting part*.
8. Monitor the FHR continuously.
9. Administer oxygen and an intravenous fluid bolus.
10. Be prepared to accompany the woman into the operating room in this position riding on the bed.
11. The operating room staff will place the sterile drapes over you, and the physician will

let you know when the newborn is safely out of harm's way and you can remove your hand.

APPENDIX

29C

Intrapartum Management of Twin Gestation

AMY MAROWITZ

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Midwifery management of a planned twin birth should always occur in a hospital in close collaboration with a physician capable of performing a cesarean section. Many institutions require vaginal delivery of twins to occur in the operating room with anesthesia and an operating room team on standby. The neonatal (or pediatric) team should also be notified as soon as possible of a twin labor admission to ensure the presence of adequate nursing staff and personnel capable of full neonatal resuscitation of both newborns. Regardless of how the fetuses were identified during pregnancy, the first twin to deliver is referred to as "Baby A" and the second twin is "Baby B."

Incidence

1. 3% of all births
2. 40% will be vertex–vertex at onset of labor[1]

Types of Twins[1]

1. Monochorionic–monoamniotic (mono-mono)
   a. Requires referral for medical management
   b. Delivery by cesarean section typically at 32 weeks[1]
2. Monochorionic–diamniotic (mono-di)

3. Dichorionic–diamnionic (di-di)

Risks Associated with Twin Deliveries[1]

1. Preterm birth
2. Malpresentation
3. Prolapsed umbilical cord
4. Placental separation (especially with Twin B)
5. Postpartum hemorrhage
6. Preeclampsia
7. Placenta previa

Intrapartum Management Guidelines for Twin Gestations

1. Upon arrival in labor, the presentation and lie of each fetus should be reassessed with ultrasound.
2. These findings should be reviewed with the consulting physician to determine whether the planned vaginal delivery is still an option. This plan should be shared with the woman and her informed consent obtained.
3. The woman should have an intravenous line inserted with blood drawn for a blood type, antibody screen, and cross-match.
4. Management of labor pain is no different for twins, including use of epidural anesthesia if the woman desires. Epidural analgesia is recommended in many institutions as it can provide anesthesia for an emergency cesarean section if needed.
5. Continuous fetal monitoring of both fetuses is required throughout labor with an electronic fetal monitor that can record both heart rates simultaneously. A fetal scalp electrode should be inserted on Baby A only if medically indicated.
6. The consulting physician should be in the hospital and present in the room during the births. It is generally recommended that the births occur in an operating room as there is a significant risk of needing an emergency cesarean section for Baby B.
7. The management of Baby A's birth is no different than a singleton gestation. An episiotomy should be done only if medically indicated.
8. The umbilical cord from Baby A should be clamped and marked so that it is clear that this cord is from Baby A. For example, use one extra clamp for Baby A and two extra clamps for Baby B.
9. After the birth of Baby A, Baby B's position and presentation should be immediately assessed with ultrasound and the woman's cervix checked for dilation and station of the presenting part.
10. Baby B's fetal heart rate should be monitored continuously, as this is the period of risk

for umbilical cord prolapse.

11. If Baby B is presenting vertex, care should be taken when rupturing the membranes to avoid a prolapsed umbilical cord. Do not rupture the membranes if Baby B is unengaged.
12. If Baby B is not vertex, the physician should take over the management, especially if a breech extraction is anticipated.
13. A postpartum hemorrhage should be anticipated and the appropriate uterotonic medications and/or blood products should be immediately available.
14. The umbilical cords should be carefully marked to differentiate Baby A from Baby B and the placenta(s) sent to pathology.

Reference

1. Lee YM. Delivery of twins. *Semin Perinatol.* 2012;36:195-200.

APPENDIX

29D

Breech Birth

AMY MAROWITZ

The editors acknowledge Linda A. Hunter, who was the author of this appendix in the previous edition.

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As discussed in the chapter, there may be circumstances when a woman with a known breech presentation is planning a vaginal delivery. Planned vaginal breech births should always take place within a hospital setting under the primary management of an obstetrician. Midwives in clinical practice are more likely to encounter a woman in advanced labor with a previously unknown breech presentation. While every attempt should be made to mobilize a team to perform a cesarean section, if the birth is imminent, it will occur vaginally. This appendix reviews the management techniques for a breech birth, and midwives are encouraged to periodically review these steps. As with other obstetric emergencies, participation in vaginal breech simulation drills has been shown to improve provider confidence and team communication. If available in their practice settings, midwives are encouraged to participate in these types of training experiences.

Incidence of Breech Presentation

Of all pregnancies, 3% to 4% at term.[1]

Types of Breech Presentations[1]

See **Figure 29D-1**.

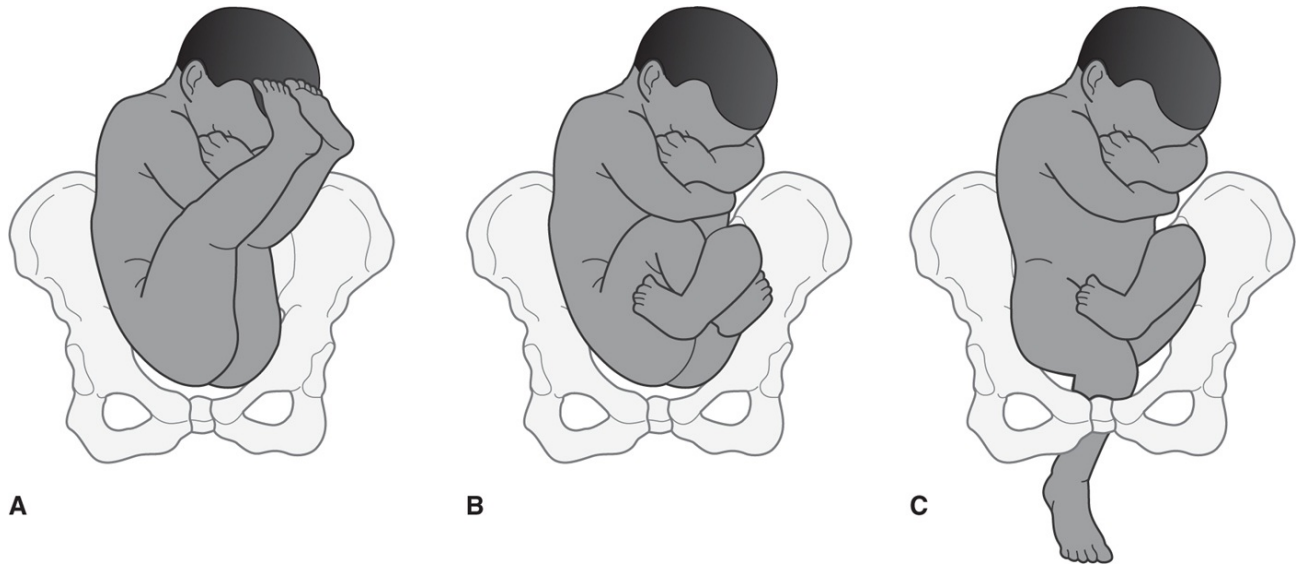


Figure 29D-1 Types of breech presentation. **A.** Frank breech. **B.** Complete breech. **C.** Footling (incomplete) breech.

1. *Frank breech (50–70%).* The lower extremities are flexed at the hips, with the legs extended and the feet near the infant's head ("pike" position). The presenting part is the infant's buttocks.
2. *Complete breech (5–10%).* Both the hips and knees are flexed, with both feet above the infant's buttocks ("cannonball" position). The presenting part is the infant's buttocks.
3. *Footling breech (10–30%).* One or both feet extend below the infant's buttocks and present in the vagina. This can be either a single or double footling presentation. A single footling is sometimes referred to as an "incomplete breech."

Predisposing Factors[2]

1. Preterm gestation
2. Multifetal gestation
3. Multiparity with lax uterine tone
4. Polyhydramnios
5. Oligohydramnios
6. Uterine anomalies (bicornuate uterus)
7. Uterine fibroids (especially in the lower uterine segment)
8. Placenta previa
9. Fetal anomalies (hydrocephaly, aneuploidy, nuchal masses)

Factors That May Improve the Success of Vaginal Breech Birth[3,4]

1. Average-size fetus (2500–3800 grams) with no known anomalies
2. Proven adequate pelvis
3. Frank or complete breech
4. Documentation of fetal head flexion (by ultrasound)
5. Spontaneous labor with normal progress
6. Easy spontaneous delivery of the buttocks and thighs

Key Points

1. Leopold's maneuvers are an important assessment tool but may be unreliable in determining presentation during active labor.
2. When performing a cervical examination on a woman in labor, palpation of the fetal skull sutures confirms a vertex presentation.
3. *Any* suspicion for breech presentation should always be confirmed with ultrasound.
4. In out-of-hospital sites, the midwife must rely on Leopold's maneuvers and a careful vaginal examination to palpate the fetal skull sutures. There should be no hesitation to transfer the woman to a hospital if a breech presentation is suspected. The midwife should also call 911 for additional help and notify the consulting physician, especially if the woman refuses transfer or the birth is imminent.
5. The infant's sacrum is the denominator (anatomic landmark for determining the position in the pelvis).
6. When a breech birth is imminent, the midwife should request the assistance of any available obstetrician (or a more experienced midwife if a physician is not available). The neonatal or pediatric team should also be called and staff prepared for full infant resuscitation. Anesthesia and an operating room team should be on standby in the event that surgical intervention is needed.
7. During the birth, the infant may pass thick meconium stool from compression of the breech in the vagina. This is often the first indication during labor of a "missed breech" presentation.
8. The fetal heart rate should be monitored continuously. The woman should also have an intravenous line inserted if possible and should be given oxygen if any signs of fetal acidemia are present.
9. The key management steps in a breech birth require a "hands-off" approach until the breech is delivered to the level of the umbilicus and considerable patience to allow for spontaneous progression of the normal cardinal movements. The woman should be allowed to push and her progress monitored closely.
10. If the woman has any contraindications to a vaginal breech birth, transfer to a hospital or operating room and preparation for a cesarean section is indicated. If contraindications are present, the midwife should assist with a vaginal birth only if the birth occurs before a cesarean section can be facilitated.

Cardinal Movements and Management Techniques for a Breech Birth

1. Descent occurs throughout and is facilitated by maternal pushing efforts.
2. Engagement of the hips usually takes place when the infant's bitrochanteric diameter (the distance between the outer points of the hips) enters the oblique diameter of the maternal pelvis. The position will then be either right sacrum anterior (RSA) or left sacrum anterior (LSA).
3. The anterior hip usually descends more rapidly than the posterior hip.
4. As resistance from the pelvic floor is encountered, internal rotation of 45 degrees will occur and the anterior hip will pass under the pubic bone. The bitrochanteric diameter of the hips is now in the anterior–posterior diameter of the woman's pelvis in either a right sacrum transverse (RST) or left sacrum transverse (LST) position.
5. As descent continues, the anterior hip provides a pivot for the posterior hip, which usually crosses the perineum first. Delivery of the anterior hip will follow through lateral flexion of the spine.
6. Until this point in time, the woman should be allowed to push as needed and descent of the breech observed closely. Resist the temptation to pull the buttocks out and allow the maternal pushing efforts to continue as in any normally progressing second stage. If the infant is born too quickly, the head may not have time to flex, which is necessary for birth of the head.
7. Once the buttocks are born and the umbilicus is visible at the perineum, the legs should be gently released one at a time through the vaginal opening. This should be done in between pushing efforts by following the thigh to the knee and sweeping the flexed leg across the infant's abdomen.
8. If the infant is in a frank breech presentation and the full extension of the legs has hindered descent, *Pinard's maneuver* can be used to "break up the breech" (**Figure 29D-2**). Insert either the right or left hand (depending on the sacrum's position) and follow the thigh to the popliteal fossa behind the knee. The thumb should be on the anterior side of the thigh. Gently press on the popliteal fossa and move the leg laterally away from the midline. This will cause the leg to flex at the knee, thereby bringing the foot down to where it can be grasped. Bring the leg down and out of the vagina by drawing it across the infant's abdomen in its natural range of motion. Repeat the maneuver for the other leg if needed.

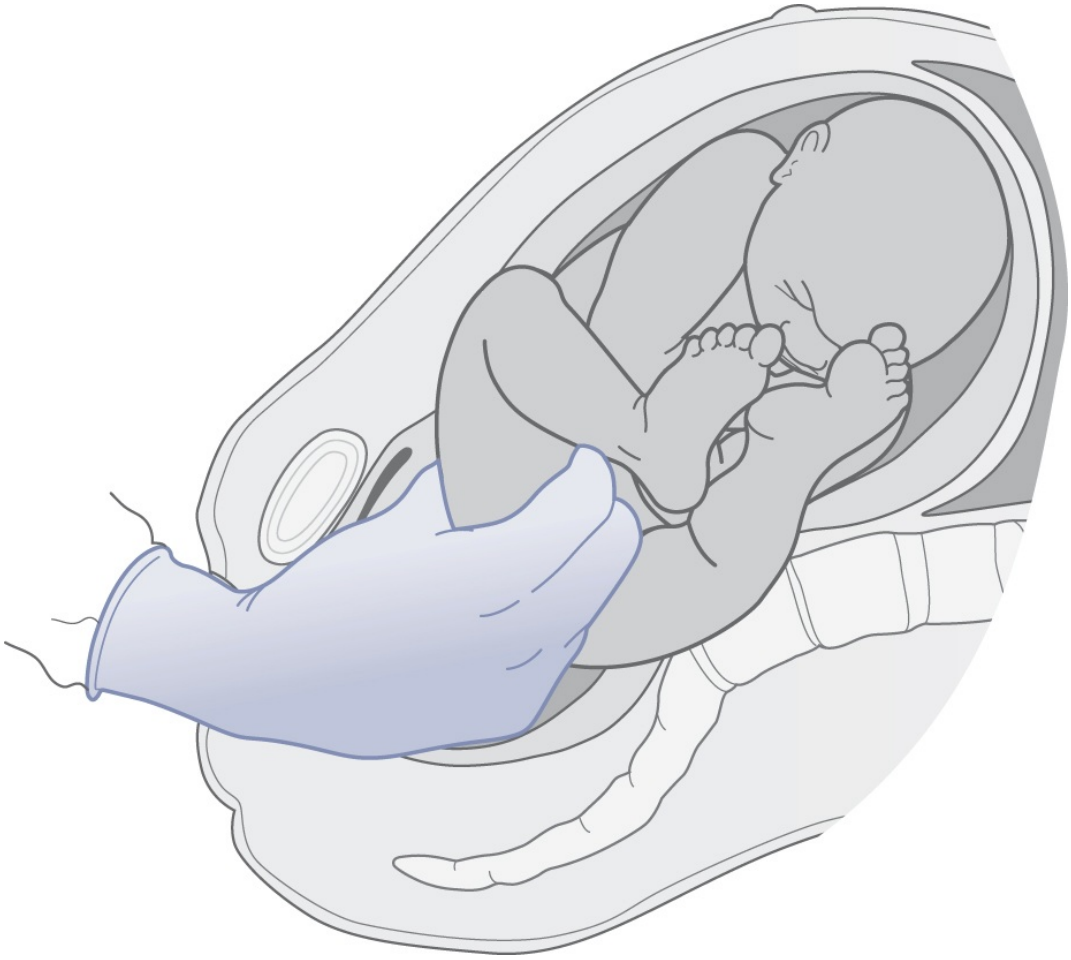


Figure 29D-2 Pinard's maneuver.

9. After the trunk, legs, and feet are delivered, external rotation will occur, aligning the infant's back into a sacrum anterior position. This will cause the shoulders to rotate internally into the oblique diameter of the maternal pelvis and pass under the pubic bone.
10. The remainder of the birth should occur within the next 3 to 5 minutes to avoid hypoxia from compression of the umbilical cord. A generous loop of the umbilical cord can be pulled down to prevent stress at the umbilical insertion site.
11. Wrap a warm towel around the infant's lower body just below the umbilicus. This will keep the infant warm and provide a nonslippery hold on the hips.
12. To facilitate birth of the shoulders, grasp the hips by placing the thumbs on the sacrum and the fingers on the corresponding iliac crests (**Figure 29D-3**). Avoid any pressure on the soft-tissue areas of the abdomen or flank.

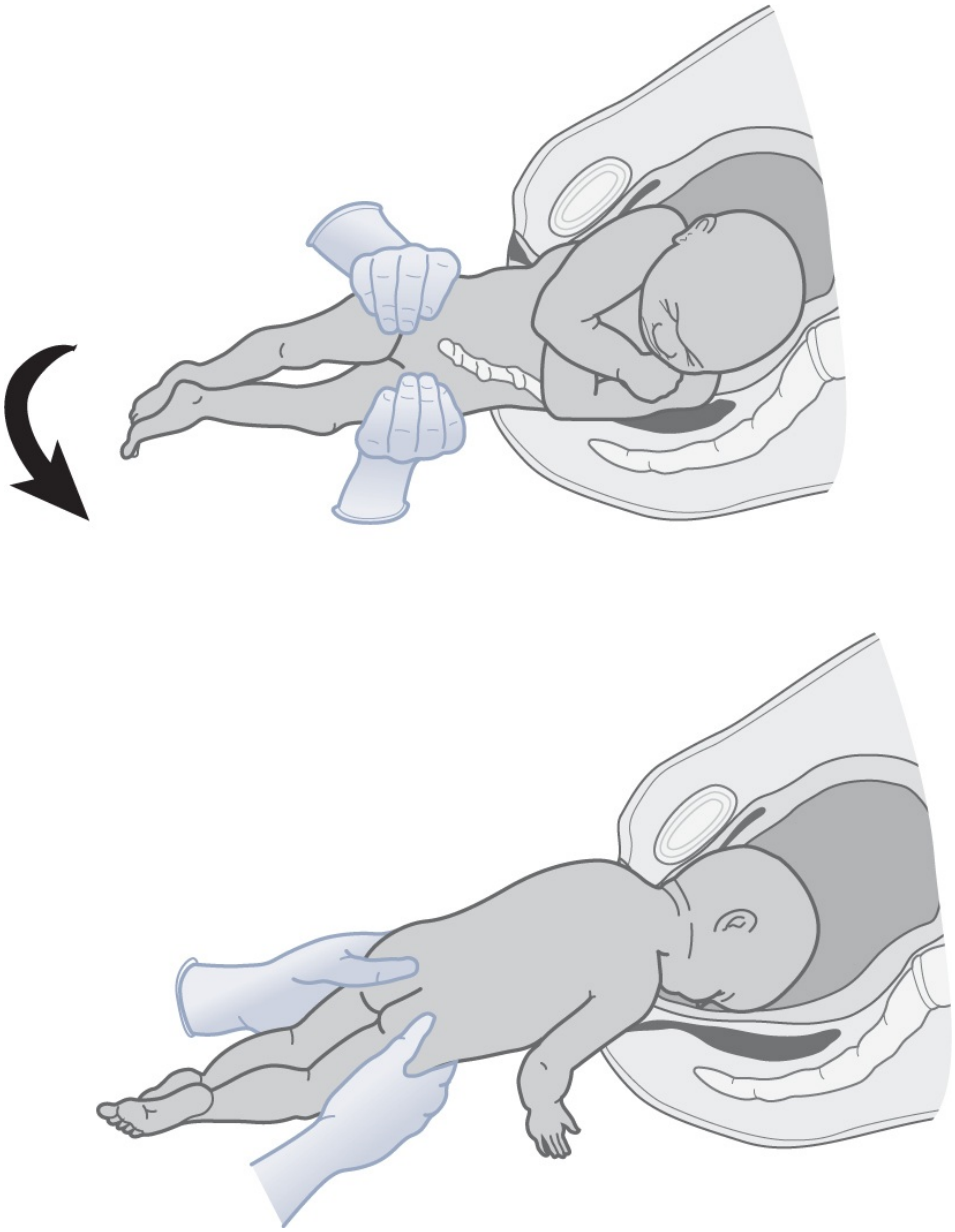


Figure 29D-3 The midwife's hands placed on the infant's lower body with thumbs over the sacral iliac joint.

13. Provide downward and outward traction on the body with coordinated pushing from the mother, and gently rotate the infant to either an RST or LST position (**Figure 29D-4**).

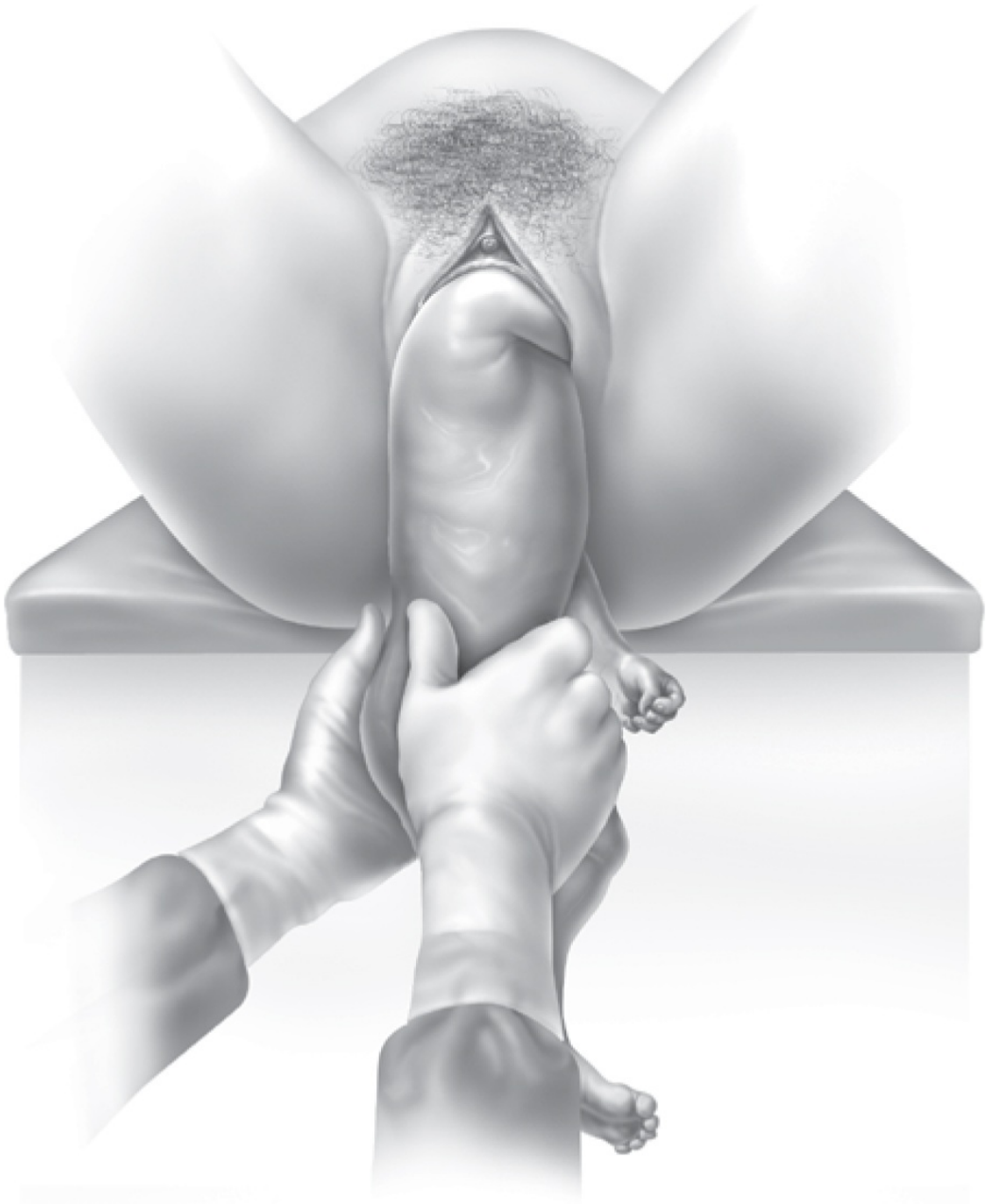


Figure 29D-4 Birth of the anterior shoulder by downward traction. Note the placement of the midwife's hands on the infant's hips.

14. Continue downward traction with maternal pushing efforts until both the scapula and the axilla of the anterior shoulder are visible.
15. *Delivery of shoulders and arms*. There are different techniques for delivery of the shoulders and arms, and it does not matter which shoulder is delivered first. Typically, the anterior shoulder will appear as a result of the downward traction placed on the body. Once the axilla is visible, the arm can then be delivered by reaching in to the elbow and sweeping the arm up across the chest. Once the anterior arm is out, the infant should be rotated 180 degrees, keeping the back up. This will bring the posterior scapula and axilla into view anteriorly, and this arm can be delivered in the same manner.

16. *Delivery of the posterior arm.* If the anterior shoulder does not appear with downward traction, the posterior arm can be delivered first. Grasp the feet securely in one hand and lift the body up and over toward the abdominal side until the scapula and axilla are visible posteriorly at the perineum. Insert the other hand along the upper arm to the elbow and sweep the arm across the chest. Now the infant can be either repositioned with downward traction again or rotated until the anterior shoulder and axilla are in view (**Figure 29D-5**).

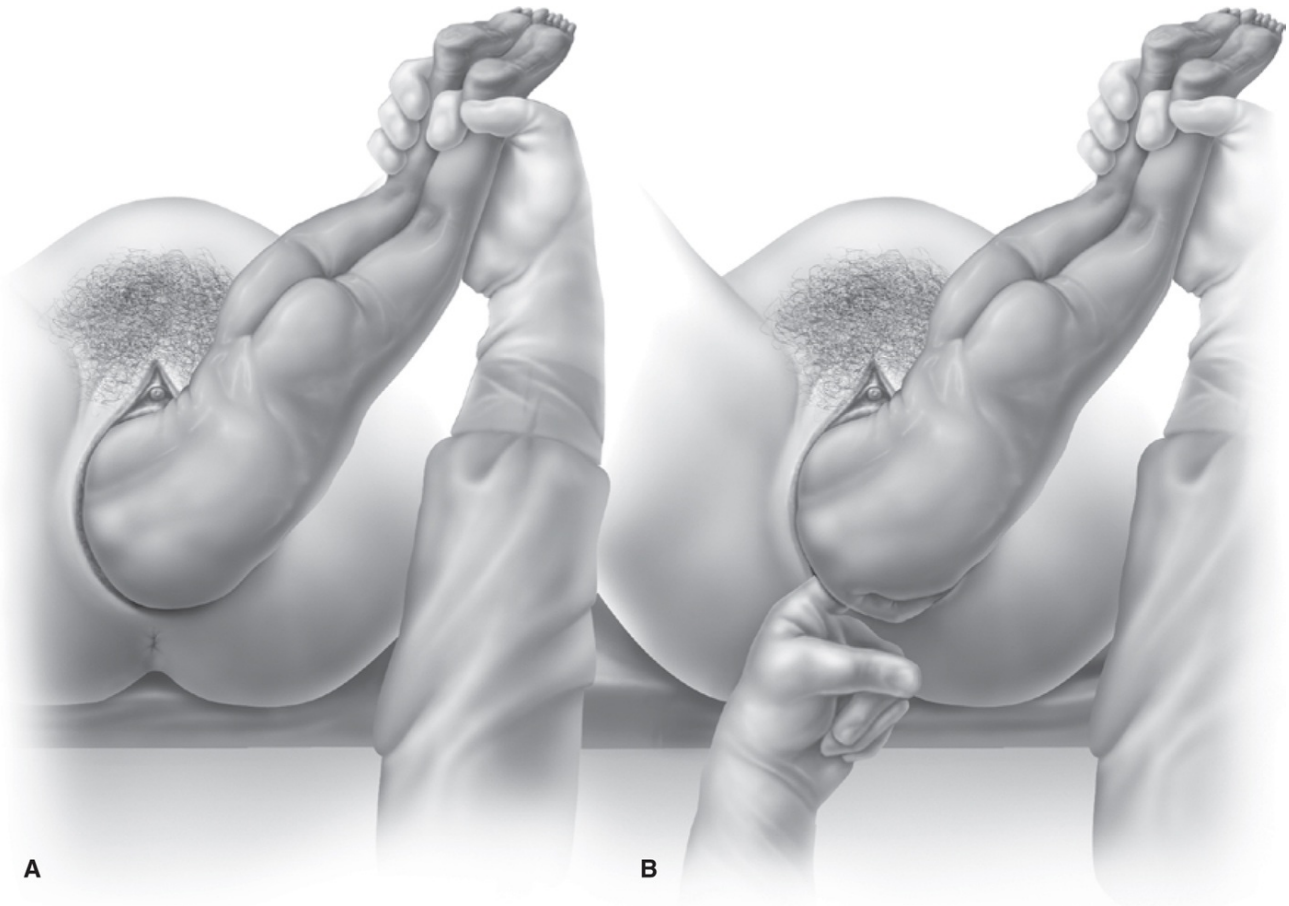


Figure 29D-5 Delivery of the posterior arm. **A.** Birth of the posterior shoulder by upward traction. **B.** Freeing the posterior arm.

17. Engagement of the head usually occurs in one of the oblique pelvic diameters. Internal rotation is facilitated by delivery of the shoulders; once the arms are released, the head is generally in an occiput anterior position, with the back of the neck visible at the vaginal introitus.
18. It is very important *not* to let the head rotate to the occiput posterior position, as any traction on the body will cause extension of the head and increase the chances of entrapment.
19. Delivery of the head is best accomplished using the *Mauriceau–Smellie–Veit maneuver* (**Figure 29D-6**). There are several variations of this maneuver, in which the hand is inserted posteriorly into the vagina and the fingers are positioned carefully in and/or around the baby's mouth to facilitate flexion of the head. Most commonly, the index and

middle fingers are placed on either side of the nose on the maxilla and downward pressure is applied to maintain flexion of the head. Extreme care must be taken to avoid any pressure on the lower jaw or accidentally inserting the fingers into the orbit of the eyes. Although insertion of a finger into the infant's mouth is sometimes described as part of this maneuver, this modification could cause serious damage to the tongue and surrounding tissues.

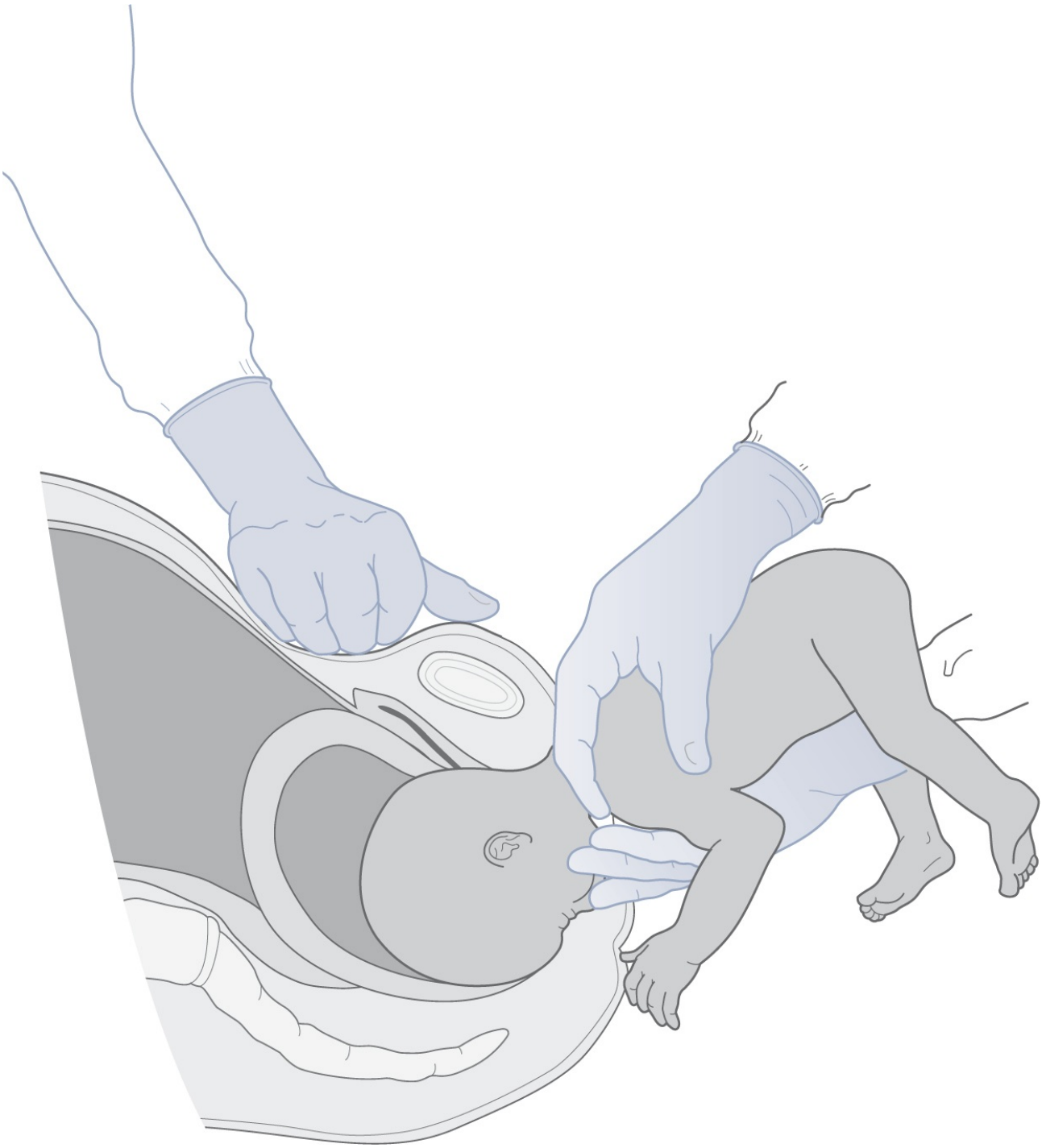


Figure 29D-6 Mauriceau–Smellie–Veit maneuver with suprapubic pressure.

20. Once the fingers of the posterior hand are in place on the maxilla, rest the infant's body

astride the length of the arm. Place the other hand on top of the infant and hook the index and middle fingers of this hand onto the shoulders on each side of the infant's neck. The fingers can also be placed along the occiput to splint the infant's head between the hands to prevent any extension of the neck and head.

21. An assistant should be directed to perform suprapubic pressure to maintain flexion of the head during this part of the birth.
22. Continue to apply downward traction with the infant sandwiched between the arms/hands until the hairline is visible under the symphysis pubis. Take care to avoid pressure on the brachial plexus.
23. Once the hairline is visible, apply upward traction while elevating the body of the baby. Following the curve of Carus, the chin, face, brow, anterior fontanelle, and the remainder of the head are delivered in sequence across the perineum.

References

1. Yeomans ER, Gilstrap LC. Breech delivery. In: Queenan JT, Spong CY, Lockwood CJ, eds. *Queenan's Management of High-Risk Pregnancy: An Evidence-Based Approach*. 6th ed. Malden, MA: Wiley-Blackwell; 2012:424-428.
2. Glezerman M. Planned vaginal breech delivery: current status and the need to reconsider. *Expert Rev Obstet Gynecol.* 2012;7:159-166.
3. Alarab M, Regan C, O'Connell MP, Keane DP, O'Herlihy C, Foley ME. Singleton vaginal breech delivery at term: still a safe option. *Obstet Gynecol.* 2004;103:407-412.
4. American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 306: mode of term singleton breech delivery. *Obstet Gynecol.* 2006;108:235-237.

CHAPTER

30

Third Stage of Labor

MAVIS N. SCHORN

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Introduction

The birth of a child is exciting and the pinnacle of an intense and emotional period for the birthing woman and her family. However, the time immediately following the birth is also a critical one for both the woman and her newborn. The expulsion of the placenta and physiologic stabilization immediately following this event represent a period of great vulnerability. Postpartum hemorrhage (PPH) is one of the leading causes of childbirth-related maternal morbidity and mortality, both worldwide and in the United States; when it occurs, it most often does so during the third or fourth stage of labor.[1] Moreover, the first hour after birth is a "sensitive period" psychologically and physiologically, wherein continuous contact potentially has significant benefits for both the woman and her infant.[2] This chapter reviews the third stage of labor, methods for promoting normal physiologic adaptation, maternal complications that can occur during this time period, and management strategies for complications of the third stage.

Definitions

The third stage of labor is defined as the period following the birth of the newborn through the expulsion of the placenta. A duration of longer than 30 minutes without placental expulsion is the conventionally accepted criterion for the diagnosis of *prolonged third stage of labor*, which is also known as *retained placenta*. Retained placenta is associated with an increased risk for hemorrhage and complications that are partially related to the anesthesia used when the placenta must be manually removed.[3-6]

Physiology of the Third Stage of Labor

Duration of the Third Stage

The length of time for the third stage of labor to be completed is usually within 10 minutes.[7] Based on a review of more than 12,000 births, only 3% of third stages have a duration that is longer than 30 minutes.[5] The duration of the third stage of labor is commonly longer in preterm gestations compared to term gestations.[8] Although the outer limit of normalcy has not been conclusively determined for any gestational age, the risk of complications increases with increasing duration of the third stage at all gestations.[5-7] A study of more than 45,000 births found that following a term gestation birth, 90% of placentas will be expelled spontaneously within 15 minutes and 97.8% will be expelled within 30 minutes.[6]

In a setting in which uterotonic medications were routinely administered during the third stage of labor, researchers reported that the mean duration of the third stage was 5.6 minutes and the 99th percentile was 28 minutes.[5] In this analysis, the risk for postpartum hemorrhage was significantly increased (adjusted odds ratio [aOR] 1.82; 95% confidence interval [CI], 1.43–3.21; 13.2% vs 8.3%) when the duration of the third stage was greater than 20 minutes.[5] Although some research suggests the diagnosis of retained placenta should be applied when the third stage lasts 20 minutes or more,[5,6] 30 minutes without placental expulsion continues to be the standard definition for the prolonged third stage or retained placenta. While most settings in the United States initiate intervention after 30 minutes, the World Health Organization (WHO) recommends that, in the absence of bleeding, intervention not be initiated until 60 minutes after birth if the placenta has not delivered.[9]

Placental Separation

Contraction frequency and intensity in the third stage of labor are similar to the corresponding measures during the second stage of labor,[8,10] although the intensity of the contractions often is felt minimally, if at all by the woman, especially in comparison to the sensations associated with contractions immediately before the birth of the neonate. These post-birth contractions constrict the blood vessels that supply the placenta, thereby causing hemostasis. Placental separation is the result of an abrupt decrease in size of the uterine cavity following birth of the neonate. As the uterus contracts, the placental implantation surface area decreases without a corresponding change in the placental size. This sudden disproportion between the area of implantation and placental size causes the placenta to buckle and separate from the decidua.

After the placenta separates, it descends through the lower uterine segment and into the upper vaginal vault, causing clinical signs of placental separation. **Table 30-1** lists the common signs of placental separation. During placental separation, women who do not have analgesia may experience a slight increase in the perception of contraction intensity and pelvic floor discomfort.

Table 30-1	Signs of Placental Separation

Signs of Placental Separation	Physiologic Explanation
A small gush of blood	With separation, some blood escapes from between the placenta and the decidua.
Lengthening of the umbilical cord	As the placenta descends into the vagina, the cord lengthens at the vaginal introitus.
Rise of the uterus into the abdomen	As the placenta descends into the vagina, the uterus is displaced cephalad.
The uterus becomes firm and rounded	Once the placenta is expelled from the uterus, the uterine smooth muscles contract more firmly, altering the shape of the uterus.

Placental Expulsion

Expulsion of the placenta occurs by one of two mechanisms: Shultz or Duncan. The majority of the time the Schultz mechanism occurs, although both mechanisms are considered normal.

In the *Schultz mechanism*, the placenta separates centrally. The fetal side of the placenta—the side with a smooth, shining membrane continuous with the sheath of the chorion (which also covers the umbilical cord)—drops to the lower portion of the uterus, and then exits through the cervix into the vagina and onto the woman's pelvic floor. At that point, the placenta is expelled, fetal side first. The membranes are expelled last, with the maternal side of the placenta inside the amniotic sac. This mechanism, in effect, inverts the placenta and amniotic sac and causes the membranes to peel off the remainder of the decidua and trail behind the placenta. The majority of bleeding that occurs during this mechanism may remain hidden until the placenta and membranes are expelled, as the blood is captured behind the placenta and inside the amniotic sac (**Figure 30-1**).

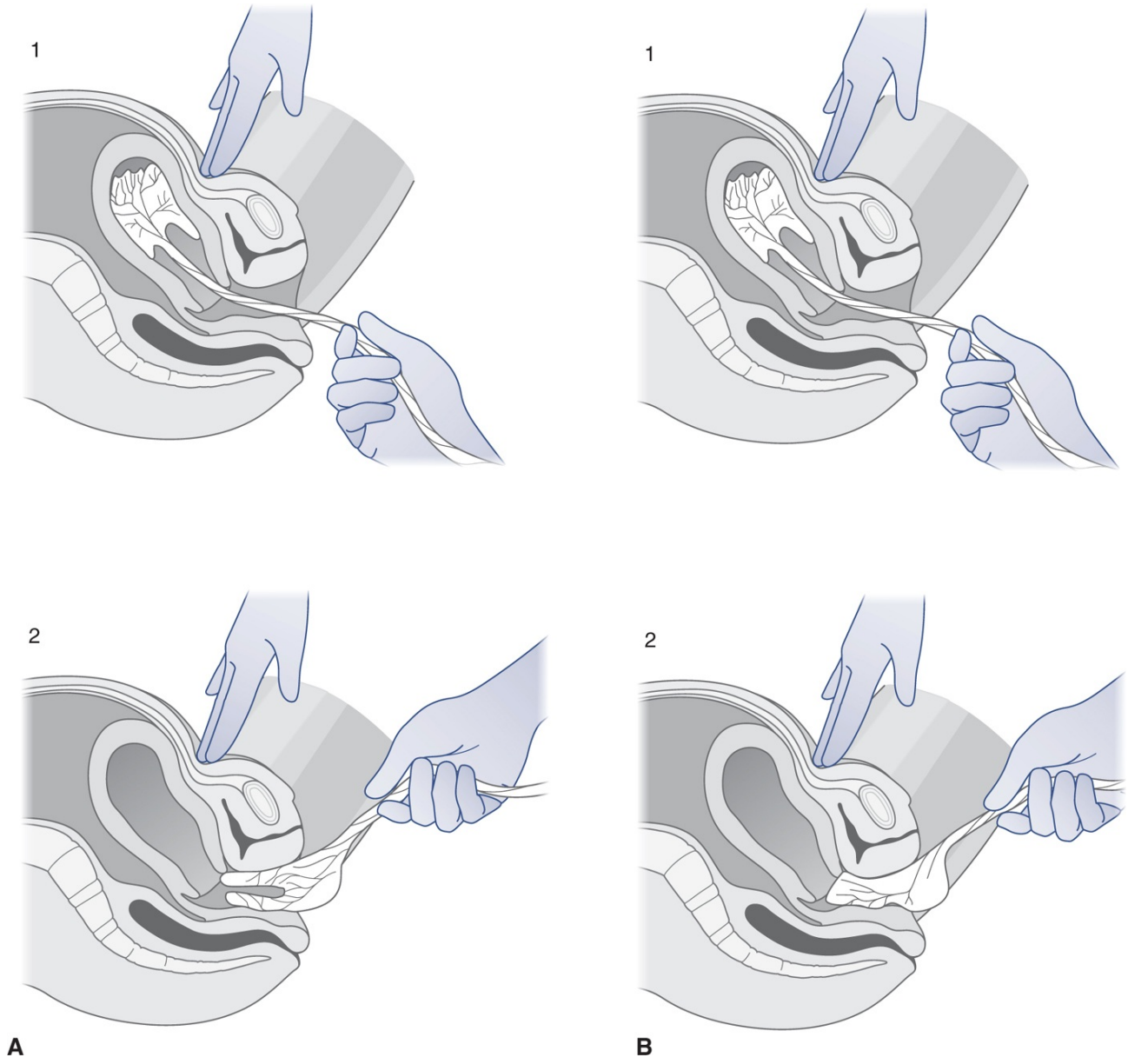


Figure 30-1 Mechanisms of placental expulsion. **A.** Schultz. **B.** Duncan.

In contrast, in the *Duncan mechanism*, the placenta separates marginally. During this process of expulsion, the placenta slides into the cervical opening without inverting, so that the maternal side of the placenta, with dark red cotyledons, is expelled at the same time as the fetal side. Blood escapes between the membranes and uterine wall and is more likely to be visible externally earlier in the separation process. The amniotic sac is not inverted but trails behind the placenta.

The memory aid for correctly identifying the mechanisms of placental expulsion is based on the appearance of the two different sides of the placenta first seen at the vaginal introitus. The fetal side is shiny and glistening from the fetal membranes covering the placenta, whereas the maternal side is dark and beefy—hence the sayings “Shiny Schultz” and “Dirty Duncan.” In past years, some sources have suggested that there is a higher risk of postpartum bleeding when trailing membranes are apparent (as in the Duncan mechanism), but no evidence has been found to substantiate this belief.

Management of the Third Stage of Labor

Although research in the area of third-stage management has been increasing, little is known about how the first and second stages of labor affect the third stage. The primary focus of routine management techniques used during the third stage of labor is to prevent postpartum hemorrhage. Evidence-based management of the third stage is controversial and practices vary. Best practice is rapidly evolving as research identifies new evidence.

The two standard approaches to third-stage management are commonly described as physiologic (expectant) management and active management.[11] The physiologic approach is a noninterventionist approach.[12] This strategy is more commonly followed by midwives in many parts of the world compared to active management.[13-15] Nevertheless, active management is the approach recommended by the International Confederation of Midwives (ICM), the International Federation of Gynecologists and Obstetricians (FIGO), and the WHO based on their interpretation of data reporting decreased PPH.[9,16] **Table 30-2** compares these two approaches. Studies have shown that birth attendants and birth facilities often use a mixed approach that incorporates portions of each approach, rather than strictly following one or the other.[15,17,18]

Table 30-2 **Comparison of Physiologic and Active Management Approaches to Management of the Third Stage of Labor**

Intervention	Physiologic Management	Active Management
Maternal positioning	Upright positioning of the woman to promote gravity assistance	Not addressed
Delayed cord-clamping	Yes	WHO recommends delayed cord-clamping ICM/FIGO has a footnote attached to the recommendation for controlled cord-clamping stating that delaying cord-clamping by 1 to 3 minutes reduces anemia in the newborn
Cord traction	No—generally a “hands-off” approach with spontaneous expulsion by the mother	Yes—continuous gentle cord traction following signs of placenta separation
Maternal efforts to expel the placenta	Delivered with aid of gravity or maternal efforts	Not addressed
External uterine massage following placental expulsion	No—generally a “hands-off” approach to the uterus	Yes (ICM/FIGO only)
Routine administration of a uterotonic agent	No	Yes
Early infant suckling	Yes	Not addressed
Draining the umbilical cord	Not addressed	Not addressed

Abbreviations: FIGO, International Federation of Gynecology and Obstetrics; ICM, International Confederation of Midwives; WHO, World Health Organization.

Based on World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. 2012. Available at: http://apps.who.int/iris/bitstream/10665/75411/1/9789241548502_eng.pdf. Accessed January 28, 2017[9]; Begley CM, Gyte GML, Devane *D, McGuire W, Weeks A. Active versus expectant management for women in the third stage of labour. Cochrane Database Syst Rev.* 2015;3:D007412. doi:10.1002/14651858.CD007412.pub4[11]; International Confederation of Midwives, International Federation of Gynecology and Obstetrics. Joint statement: management of the third stage of labour to prevent post-partum hemorrhage. Available at: http://www.internationalmidwives.org/assets/uploads/documents/FIGO/PPH%20Joint%20Statement.pdf. Accessed January 28, 2017.[16]

Active Management of the Third Stage

Active management of the third stage of labor has been shown to decrease the risk of postpartum hemorrhage in a general perinatal population.[11] The most recent meta-analysis of active management protocols compared active management of the third stage of labor to physiologic management for the Cochrane Collaboration.[11] Women in the active management arm had a significant reduction in estimated blood loss exceeding 500 mL (relative risk [RR], 0.33; 95% CI, 0.20–0.56; *n* = 2941), blood transfusion (RR, 0.30; 95% CI, 0.10–0.88; *n* = 3134), and need for therapeutic uterotonic administration (RR, 0.15; 95% CI, 0.11–0.21; *n* = 3134) compared to women whose third stage of labor was managed expectantly.[11] Thus, this strategy is of particular import in low-resource settings where treatment of women with postpartum hemorrhage is not readily available.

The active management approach initially evaluated in randomized controlled trials (RCTs) included four elements: (1) controlled cord traction to facilitate placental expulsion, (2) early cord-clamping and cutting, (3) the routine administration of a prophylactic uterotonic, and (4) fundal massage (in some studies).[10] However, the individual components of active management were not well studied, and each has possible adverse consequences when examined independently. In addition, the management of first and second stages of labor also may influence the third-stage management, but third-stage management has not been studied in association with management that occurred during labor.[12] Therefore, as active management of labor protocols have evolved, some individual components have been deleted from the package of care practices.

The ICM/FIGO joint statement recommends three steps: (1) controlled cord traction (once pulsation stops in a healthy newborn); (2) use of a uterotonic agent; and (3) fundal massage after expulsion of the placenta.[16] Early cord-clamping was not included in this recommendation because delayed cord-clamping has known positive benefits for the newborn.[19-21] The WHO recommends use of a uterotonic agent in all settings, but controlled cord traction only in settings where a skilled birth attendant is present; in support of this recommendation, the WHO cites evidence that both controlled cord traction and early cord-clamping can be harmful to the mother and the infant, respectively.[9] Sustained fundal massage is not recommended by the WHO.[9]

Since the initial active management protocols were published, controlled cord traction, uterotonics, fundal massage, and early versus delayed cord-clamping have all been the subject of considerable research.[19-25] As new information has emerged, the recommended components

of active management have further evolved.[11,26] Current evidence suggests that the administration of a uterotonic agent during the third stage of labor is the most effective treatment for prevention of postpartum hemorrhage and likely the only component within the active management triad that has consistent clinical utility.[26]

A variety of uterotonic drugs are available, and controversy exists about the best route, dose, and timing when used as part of an active management of the third stage protocol. Current guidelines from the ICM/FIGO and the WHO recommend oxytocin (Pitocin) 10 U administered intramuscularly or intravenously after being diluted in solution as the first-choice agent.[9,16] Although the guidelines refer to administration of oxytocin after the birth of the infant, this medication can be given as the anterior shoulder is released under the symphysis pubis, during the birth of the neonate, after the birth of the infant, or after placental expulsion. The timing does not influence the amount of bleeding or the rate of postpartum hemorrhage.[27] Administration of a uterotonic agent with the birth of the neonate is effective and safe unless the woman has a multifetal gestation with another fetus in the uterus. Some midwives have expressed concern that administering the uterotonic before the placenta is expelled will increase the risk of an entrapped placenta; however, this outcome has not occurred in any of the studies of uterotonics, so it remains a theoretical concern that does not have supporting clinical evidence.

Physiologic Management

Physiologic management of the third stage of labor includes no routine uterotonic administration, delayed cord-clamping, and gentle—if any—cord traction. Maternal efforts to expel the placenta are encouraged. A Cochrane Collaboration analysis found heterogeneity in how the components of physiologic management were implemented in the studies analyzed.[11] However, the analysis also found that there were no significant differences in the risk for severe postpartum hemorrhage (RR, 0.31; 95% CI, 0.05–2.17; n = 2941) among women who did not have an a priori risk for postpartum hemorrhage when active management was compared to expectant management.

A secondary analysis of data from RCTs suggests that prophylactic oxytocin (Pitocin) for women who do not receive intrapartum oxytocin for induction or augmentation for the reduction of postpartum hemorrhage (greater than 1000 mL) may not have the same protective effect as for women who already have received intrapartum oxytocin.[12] Therefore, women who are at low risk for postpartum hemorrhage may be offered physiologic management in settings that have ready access to treatments for postpartum hemorrhage, whereas active management may be more beneficial in low-resource settings.

Mixed Management

Mixed management incorporates some components of both active and physiologic management of the third stage and is common practice. The primary change in practice that has resulted in a mixed management strategy is the introduction of delayed cord-clamping, which has proven benefits for the newborn.[19] In this scenario, a midwife may routinely administer a uterotonic

agent after the birth but delay clamping the cord either for an arbitrary period of time or until it stops pulsating, then apply gentle controlled cord traction.[19] In clinical practice, the combination of a midwife's usual practice, the birth site, institutional or practice guidelines, the birthing woman's history, and birth situation, along with the woman's particular preferences likely influence how the third stage of labor is managed and rarely conforms strictly to either expectant or active.

A survey of midwives and physicians reported more than 100 interventions that they or others may use during the third stage of labor.[14,17] However, the myriad techniques used during the third stage were found to cluster into distinct practice patterns ranging from primarily hands-off care to higher hands-on interventions.[17] The effect of these patterns—that is, these clusters of care practices—on the rate of postpartum hemorrhage remains unknown.

Uterotonics to Prevent Postpartum Hemorrhage

Although oxytocin (Pitocin) is the first choice for postpartum hemorrhage prophylaxis, it may not be as effective for women who received large doses of oxytocin during labor for induction or augmentation.[12] In this case, a different uterotonic agent may be indicated. Ergot alkaloids (methylergonovine maleate), prostaglandins, and Syntometrine (an oxytocin/ergometrine combination drug not available in the United States) are second-line prophylactic uterotonic agents.[28] Misoprostol is not as effective for prophylaxis but may be used if other uterotonics are not available.[28]

The routine use of oxytocin, or any other uterotonics, with normal saline via umbilical vein injection has not been found to be effective and should not be used for prevention of postpartum hemorrhage.[29] The effectiveness of complementary treatments such as herbs or homeopathic remedies for postpartum hemorrhage reduction is still unclear.[30]

Cord Traction

Continuous cord traction initially was recommended as part of the active management package, along with early cord-clamping, because of concerns related to early administration of a uterotonic agent—namely, the belief that the placenta should be expelled quickly to avoid entrapment when the cervix reconstitutes. However, entrapment of the placenta has not been found to occur in studies that evaluated delayed cord-clamping,[31] and the rationale for controlled cord traction is less convincing.

Gentle cord traction is needed to guide the placenta out of the vagina once the placenta has separated. Following signs of placental separation, the midwife applies gentle counter-traction to the lower uterine segment abdominally with one hand, while the other hand applies continuous gentle downward traction on the cord. An instrument such as a hemostat is used to clamp the umbilical cord close to where the cord exits the introitus; alternatively, a piece of gauze can be wrapped around the cord to promote easy traction. The traction should follow the curve of Carus. For example, if the woman is in a semi-recumbent position, the cord is guided inferiorly as the placenta descends to the pelvic floor, and as the placenta becomes visible at the introitus, the cord is guided superiorly.

If the woman is in a squatting or other upright position, cord traction is likely not necessary and may even be dangerous. Excessive cord traction increases the risk for uterine inversion—a risk that theoretically might be further increased if the woman is in an upright position. A woman in an upright position will likely feel the pressure of the placenta as it descends to the pelvic floor and spontaneously expel it.

Umbilical Cord Drainage

Umbilical cord drainage in the third stage of labor involves the removal of the clamp from the previously clamped and cut umbilical cord (after necessary cord blood samples have been obtained) and the draining of the blood from the cord and placenta. In a meta-analysis of three RCTs (n = 1257), Soltani et al. found that cord drainage may reduce the length of the third stage of labor by approximately 3 minutes and total blood loss by less than 80 mL when compared to no cord drainage.[32] It is questionable whether this is a clinically important finding. No decrease in third-stage complications was found when the umbilical cord was drained as compared to when the umbilical cord was not drained.[32] If umbilical cord drainage is incorporated into third-stage management, the blood should not be counted in the estimated or weighed measurement of maternal blood loss, as this is fetal blood. In some settings, such as an out-of-hospital birth site, disposal of the blood may be more problematic than when it is retained in the placenta.

External Uterine Massage Following Placental Expulsion

Uterine massage following the expulsion of the placenta is a component of active management of the third stage of labor that is recommended by both ICM and FIGO.[16] However, the precise benefit (if there is one) of routine external uterine massage is still undetermined.

One RCT has been published that compared uterine massage to no uterine massage in three groups of women who received uterine massage, uterotonics, or both, respectively.[33] The incidence of blood loss of more than 300 mL and need for more uterotonics was statistically higher in the women who received uterine massage only.[33] Nevertheless, when oxytocin (Pitocin) was used, there appeared to be no additional benefit from uterine massage.

If a uterotonic agent is administered, external massage may not further increase the protection against an early postpartum hemorrhage; moreover, it can cause significant maternal discomfort.[34] Uterine massage before expulsion of the placenta is not recommended because if the uterus is massaged before the placenta separates from the uterine wall, the massage may cause incomplete separation of the placenta, resulting in iatrogenic hemorrhage.

Maternal Blood Loss Measurement

Measurement of maternal blood loss at birth provides objective data that will help inform care of the woman following birth. Visual estimation of blood loss has been routine practice for many years, although repeated studies have demonstrated the inaccuracy of this simple method.[35] If a large blood loss occurs in the immediate postpartum period, the amount is

commonly underestimated visually.[35-37] If visual estimation is the usual method of evaluating blood loss, it is beneficial to have experience through regular laboratory simulation to improve estimation accuracy.

Although training improves the accuracy of blood loss estimation, alternative quantitative methods of assessing blood loss are recommended. Two methods that are relatively easy to implement are measuring and weighing. A calibrated under-buttocks drape can be used to measure blood loss. If a calibrated drape is not available or the woman gives birth in a position that does not allow for an under-buttocks drape (e.g., supported squat), weighing blood-saturated items can be implemented. Because *1 gram is equivalent to 1 milliliter in volume*, weighing blood-saturated items and subtracting the weight of the item when dry provides a relatively accurate quantitative measurement of blood loss. Either the pads or calibrated drapes should be used immediately after birth to limit additional content such as amniotic fluid or urine from being included in the blood loss measurement.

Some birth sites routinely obtain a quantitative measure of blood loss; however, this step can also be instituted if blood loss is estimated to be higher than normal to improve accuracy and guide management. **A** worksheet with the weight of commonly used items can be used to expedite the process of calculating a quantitative measure of blood loss.[38,39] Some hospitals have incorporated these calculations into their electronic medical records.[39]

Midwifery Management of the Third Stage of Labor

The sequential steps for midwifery management of the third stage of labor are presented in **Appendix 30A**.

Supporting Women's Choices

Maternal requests for nonintervention during the natural progress of the third stage are important to address prior to labor. The process of shared decision making will include discussion of ways to support normalcy during the third stage and the evidence for interventions known to minimize the risk for postpartum hemorrhage. Prenatal preventive measures such as a healthy diet to prevent anemia will lessen adverse effects of blood loss at birth. Avoiding procedures during labor and birth that increase postpartum blood loss, such as induction, augmentation, and episiotomy, also decreases the risk of hemorrhage. Conversely, it is important to remember that delaying the use of uterotonics until bleeding is excessive may increase a woman's risk of hemorrhage.

The placenta, cord, and membranes have special cultural or spiritual significance in many cultures. In turn, additional maternal requests for third-stage management reflecting these beliefs may be quite varied. Rituals related to the placenta exist in many cultures today, and the woman or family may want to save the placenta for a variety of purposes. The ritual of burying the placenta may be undertaken for the purpose of protecting the child, protecting the woman, or establishing a spiritual or sacred link between the land and the child.[40,41] Parents may also be interested in leaving the placenta attached to the newborn until the cord falls off spontaneously, often referred to as a lotus birth.[41] Some women practice placentophagy or consume the placenta, raw or cooked, or encapsulated in pill form with the hope of preventing postpartum depression or as a galactogogue, although findings related to these practices generally have been inconclusive.[41-43]

Rituals also may involve the umbilical cord and even the membranes. The cord may be kept as a charm or used for medicinal purposes. Traditionally, when a newborn is born in a caul (i.e., with membranes covering the head), it means that the child will never drown; thus these membranes have been sold to sailors for protection.

When a woman has a special request, the focus should be on safety while also attempting to honor her request. Honoring the variety of beliefs surrounding this amazing organ is important whenever possible following a process of shared decision making.

Examination of the Placenta and Placental Pathology

After the uterus is evaluated and massaged, and the uterine bleeding has slowed, a methodical inspection of the placenta should be performed. Routine inspection of the placenta assists in recognition of abnormal findings and reduces the likelihood of overlooking a significant finding. The steps for inspection of the cord, placenta, and membranes are reviewed in **Appendix 30B**.

Table 30-3 lists common variations noted with the cord, placenta, and membranes. Some

variations, such as a true knot in the cord, may be simple incidental findings yet also associated with an increased risk for newborn morbidity. When observed, their presence should be noted in the health records of both the woman and the newborn.

Table 30-3 Umbilical Cord, Placenta, and Membrane Variations

Variation	Significance
Umbilical Cord	
Two-vessel cord	A two-vessel cord occurs among 0.5% to 1% of neonates. Two-vessel cords are associated with gastrointestinal, genitourinary, and cardiovascular abnormalities.
Long cord	A long cord, more than 75 cm, is associated with knots and fetal entanglement, as well as increased amniotic fluid and fetal activity.
Short cord	Short cords, less than 32 cm, are associated with disorders that limit fetal movement, such as neuromuscular disorders, fetal limb dysfunction, and Down syndrome. Short cords are associated with fetal heart rate abnormalities in labor as a result of traction on the cord with fetal descent.
Thin cord	Associated with oligohydramnios and poor fetal growth.
Thick cord	Associated with polyhydramnios and macrosomia.
Marginal (Battledore) insertion	The cord is inserted at or within 1.5 cm of the margin of the placenta. May be an insignificant finding but is associated with preterm labor, abnormal fetal heart rate patterns in labor (compression), and bleeding in labor (vessel rupture).
Velamentous insertion	The umbilical cord vessels run through the amnion and chorion before entering the placenta, leaving the vessels unprotected (see **Figure 30B-3**). This type of insertion may lead to rupture and fetal hemorrhage, particularly if associated with vasa previa (transcervical position). This finding is more common in twin placenta.
Knots, loops, torsion, or strictures	These variations are associated with increased fetal mortality and morbidity.
Placenta	
Pale placenta	A pale placenta is associated with immature neonates and anemia. Edematous, pale, and bulky placentas are seen with immune and nonimmune hydrops fetalis, twin-to-twin transfusion syndrome, fetal congestive heart failure, and infection.
Green-stained placenta	With extended exposure to meconium, a placenta may be stained green.
Calcifications	The maternal surface is usually smooth but may have gritty white areas of calcification. Generally, the quantity of calcifications has no clinical importance.
Missing cotyledon	Incomplete expulsion of the placenta. It is associated with uterine atony and postpartum infection.
Small, light placenta	A small placenta is less than the 10% percentile for gestational age and may be associated with intrauterine infections, genetic mutations, or hypertension.
Large, heavy placenta	A large placenta, more than the 90% percentile for gestational age, is often due to various maternal or fetal conditions such as maternal diabetes, fetal erythroblastosis and fetal hydrops, fetal congestive heart failure, and maternal–fetal syphilis.
Infarctions	A localized area of ischemic tissue necrosis that occurs when the villus blood supply is obstructed. Placental infarctions are most common at the periphery of the placenta. Small infarctions are normal variants. Larger infarctions are associated with hypertension, stillbirth, placental abruption, and fetal growth restriction. Any condition that interferes with uteroplacental circulation can lead to placental infarcts.

Nodules or plaques on the fetal surface	Associated with oligohydramnios and renal agenesis, squamous metaplasia (benign), and infection.
Foul odor	Associated with infection.
Placenta	
Circumvallate placenta	With this configuration, the membranes appear to arise not from the edge of the placenta, but rather a short distance inward toward the umbilical cord. As a consequence, the area of the chorionic plate is reduced. Fetal membranes fold back upon themselves, creating a dense, gray/white ring that surrounds the outer margin of the placenta. The fetal vessels stop at this ring. The risk of placental abruption is increased with this variation. Possible causes include a variety of developmental abnormalities. Usually this finding is insignificant, but it has been associated with threatened abortion, preterm labor, painless vaginal bleeding after 20 weeks' gestation, placental insufficiency, fetal growth restriction, and intrapartum and postpartum hemorrhage.
Marginate or circummarginate placenta	A form of circumvallate placenta in which the fetal membranes do not fold back, creating the white ring. In this variation, placental tissue appears to extend beyond the marginate ring where the membranes arise. Usually this finding is insignificant.
Succenturiate placenta	One or more smaller accessory lobes of placenta are developed in the membranes and are attached to the main placenta by fetal vessels. This finding is more common in multifetal gestations. The accessory lobes may be retained, leading to postpartum hemorrhage or infection. These placentas can be associated with velamentous insertion of the cord.
Bilobate placenta	Also referred to as bipartite or duplex. A placenta with three or more lobes is referred to as multilobate.
Membranes	
Cloudy membranes	Associated with infection.
Incomplete membranes	Remnants of membranes are usually spontaneously expelled after the birth; however, they can cause subinvolution of the uterus, uterine atony, or infection if retained.

Placental Pathology

Placental evaluation by a pathologist is not indicated for all placentas. A pathology examination of the placenta can be diagnostic in some situations, however, and the College of American Pathologists has published recommendations for when placental pathology examination should be performed.[44] For example, a pathology examination of the placenta can help establish a diagnosis when stillbirth occurs.[45] Placental pathology is commonly organized into four categories:

- Identification of a previously unsuspected disease process in the woman or neonate that requires immediate attention (e.g., unusual infection such as *Listeria*)
- Conditions with a high probability of subsequent reoccurrence (e.g., placenta accreta)
- Information that can guide management of future pregnancies or influence long-term care of the woman or neonate (e.g., spontaneous preterm birth with histologic chorioamnionitis)
- Diagnosis that provides an explanation for an adverse outcome such as fetal death or growth restriction[46]

A detailed placental pathology examination may provide valuable information or an explanation for neonatal neurodevelopmental impairment or other adverse pregnancy outcomes.[47,48] **Table 30-4** lists some of the clinical scenarios that are commonly cited as indications for requesting a detailed pathology examination of the placenta.[44,48]

Table 30-4 Clinical Conditions That May Indicate Pathology Examination of the Placenta After Birth

Maternal Indications[a]	Fetal or Neonatal Indications	Placental Indications
Abruption, unexplained third-trimester bleeding, or excessive bleeding	Congenital anomalies	Gross placental abnormalities (shape, infarct, mass, amnion nodosum, abnormal coloration, malodor, thrombosis)
Infection during this pregnancy (e.g., human immunodeficiency virus, syphilis, cytomegalovirus, parvovirus, primary herpes, toxoplasmosis, rubella, Zika)	Fetal growth restriction or birth weight less than 10th percentile	Morbidly adherent placenta
Invasive procedure with suspected placental trauma	Gestational age < 34 weeks or > 42 weeks	Placental abruption
Maternal fever or chorioamnionitis	Hydrops fetalis	Umbilical cord abnormalities (long or short, marginal or velamentous insertion, two-vessel cord)
Maternal trauma	Infection or suspected neonatal sepsis	
Oligohydramnios	Neonatal neurologic problems (e.g., seizures)	
Polyhydramnios	Newborn with abnormal umbilical cord blood gas values, low Apgar scores or requiring NICU admission	
Systemic disorder with clinical concerns for mother or neonate (e.g., autoimmune disease, diabetes, hypertension, collagen disease, seizures, severe anemia, thrombophilias or thromboembolic disorder)	Multifetal gestation	
Substance abuse	Stillbirth/early neonatal death	
	Thick meconium	
	Vanishing twin first trimester	
	Very-low-birth-weight infant	

[a] Some experts recommend placental pathology be performed if the woman experienced ruptured membranes for > 24 hours.

Based on Langston C, Kaplan C, Macpherson T, et al. Practice guideline for examination of the placenta: developed by the Placental Pathology Practice Guideline Development Task Force of the College of American Pathologists. *Arch Pathol Lab Med.* 1997;121:449-476[44]; Curtin WM, Krauss S, Metlay LA, Katzman PJ. Pathologic examination of the placenta and observed practice. *Obstet Gynecol.* 2007;109:35-41.[48]

Complications of the Third Stage of Labor

Although most births end with an uneventful third stage of labor, some of the most severe childbirth complications occur during the third stage, including retained placenta, placenta accreta, and uterine inversion. Postpartum hemorrhage can occur in the third stage of labor or in the first hours postpartum.

Retained Placenta

When active management of the third stage is practiced, the length of the third stage should be less than 20 minutes, and 98% of women will have expelled the placenta by 30 minutes after birth of the newborn.[49] Following physiologic management of third-stage labor, 98% of women will have expelled the placenta by 60 minutes after birth of the newborn.[50]

Risk factors for a retained placenta include previous retained placenta, previous uterine curettage, preterm birth, preeclampsia, augmented labors, birth in a dorsal position, nulliparity, and use of ergotamine (Methergine).[49,51] It is theorized that ergotamine may trap the placenta when this medication is used routinely prior to expulsion of the placenta for prevention of postpartum hemorrhage, as it causes a clonic or tetanic contraction. The mechanism of action of ergotamine contrasts with that of oxytocin (Pitocin), which is not associated with an increased risk for a retained placenta.

The primary risk associated with retained placenta is hemorrhage. The risk of hemorrhage begins to increase at approximately 20 minutes after birth with the placenta not expelled and continues to increase until it plateaus at approximately 75 minutes after birth.[51] A consultation should be obtained once a prolonged third stage has been diagnosed. The midwife may initiate several interventions when the third stage is increasing in length, including encouraging an upright position for the woman, nipple stimulation via encouraging the newborn to breastfeed or manual nipple stimulation, and ensuring an empty bladder, by means of catheterization if indicated.

If the placenta still has not separated following conservative measures and the time interval has passed 30 minutes, the next step is manual removal of the placenta. Nonemergency manual removal of the placenta should occur in a hospital setting, both for reasons of safety and to enable the woman to receive anesthesia or deep analgesia for this usually painful procedure. **Appendix 30C** reviews the procedure for manual removal of the placenta. In the nonemergency situation, the midwife can manually remove the placenta in consultation with a physician or wait for a physician to conduct the procedure. If the woman is hemorrhaging, the midwife may need to manually remove the placenta emergently.

Avulsed Cord

Excessive cord traction before complete placental separation can cause the umbilical cord to detach or avulse from the placenta, necessitating manual removal of the placenta—an intervention that exposes the woman to unnecessary pain and an increased risk of infection. A sensation of tearing will be felt as the cord is detaching. If this is noticed, cord traction should

be discontinued immediately and signs of placental separation should be apparent before resuming. Repositioning the woman in an upright position may facilitate placental expulsion.

Uterine Inversion

Uterine inversion occurs when the fundus collapses into the endometrial cavity, which turns the uterus partially or completely inside out. Uterine inversion is a rare event, occurring in approximately 2.9 in 10,000 births; however, it has been found to occur primarily in women who have low-risk pregnancies.[52] Strong umbilical cord traction and fundal placental implantation are associated with this condition, although it may also occur spontaneously. The uterine inversion can be incomplete (fundus within endometrial cavity), complete (fundus protrudes through the cervical os), prolapsed (fundus protrudes to or beyond vaginal introitus), or total (both the uterus and the vagina are inverted). Incomplete or complete inversions feel like a soft tumor that can be felt in the cervical or vaginal orifice, while abdominally a funnel-like depression may be felt instead of a rounded fundus.

Uterine inversion is associated with maternal shock and disseminated intravascular coagulation (DIC). Thus, this complication can be a life-threatening emergency. Uterine inversion should be suspected and a vaginal examination performed if the woman develops symptoms of shock after giving birth without obvious reason. Excessive bleeding and severe uterine pain may or may not be present. **Appendix 30D** presents the management of uterine inversion and describes the position of the midwife's hands during this maneuver.

Placenta Accreta

Placenta accreta is the term for a placenta that is abnormally adherent to the uterine wall. Placenta accreta is further subcategorized as *placenta increta*, when the placenta invades the myometrium, and *placenta percreta*, when it has extended through the myometrium and uterine serosa and into adjacent tissue, often the bladder. Placenta accreta most often occurs when the placenta implants over an area where there is a deficiency of decidual formation, such as over a uterine scar or in the lower uterine segment.[53] Women who have a cesarean birth are at increased risk for placenta accreta in subsequent pregnancies. Women who have had one or more previous cesarean births and who have a placenta previa in the current pregnancy are particularly at risk for placenta accreta. The risk of occurrence increases with increasing number of cesarean births.

The overall rate of placenta accreta has been reported to range from 1 in 2500 pregnancies to (more recently) 1 in 500 pregnancies.[53] It has been proposed that the increasing rate of placenta accreta is occurring secondary to the rising rate of cesarean births.

If the placenta is completely adherent, there usually is minimal bleeding. If it is partially separated or partially adherent, there will likely be more bleeding, which may or may not be visible. If placenta accreta is suspected due to a retained and adherent placenta, physician consultation and in-hospital care are necessary. Hysterectomy while leaving the placenta intact is generally the recommended treatment for placenta accreta.

Postpartum Hemorrhage

Postpartum hemorrhage can occur at any time in the first 6 weeks postpartum and is divided into two types. Postpartum hemorrhage is termed *early* or *primary* when it occurs within the first 24 hours postpartum; in contrast, it is described as *late* or *secondary* when it occurs after 24 hours and up to 6 weeks postpartum.[54,55]

Internationally, postpartum hemorrhage is the leading cause of maternal mortality. The incidence of early postpartum hemorrhage—and especially severe hemorrhage requiring blood transfusion—has markedly increased in the last several years in many developed nations, including the United States.[56] Although many factors are involved in the increasing incidence of postpartum hemorrhage, a significant proportion of this increase is associated with more frequent use of oxytocin for augmentation and cesarean section birth.[56] In the United States, postpartum hemorrhage occurs in 2% to 3% of all births but is the cause of 20% of all maternal deaths.[56] Most importantly, more than half of all maternal deaths attributed to hemorrhage are preventable. In developed nations where adequate resources are present, these deaths are mostly attributed to failure to coordinate the management of this emergency.[57]

Identification of risk factors, preventive measures, and early detection and management can help mitigate the adverse consequences of this complication. Over the last several years, use of simulation, clinical guidelines, bundles, and checklists has been promoted as a means to improve how a team responds and manages emergencies. Use of guidelines for managing postpartum hemorrhage has lowered the incidence of severe maternal morbidity,[58] and a list of these resources is included at the end of this chapter.[59,60]

Approximately 800–1000 mL of blood flows to the gravid uterus each minute. Thus, significant blood loss can occur quite quickly if the uterus does not contract effectively immediately after birth.

Postpartum hemorrhage has been defined in several ways in the past. In 2014, to encourage standardization, a U.S. multidisciplinary collaborative, including member representation from the American College of Nurse-Midwives, defined early postpartum hemorrhage as a cumulative blood loss of 1000 mL or more or blood loss accompanied by sign/symptoms of hypovolemia within 24 hours following the birth (including intrapartum loss).[55] Previously, the generally accepted definition was a blood loss of more than 500 mL after a vaginal birth or more than 1000 mL blood loss following a cesarean birth.[54] Most healthy women, however, can tolerate a blood loss of 1000 mL without adverse consequences. Moreover, estimated blood loss is very often underestimated in clinical settings.[37] In fact, when blood loss is measured, it appears that 500 mL is the *average* blood loss following a normal spontaneous vaginal birth. Thus, the “greater than 500 mL” definition was somewhat arbitrary and not evidence based.

Other authors have suggested that a postpartum hemorrhage occurs when the postpartum hematocrit represents more than a 10-point drop from the prenatal value. This retrospective definition is not clinically helpful during the third stage of labor.

Finally, signs and symptoms of hypovolemia can be used to define a postpartum hemorrhage. Because symptoms such as hypotension and tachycardia appear only after 15% of blood volume is lost, these developments are not a sensitive indicator that the midwife can use to

identify a postpartum hemorrhage in the early stages, at the point when the excessive bleeding needs to be recognized and controlled effectively. In clinical practice, postpartum hemorrhage is a subjective assessment of an estimated blood loss that threatens hemodynamic stability. It is important to initiate steps to control postpartum bleeding before reaching any of these thresholds. Early action in the presence of excessive bleeding may prevent an actual hemorrhage.

Causes of Postpartum Hemorrhage

Table 30-5 lists the most common causes of early postpartum hemorrhage, along with a mnemonic (tone, tissue, trauma, thrombin) that can be used as a memory prompt.[39,60-62] Uterine atony and retained placental fragments are the causes of approximately 80% of early postpartum hemorrhages, although most women who experience a postpartum hemorrhage do not have an identifiable risk factor.[56,62] Late postpartum hemorrhage is most often caused by retained placental fragments, infection, coagulopathies, or subinvolution at the placental site.

Table 30-5 The "Four Ts" Mnemonic for Causes of Immediate Postpartum Hemorrhage

Cause	Etiology	Clinical Presentation
Tone		
Overdistended uterus—large fetus, multifetal gestation, polyhydramnios Prolonged labor (all stages) Rapid labor Oxytocin-induced or -augmented labor High parity Postpartum hemorrhage with previous birth Chorioamnionitis Poorly perfused myometrium—hypotension Medications such as magnesium sulfate, nifedipine, and some general anesthetics Uterine abnormalities such as fibroids	70%	Bright red bleeding, clots, boggy or soft uterus
Tissue, retained		
Avulsed cotyledon, succenturiate lobe Abnormal placentation—accreta, increta, percreta Retained blood clots	20%	Bright red bleeding, clots, boggy or soft uterus
Trauma		
Episiotomy, especially mediolateral Hematoma Lacerations of perineum, vagina, or cervix Ruptured uterus Uterine inversion	10%	Bright red bleeding, firm uterine fundus Hematomas: intense pain at site of bleeding Bright red bleeding firm uterine fundus

		Shock greater than expected from estimated blood loss
Thrombin		
Coagulopathies	1%	Excessive bleeding
Acquired coagulopathies such as placental abruption, amniotic fluid embolism, disseminated intravascular coagulation, HELLP syndrome (i.e., hemolysis, elevated liver enzymes, and low platelet count), stillbirth, or sepsis		
Congenital coagulation defects such as von Willebrand's disease		
Therapeutic anticoagulation		

Based on Rani PR, Begum J. Recent advances in the management of major postpartum hemorrhage: a review. *J Clin Diagnost Res*. 2017;11(2):QE01-QE05. doi:10.7860/JCDR/2017/22659.9463[61]; Evensen A, Anderson JM, Fontaine P. Postpartum hemorrhage: prevention and treatment. *Am Fam Physician.* 2017;95(7):442-449.[62]

Many strategies have been used to identify women who are at risk for excessive blood loss. Stratifying women into low-, medium-, and high-risk groups is valuable as a guide for anticipatory preparation. Even so, most postpartum hemorrhages occur in women who have no risk factors.[55,58,60]

Stages of Postpartum Hemorrhage

As shown in **Table 30-6**, symptoms of hypovolemia usually parallel the amount of blood lost. Thus, postpartum hemorrhage can be categorized as compensated, mild, moderate, or severe depending on symptoms and the amount of blood loss.[59] Women who are already volume depleted, such as those with preeclampsia, may decompensate more rapidly.

Table 30-6 Stages of Hemorrhagic Shock

	Compensated PPH	Mild PPH	Moderate PPH	Severe PPH
Blood volume loss (mL)	< 1000	1000–1500	1500–2000	≥ 2000
Percentage of blood volume lost (%)	10–15	15–30	30-40	≥ 40
Heart rate (bpm)	< 100	> 100	≥ 120	≥ 140
Systolic blood pressure (mm Hg)	Normal	Normal	Hypotension	Profound hypotension
Pulse pressure	Normal or increased	Narrowed	Narrowed	Narrowed
Respiratory rate/min	14–20	20–30	30–40	> 40
Urine output (mL/hr)	≥ 30	20–30	5–15	Slight or anuria
Mental status	Normal	Weakness, sweating, agitation or mild anxiety	Restless, pallor, confusion	Air hunger, collapse, lethargy

Abbreviation: PPH, postpartum hemorrhage.

The first physiologic compensation that occurs when blood loss is excessive is systemic vasoconstriction, which is clinically evident as a narrowed pulse pressure. Systolic blood

pressure begins to fall and pulse rises after 15% to 25% of blood volume is lost. Hypotension and tachycardia are relatively late signs of hemorrhage. Narrowing trends in pulse pressure are a better vital sign to initially monitor when bleeding becomes excessive.

Complications of Postpartum Hemorrhage

Postpartum hemorrhage can result in anemia, hypotension, and, in severe cases, hypovolemic shock that can lead to DIC, adult respiratory distress syndrome (ARDS), and Sheehan's syndrome.

DIC is a consumptive coagulopathy that results in depletion of platelets and coagulation factors. Its etiology is widespread activation of the clotting and fibrolytic systems. This cascade of events is stimulated by circulatory exposure to large amounts of tissue factor, which is present in the placenta. The result is uncontrolled bleeding. Recent studies of postpartum hemorrhage have found that DIC occurs earlier than previously thought.[61] Early treatment of a woman with DIC can significantly improve maternal outcomes. Evaluation of coagulation studies is part of the initial response to postpartum hemorrhage, and transfusion protocols for postpartum hemorrhage recommend treating DIC as soon as it is determined to be a significant risk.

ARDS and Sheehan's syndrome are rare complications of postpartum hemorrhage. ARDS results from damage to the endothelial cells in the pulmonary vasculature that causes fluid to leak into the alveoli, producing respiratory distress. Sheehan's syndrome is the result of pituitary necrosis.[63] The pituitary is perfused via venous pressure, which makes it vulnerable to ischemia and damage in the presence of hypotension. The most common initial symptom of Sheehan's syndrome is failure of lactogenesis II. Amenorrhea, adrenal insufficiency, hyponatremia, and hypoglycemia are additional complications of pituitary hypofunction.

Strategies to Prevent Postpartum Hemorrhage and Minimize Adverse Effects

Strategies to minimize the adverse effects of postpartum hemorrhage include correction of anemia preconceptionally or in the prenatal period, elimination of routine episiotomies, and avoidance of genital tract lacerations.[62] The best prevention strategy during the third stage of labor is active management of the third stage, starting with administration of a uterotonic agent during or immediately following the birth of the infant.[11] **Table 30-7** lists recommended preparations to implement for women at a moderate or high risk of early hemorrhage.

Table 30-7 Management Plan for a Woman at Moderate or High Risk for a Postpartum Hemorrhage

1. Provide anticipatory guidance to the woman and her significant other/family (with the woman's permission).
2. Choose the birth site based on the risk assessment.
3. Prepare a plan of care and communicate it to all healthcare team members, including nursing and anesthesia staff, when a woman is in labor. A physician may be alerted when the woman enters labor based on her level of risk.
4. Based on her risk, the plan of care for the woman in labor may include the following:

a. Order a blood type and screen or type and cross-match for blood if needed.
b. Initiate a large-bore intravenous line during labor (at least 18 gauge) for rapid access.
c. Catheterize the woman's bladder if it is full and she is unable to void, as a full bladder may impede postpartum uterine contraction effectiveness.
d. Consider pain management methods that will support the steps undertaken to manage a postpartum hemorrhage, and make a plan with the woman early in her labor.
e. Administer a uterotonic agent with the release of the neonate's anterior shoulder, assuming there is assurance that a second fetus does not exist.
f. Promptly initiate and maintain uterine massage after placental expulsion and continue close observation for bleeding.

If hemorrhage occurs, proceed to the steps outlined in Appendix 30E.

Management Steps for Treating Postpartum Hemorrhage

Vital signs such as narrowing pulse pressure or quantified blood loss can be used as a trigger or alert to initiate treatment guidelines and team assistance.

Table 30-8 summarizes the dose, contraindications, and side effects of the uterotonic agents commonly used to treat postpartum hemorrhage.[64-66] Oxytocin (Pitocin) is considered the first-line uterotonic. If the hemorrhage does not resolve with its use, second-line drugs—such as methylergonovine maleate (Methergine) or one of the prostaglandin analogues—are administered. Tranexamic acid is a fibrinolytic agent that has recently been studied for prevention and treatment of women with postpartum hemorrhage. Data from the few available RCTs that have been published suggest use of tranexamic acid decreases the incidence of maternal mortality when it is used to treat postpartum hemorrhage.[65] Further studies of this agent are needed to determine its adverse effects and relative effectiveness.

Table 30-8 Uterotonic Treatment of Immediate Postpartum Hemorrhage Due to Uterine Atony

Drug Generic (Brand)	Dose and Route	Contraindications	Side Effects	Clinical Considerations
First-Line Therapies				
Oxytocin (Pitocin)	10–80 U in 250 or 500 mL of normal saline or lactated Ringer's solution OR 10 U IM injection	Hypersensitivity	Cramping Large doses can cause hyponatremia	Onset of action: 2–3 min Effective in 15–30 min Duration of action: 2–3 hr if given IM Rapid infusion of an undiluted bolus can cause hypotension and cardiac collapse
Second-Line Therapy				
Methylergonovine maleate (Methergine)	0.2 mg IM May repeat in 5 min Thereafter every 2–4 hr	Hypertension, preeclampsia Do not give IV secondary to risk of sudden vasospasm and hypertensive or cerebrovascular accident	Cramping Nausea, vomiting Hypertension, seizure, headache	It is recommended that the woman's blood pressure be taken prior to administration Onset of action: 2–5 min Peak plasma concentration: 20–30 min Plasma half-life: 3–4 hr Women with coronary artery disease may have increased risk for myocardial infarction FDA black box warning to avoid breastfeeding for first 12 hours of newborn's life
Carboprost tromethamine or 15 methyl prostaglandin $F_{2\alpha}$ analogue (Hemabate)	250 mcg IM May repeat every 15–90 min, up to 8 doses	Asthma or active cardiac, pulmonary, renal, or hepatic disease	Nausea, vomiting, diarrhea, bronchospasm, hypertension Pyrexia is common	FDA black box warning to use recommended doses only and in a hospital setting as well as to avoid breastfeeding for first 12 hours of newborn's life Peak serum concentration: 30 min Less effective than methylergonovine
Dinoprostone (Prostin E_2)	20 mg vaginal or rectal suppository May repeat every 2 hours	Hypotension Cardiac disease	Nausea, vomiting, and diarrhea Pyrexia is common	FDA black box warning to use recommended doses only and in a hospital setting Onset of action: 10 min
Misoprostol (Cytotec)	600–800 mcg sublingual or 800–1000 mcg per rectum; 1 dose		Nausea, vomiting, diarrhea, abdominal pain Pyrexia can occur with higher doses	Onset of action: 3–5 min Peak concentration: 30 min following sublingual administration; 40–60 min following rectal administration
Tranexamic acid[a] (Cyklokapron)	1 gm IV, can be repeated after 30 minutes		Mild gastrointestinal signs	Recommended by WHO if other measures fail. Inhibits fibrinolysis so has a potential risk of thrombosis.

Abbreviations: FDA, U.S. Food and Drug Administration; IM, intramuscular; IV, intravenous; WHO, World Health Organization.

[a] Tranexamic acid is an antifibrinolytic that is being evaluated for prevention or treatment of postpartum hemorrhage. Contraindications and adverse effects have not been fully elucidated, studies are ongoing.

Modified with permission from Lowe N, Opensahw M, King TL. Labor. In: Brucker MC, King TL, eds. *Pharmacology for women's health*. Burlington, MA: Jones & Bartlett Learning; 2017:1088[64]; WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. *Lancet*. 2017. pii: S0140-6736(17)30638-4.[65]

Appendix 30E lists the steps recommended for managing a postpartum hemorrhage, and **Appendix 30F** reviews the steps taken to explore the intrauterine cavity if needed.[67-69] Standard procedures that are practiced in simulations or rehearsals can improve the response time when a real postpartum hemorrhage occurs.[57,59] The WHO recommends the development of formal protocols for prevention and treatment of postpartum hemorrhage,[9] although no specific protocol has been established as best practice.

Many sources delineate the sequential steps the midwife should proceed through when

managing a postpartum hemorrhage.[58,59] In addition, the midwife can find some excellent flowcharts that institutions use to guide management of a postpartum hemorrhage. Evidence-based flow charts and pocket algorithms that can be used to help manage obstetric hemorrhage and improve outcomes are available and examples are listed in the Resources section at the end of this chapter.[39]

A combination of bimanual compression and uterotonic drugs controls bleeding in the vast majority of women with postpartum hemorrhage. If management steps are initiated rapidly, excessive blood loss can be avoided. An interprofessional team should be called to address the emergency if the hemorrhage continues despite bimanual compression and use of uterotonic drugs, or if the woman has signs of hypovolemia. The use of agreed-upon vital sign "triggers" that signal the need for more aggressive treatment can help standardize management of postpartum hemorrhage and minimize morbidity. For example, triggers that could signal the need to initiate a hemorrhage protocol could be maternal pulse more than 110 bpm, estimated blood loss more than 500 mL, or systolic blood pressure less than 85 mm Hg. The team that responds to such emergencies commonly includes an anesthesia provider, an obstetric physician, and additional nursing staff. Once the hemorrhage is controlled, close postpartum surveillance is maintained until bleeding is minimal and the woman is stable.

Hematoma

Hematomas are the result of damage to blood vessels. When they occur in association with childbirth, they are most likely to become apparent during the intrapartum or postpartum periods. The most common locations for hematomas are (1) the vulva, (2) the vagina, and (3) the retroperitoneum.[69] The types of hematomas and their initial presentation are described in more detail in the *Genital Tract Injury: Immediate Postpartum Inspection of the Vulva, Perineum, Vagina, and Cervix* appendix in the *Second Stage of Labor and Birth* chapter and management is reviewed in the *Postpartum Complications* chapter.

Hematomas are very painful in women who do not have epidural analgesia, such that pain is the cardinal sign of a developing growth.[69] Women who have a functioning epidural may report sudden rectal pressure. In addition to pain, other clinical indicators of hematoma include signs and symptoms of blood loss such as hypotension or tachycardia.

Because a hematoma may not be visible externally, a gentle internal digital examination is necessary to establish the diagnosis. If the midwife finds a firm, rounded swelling into the vaginal canal or on the vulva that is small and not expanding quickly, it can probably be managed expectantly with frequent observation to make sure it is not expanding, placement of cold packs, and establishing intravenous access in case volume resuscitation is needed (**Table 30-9**). A Foley catheter may be needed to aid urination and a complete blood count to establish a baseline may be indicated. The midwife should communicate to the woman and caregivers (nurses in the hospital or family members if out of the hospital) the importance of reporting increasing pain or swelling.

Table 30-9	**Genital Tract Hematoma: Expectant Management**

Management	Rationale
Frequent assessment	The frequency will depend on when the hematoma is diagnosed, whether it is changing, and how much maternal discomfort is present. Assessments may be spaced out once the hematoma is determined to be stable.
Ice packs to the affected area for 24–48 hours	Ice packs assist with vasoconstriction, thereby decreasing bleeding and edema.
Offer pharmacologic pain management	Even minor hematomas may be uncomfortable. Nonsteroidal anti-inflammatory drugs (NSAIDs) should be adequate for minor hematoma pain, but opioids may be required for larger hematomas.
Encourage side-lying positioning	A side-lying position may relieve pressure and decrease pain. This is often an effective position for assessment and diagnosis of a hematoma. Positioning does not alter the hematoma progression.

If the pain is severe, the hematoma is increasing rapidly, or signs of hypovolemia develop, emergency measures are needed. If the woman is in an out-of-hospital setting, emergency transport is begun. The midwife's consulting physician and anesthesia staff should be notified and immediate assistance requested. The midwife should not incise the hematoma without additional aid because of the potential for hemorrhage. Measures to initiate intravenous fluid volume replacement and possible blood transfusion should be initiated while waiting for the healthcare team.

Rare Emergency Complications

If a newly postpartum woman experiences respiratory impairment, cardiovascular collapse, altered mental status, or loss of consciousness, emergency measures are indicated. The differential diagnoses include pulmonary embolus, anaphylactoid syndrome of pregnancy (i.e., amniotic fluid embolus), septic shock, peripartum cardiomyopathy, and atypical eclampsia, among others. Sudden and rapid deterioration of the woman necessitates a quick response. Initial management steps for any maternal emergency are listed in **Appendix 30G**.[70,71]

Life support measures focusing on adequate oxygenation are important regardless of the cause of the emergency. Once the emergency team is in place, the midwife plays an important role in communicating the past history, including the labor and birth; reviewing how the emergency presented; and identifying which management steps were implemented. Finally, maintaining communication with the family members to keep them informed is a critical component of the management of any maternal emergency that a midwife can perform.

Conclusion

The third stage of labor is critical for a healthy start for the new family. Attention to prevention and early identification and treatment of maternal complications can mitigate potential long-term effects. Witnessing the welcome of a newborn into a family is a sweet reward for the midwife. No matter how hard the midwife may have worked to assist the woman and her significant others through the labor and birth, complimenting the woman and stressing her accomplishment is a gift the midwife can offer in return.

Resources

Organization	Description	Webpage
California Maternal Quality Care Collaborative (CMQCC)	Collaborative of researchers and stakeholder organizations that analyzes data and quality improvement initiatives to end preventable morbidity, mortality, and racial disparities in maternity care in California. This organization is a leader in the publication of guidelines for managing postpartum hemorrhage, preventing cesarean birth, and treatment of preeclampsia. The website has multiple resources, slide sets, toolkits, and flow charts for managing postpartum hemorrhage.	https://www.cmqcc.org
Council on Patient Safety in Women's Health Care	Collaborative of national professional organizations that works to improve safe care through multidisciplinary collaboration. Teams in this organization create and disseminate safety bundles.	http://safehealthcareforeverywoman.org/patient-safety-bundles/obstetric-hemorrhage/

References

1. GBD 2015 Maternal Mortality Collaborators. Global, regional and national levels of maternal mortality, 1990–2015: a systematic analysis of the Global Burden of Disease study 2015. *Lancet*. 2016;388:1775-1812.
2. Moore ER, Bergman N, Anderson GC, Medley N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;11:CD003519. doi:10.1002/14651858.CD003519.pub4.
3. Combs CA, Laros RK. Prolonged third stage of labor: morbidity and risk factors. *Obstet Gynecol*. 1991;77:863-867.
4. Frolova AI, Stout MJ, Tuuli MG, López JD, Macones GA, Cahill AG. Duration of the third stage of labor and risk of postpartum hemorrhage. *Obstet Gynecol*. 2016;127:951-956.
5. Magann EF, Evans S, Chauhan SP, Lanneau G, Fisk AD, Morrison JC. The length of the third stage of labor and the risk of postpartum hemorrhage. *Obstet Gynecol*. 2005;105:290-293.
6. Abdel-Aleem H, Abdel-Aleem MA, Shaaban OM. Tocolysis for management of retained placenta. *Cochrane Database Syst Rev*. 2011;1:CD007708. doi:10.1002/14651858.CD007708.pub2.
7. Soltani H, Hutchon DR, Poulose TA. Timing of prophylactic uterotonics for the third stage of labour after vaginal birth. *Cochrane Database Syst Rev*. 2010;8:CD006173. doi:10.1002/14651858.CD006173.pub2.
8. Dombrowski MP, Bottoms SF, Saleh AA, Hurd WW, Romero R. Third stage of labor: analysis of duration and clinical practice. *Am J Obstet Gynecol*. 1995;172:1279-1284.
9. World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. 2012. Available at: http://apps.who.int/iris/bitstream/10665/75411/1/9789241548502_eng.pdf. Accessed January 28, 2017.
10. Schorn MN. Uterine activity during the third stage of labor. *J Midwifery Womens Health*. 2012;57:151-155.
11. Begley CM, Gyte GML, Devane D, McGuire W, Weeks A. Active versus expectant management for women in the third stage of labour. *Cochrane Database Syst Rev*. 2015;3:D007412. doi:10.1002/14651858.CD007412.pub4.
12. Erickson E. Role of prophylactic oxytocin in the third stage labor: physiologic versus pharmacologically influenced labor an birth. *J Midwifery Womens Health*. 2017. 62(4):418-424.
13. Fahy K, Hastie C, Bisits A, Marsh C, Smith L, Saxton A. Holistic physiological care compared with active management o the third stage of labour for women at low risk of postpartum haemorrhage: a cohort study. *Women Birth*. 2010;23:146-152.
14. Schorn MN, Minnick A, Donaghey B. An exploration of how midwives and physicians manage the third stage of labor in the United States. *J Midwifery Womens Health*. 2015;60:187-198.
15. Tan WM, Klein MC, Saxell L, Shirkoohy SE, Asrat G. How do physicians and midwives manage the third stage of labor? *Birth*. 2008;35:220-229.
16. International Confederation of Midwives, International Federation of Gynecology and Obstetrics. Joint statement: management of the third stage of labour to prevent post-partum hemorrhage. Available at: http://www.internationalmidwives.org/assets/uploads/documents/FIGO/PPH%20Joint%20Statement.pdf. Accessed January 28, 2017.
17. Schorn MN, Dietrich MS, Donaghey B, Minnick AF. US physician and midwife adherence to active management of the third stage of labor international recommendations. *J Midwifery Womens Health*. 2017;62:58-67.
18. Winter C, Macfarlane A, Deneux-Tharaux C, et al. Variations in policies for management of the third stage of labour and the immediate management of postpartum haemorrhage in Europe. *BJOG*. 2007;114:845-854.
19. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on materna and neonatal outcomes. *Evid-Based Child Health*. 2014;9:303-397. doi:10.1002/ebch.1971.
20. Andersson O, Hellström-Westas L, Andersson D, Domellöf M. Effect of delayed versus early umbilical cord clamping on neonatal outcomes and iron status at 4 months: a randomised controlled trial. *BMJ*. 2011;343:d7156.
21. Hutton EK, Hassan ES. Late vs early clamping of the umbilical cord in full-term neonates: systematic review and meta-analysis of controlled trials. *JAMA*. 2007;297:1241-1252.
22. Gülmezoglu AM, Lumbiganon P, Landoulsi S, et al. Active management of the third stage of labour with and without controlled cord traction: a randomized, controlled, non-inferiority trial. *Lancet*. 2012;379:1721-1727.
23. Westhoff G, Cotter AM, Tolosa JE. Prophylactic oxytocin for the third stage of labour to prevent postpartum haemorrhage *Cochrane Database Syst Rev*. 2013;10:CD001808. doi:10.1002/14651858.CD001808.pub2.
24. Peña-Martí GE, Comunián-Carrasco G. Fundal pressure versus controlled cord traction as part of the active management of the third stage of labour. *Cochrane Database Syst Rev*. 2007;4:CD005462. doi:10.1002/14651858.CD005462.pub2.
25. Hofmeyr GJ, Mshweshwe NT, Gülmezoglu AM. Controlled cord traction for the third stage of labour. *Cochrane Database Syst Rev*. 2015;1:CD008020. doi:10.1002/14651858.CD008020.pub2.
26. Afaifel N, Weeks AD. Active management of the third stage of labor: oxytocin is all you need. *BMJ*. 2012;345:e4546.
27. Soltani H, Hutchon DR, Poulose TA. Timing of prophylactic uterotonics for the third stage of labour after vaginal birth. *Cochrane Database Syst Rev*. 2010;8:CD006173. doi:10.1002/14651858.CD006173.pub2.
28. Gizzo S, Patrelli TS, Gangi SD, et al. Which uterotonic is better to prevent the postpartum hemorrhage? Latest news in terms of clinical efficacy, side effects, and contraindications: a systematic review. *Reprod Sci*. 2013;20:1011-1019.

29. Mori R, Nardin JM, Yamamoto N, Carroli G, Weeks A. Umbilical vein injection for the routine management of third stage of labour. *Cochrane Library*. January 1, 2012. PMID: 22419311. doi:10.1002/14651858.CD006176.pub2.
30. Yaju Y, Kataoka Y, Eto H, Horiuchi S, Mori R. Prophylactic interventions after delivery of placenta for reducing bleeding during the postnatal period. *Cochrane Database Syst Rev*. 2013;11:CD009328.
31. Jackson KW, Albert JR, Schemmer GK, Elliot M, Humphrey A, Taylor J. A randomized controlled trial comparing oxytoci administration before and after placental delivery in the prevention of postpartum hemorrhage. *Am J Obstet Gynecol*. 2001;185(4):873-877.
32. Soltani H, Poulose TA, Hutchon DR. Placental cord drainage after vaginal delivery as part of the management of the third stage of labour. *Cochrane Database Syst Rev*. 2011;9:CD004665. doi:10.1002/14651858.CD004665.pub3.
33. Abdel-Aleem H, Singata M, Abdel-Aleem M, Mshweshwe N, Williams X, Hofmeyr GJ. Uterine massage to reduce postpartum hemorrhage after vaginal delivery. *Int J Gynaecol Obstet*. 2010;111:32-36.
34. Hofmeyr GJ, Abdel-Aleem H, Abdel-Aleem MA. Uterine massage for preventing postpartum haemorrhage. *Cochrane Database Syst Rev*. 2013;7:CD006431. doi:10.1002/14651858.CD006431.pub3.
35. Schorn MN. Measurement of blood loss: review of the literature. *J Midwifery Womens Health*. 2010;55:20-27.
36. Al Kadri HM, Al Anazi BK, Tamim HM. Visual estimation versus gravimetric measurement of postpartum blood loss: a prospective cohort study. *Arch Gynecol Obstet*. 2011;283:1207-1213.
37. Loverro MT, Mastrolia SA. The more you lose the more you miss: accuracy of postpartum blood loss visual estimation: a systematic review of the literature. *J Matern Fetal Neonatal Med*. December 16, 2016:1-26.
38. Gabel KT, Weeber, TA. Measuring and communicating blood loss during obstetric hemorrhage. *JOGNN*. 2012;41:551-558
39. Lyndon A, Lagrew D, Shields L, Melsop K, Bingham B, Main E, eds. *Improving health care response to obstetric hemorrhage: California Maternal Quality Care Collaborative toolkit to transform maternity care*. Version 2. California Maternal Quality Care Collaborative; July 2010. Available at: https://www.cmqcc.org/resource/obstetric-hemorrhage-20-toolkit. Accessed June 12, 2017.
40. Buckley SJ. Placenta rituals and folklore from around the world. *Midwifery Today Int Midwife*. 2006;80:58-59.
41. Burns E. More than clinical waste? Placenta rituals among Australian home-birthing women. *J Perinat Educ*. 2014;23:41-49.
42. Cole M. Placenta medicine as a galactogogue: tradition or trend?. *Clin Lact*. 2014;5:116-122.
43. Marraccini ME, Gorman KS. Exploring placentophagy in humans: problems and recommendations. *J Midwifery Womens Health*. 2015;60:371-379.
44. Langston C, Kaplan C, Macpherson T, et al. Practice guideline for examination of the placenta: developed by the Placenta Pathology Practice Guideline Development Task Force of the College of American Pathologists. *Arch Pathol Lab Med*. 1997;121:449-476.
45. Miller ES, Minturn L, Linn R, Weese-Mayer DE, Ernst LM. Stillbirth evaluation: a stepwise assessment of placental pathology and autopsy. *Am J Obstet Gynecol*. 2016;214:115.e1-115.e6.
46. Redline RW. Classification of placental lesions. *Am J Obstet Gynecol*. 2015;213(4 suppl):S21-S28.
47. Chang KT. Examination of the placenta: medico-legal implications. *Semin Fetal Neonatal Med*. 2014;19(5):279-284.
48. Curtin WM, Krauss S, Metlay LA, Katzman PJ. Pathologic examination of the placenta and observed practice. *Obstet Gynecol*. 2007;109:35-41.
49. Magann EF, Doherty DA, Briery CM, Niederhauser A, Chauhan SP, Morrison JC. Obstetric characteristics for a prolonged third stage of labor and risk for postpartum hemorrhage. *Gynecol Obstet Invest*. 2008;65:201-205.
50. Weeks AD. The retained placenta. *Best Pract Res Clin Obstet Gynaecol*. 2008;22:1103.
51. Combs CA, Laros RK Jr. Prolonged third stage of labor: morbidity and risk factors. *Obstet Gynecol*. 1991;77:863-867.
52. Coad SL, Dahlgren LKS, Hutcheon JA. Risks and consequences of puerperal uterine inversion in the United States, 2004 through 2013. *Am J Obstet Gynecol*. 2017. [Epub ahead of print]. doi:10.1016/j.ajog.2017.05.018.
53. Oyelese Y, Smulian JC. Placenta previa, placenta accreta, and vasa previa. *Obstet Gynecol*. 2006;107:927-941.
54. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 76: postpartum hemorrhage. *Obstet Gynecol*. 2006;108:1039-1047.
55. Menard MK, Main EK, Currigan SM. Executive summary of the reVITALize initiative. *Obstet Gynecol*. 2014;124:150-153.
56. Kramer MS, Berg C, Abenhaim H, et al. Incidence, risk factors, and temporal trends in severe postpartum hemorrhage. *Am J Obstet Gynecol*. 2013;209(449):449.e1-449.e7.
57. Bingham D, Jones R. Maternal death from obstetric hemorrhage. *JOGNN*. 2012;41:531.9.
58. Main EK, Cape V, Abreo A, et al. Reduction of severe maternal morbidity from hemorrhage using a state perinatal quality collaborative. *Am J Obstet Gynecol*. 2017;216: 298.e1-298.e11.
59. Arafeh J, Gregory K, Main E, Lyndon A. Definition, early recognition and rapid response using triggers: CMQCC obstetric hemorrhage toolkit. 2015. Available at: https://www.cmqcc.org/resource/ob-hem-definition-early-recognition-and-rapid-response-using-triggers. Accessed June 12, 2017.
60. Merriam AA, Wright JD, Siddiq Z, et al. Risk for postpartum hemorrhage, transfusion, and hemorrhage-related morbidity a

low, moderate, and high volume hospitals. *J Matern Fetal Neonatal Med*. April 3, 2017:1-10.
61. Rani PR, Begum J. Recent advances in the management of major postpartum hemorrhage: a review. *J Clin Diagnost Re* 2017;11(2): QE01-QE05. doi:10.7860/JCDR/2017/22659.9463.
62. Evensen A, Anderson JM, Fontaine P. Postpartum hemorrhage: prevention and treatment. *Am Fam Physician*. 2017;95(7):442-449.
63. Karaca Z, Laway BA, Dokmetas HAS, Atmaca H, Kelestimur F. Sheehan syndrome. *Nat Rev Dis Primers*. 2016;2:16092. doi:10.1038/nrdp.2016.92.
64. Lowe N, Opensahw M, King TL. Labor. In: Brucker MC, King TL, eds. *Pharmacology for women's health*. Burlington, MA: Jones & Bartlett Learning; 2017:1088.
65. WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. *Lancet*. 2017. pii: S0140-6736(17)30638-4.
66. Vallera C, Choi LO, Cha CM, Hong RW. Uterotonic medications: oxytocin, methylergonovine, carboprost, misoprostol. *Anesthesiol Clin*. 2017;35:207-219.
67. Anderson JM, Etches D. Prevention and management of postpartum hemorrhage. *Am Fam Physician*. 2007;75:875-882.
68. Maughan KL, Heim SW, Galazka SS. Preventing postpartum hemorrhage: managing the third stage of labor. *Am Fam Physician*. 2006;73:1025-1028.
69. Rajan PV, Wing DA. Postpartum hemorrhage: evidence-based medical interventions for prevention and treatment. *Clin Obstet Gynecol*. 2010;53:165-181.
70. Mirza FG, Gaddipati S. Obstetric emergencies. *Semin Perinatol*. 2009;33:97-103.
71. Dubbs SB, Tewelde SZ. Cardiovascular catastrophes in the obstetric population. *Emerg Med Clin North Am*. 2015;33:483 500.

APPENDIX

30A

Management of the Third Stage of Labor

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History

- Previous birth complications during the third stage, including postpartum hemorrhage
- Risk factors for postpartum hemorrhage
- Course of this labor and birth
- Maternal requests for specific management in the third stage of labor

Physical Examination

- Maternal response to birth
- Vital signs stable
- Continuing evaluation of any previous significant findings
- Evaluation of new findings—for example, nausea is unusual in the third stage
- Evaluation of progress of the third stage, including signs of placental separation
- Continual assessment of bleeding

Management Steps

These steps include components of both physiologic management and active management of the third stage of labor. Individual situations may require alterations in the order of individual steps.

1. Maintain or reinstate a calm environment to reduce anxiety and facilitate management of normal progress and treatment of maternal complications if they occur.
2. Communicate the next steps and reassurance to the woman.

3. Check the newborn heart rate by palpating at the base of the umbilical cord and abdomen. If the heart rate is faster than 100 bpm, the newborn is breathing, and there are no obvious abnormalities, place the newborn in skin-to-skin contact on the woman's abdomen or arrange to have the newborn placed skin to skin with a person whom the woman designates for this initial period with the infant.
4. Continue to observe the neonatal transition to extrauterine life while the newborn is in skin-to-skin contact when possible. Note that the midwife conducting the birth has a primary responsibility to the woman during the third stage of labor, not management of the newborn.
5. Administer or request administration of a uterotonic agent, if not already given during birth of the anterior shoulder (optional depending on the woman's risk for postpartum hemorrhage, policies, and amount of bleeding present).
6. Consider upright positioning, which uses gravity to assist with placental expulsion if feasible.
7. If cord blood gases are not indicated, two clamps are needed:
    a. Clamp the cord with two clamps placed close together near the newborn's umbilicus after at least 1–3 minutes or when the cord stops pulsating.
    b. Cut the cord between the two clamps or guide the father, partner, other support person, or birthing woman to do this.
8. If cord blood gases are indicated, four clamps are needed:
    a. Place an initial clamp on the cord near the woman's introitus.
    b. Place two clamps in close approximation to each other on the cord distal (toward the newborn's umbilicus) to the first clamp.
    c. Cut the cord (or have another person cut it) between the last two clamps, and place the newborn on the maternal abdomen or hand off the newborn to another provider as indicated.
    d. Place a fourth clamp close to the first clamp that was placed near the woman's introitus, and cut the cord between these two last clamps. There is now a sealed double-clamped section of cord that can be used to obtain cord blood gases (**Figure 30A-1**).

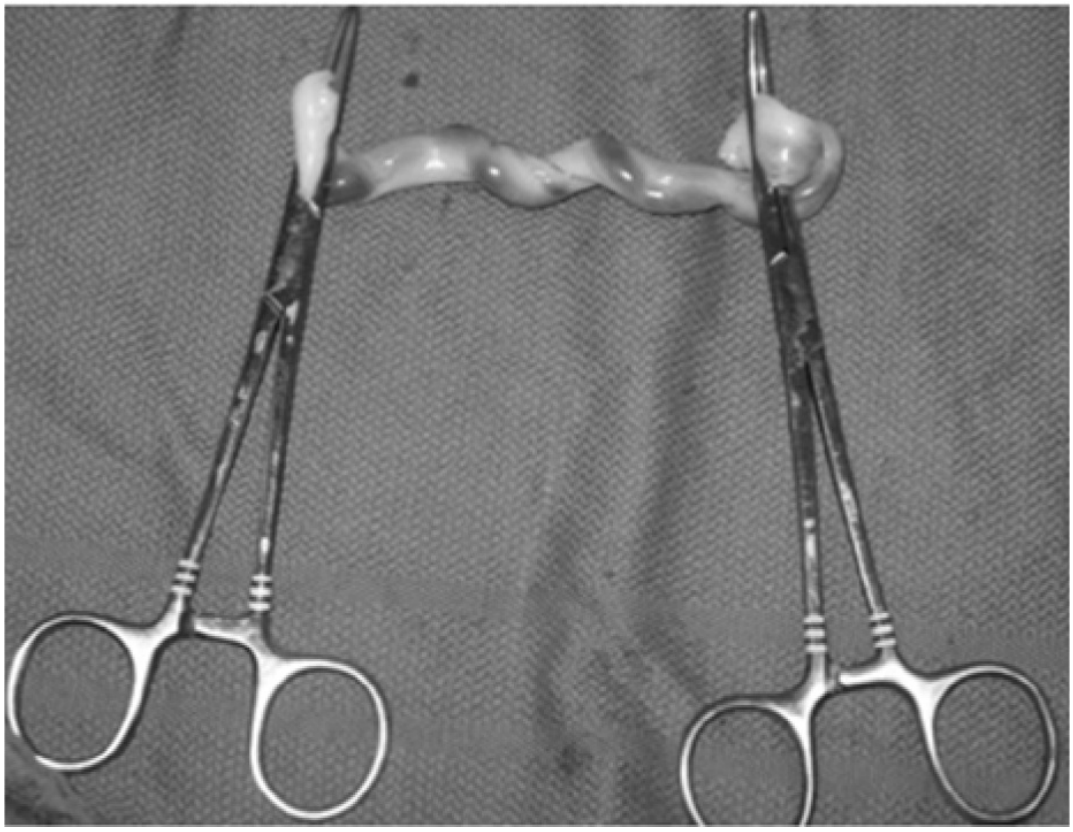


Figure 30A-1 Double-clamped cord.

Photograph courtesy of Tekoa L. King, CNM, MPH.

e. Obtain a sample of blood from the umbilical artery first, and then a sample from one of the umbilical veins, in a heparinized syringe, marking each syringe correctly as from the artery or the vein. Once the syringe is capped, the blood is stable for up to 30 minutes before the blood gas measurement has to be obtained.

9. Inspect the cut end of the umbilical cord and count the number of umbilical vessels.

10. Open the clamp nearest the woman's introitus and allow blood from the placenta to fill a non-heparinized blood collection tube. Approximately 5–10 mL is needed. This blood will be used to determine the type and Rh status of the newborn. Do not milk the cord, as tissue thromboplastin can interfere with the analysis.

11. Observe for signs of placental separation (**Table 30-1**) and do not massage the uterus before placental separation. Massaging the uterus prior to complete separation may increase uterine bleeding or lead to incomplete expulsion of the placenta.

12. Guard the uterus placing a hand suprapubically, anterior to the uterine fundus, to assess for uterine position in the abdomen and prevent uterine inversion as the placenta is expelled.

13. Determine whether the placenta has separated:

a. Use the Brandt–Andrews maneuver (**Figure 30A-2**). Apply slight downward pressure just above the pelvic brim with the hand that is guarding the uterus, while holding the cord taut with the other hand. If the placenta is still in the uterus, the cord will retract as the uterus is displaced upward into the abdomen by pressure from the

abdominal hand. If the placenta has separated and is in the vagina, upward displacement of the uterus will not cause the cord to retract, and additional cord traction can be safely provided while maintaining abdominal counter-traction.

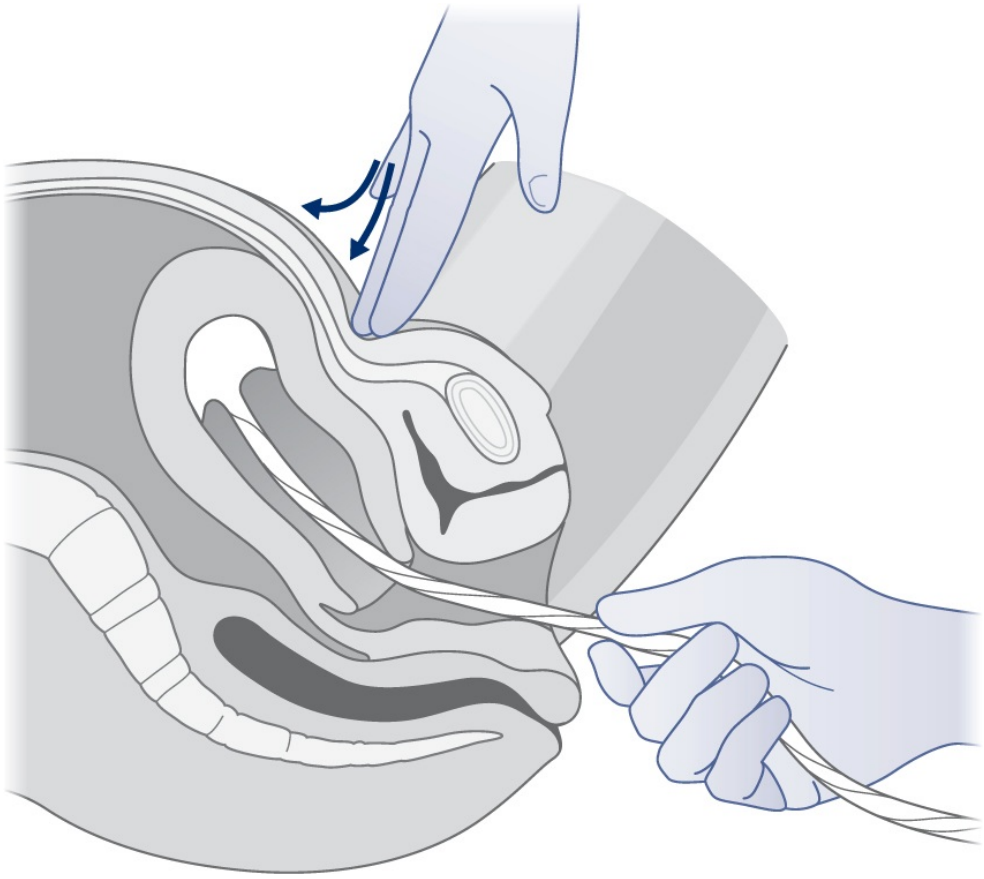


Figure 30A-2 Brandt–Andrews maneuver.

b. Another method of checking for placental location is to slide one finger (using sterile gloves) beside the umbilical cord into the vagina. If the placenta is in the vagina, it will be felt by the tip of the finger. The cord insertion into the placenta feels somewhat like an inverted umbrella. If the cord goes past the cervix, the placenta has not been expelled from the uterus. If the placenta is palpated in the vagina, additional traction can be safely performed.

14. Once the placenta has separated, the woman may spontaneously expel it by pushing. The midwife can also use a combination of the Brandt–Andrews maneuver and controlled cord traction.

15. Cord traction should *not* be performed without guarding the uterus, as this maneuver may result in an avulsed cord or inverted uterus.

16. Grasp the placenta with both hands as it is being expelled. Slowly guide the placenta out, perhaps following the curve of Carus to allow membranes to follow and not tear. If there appear to be more membranes than immediately are expelled, continue to hold the placenta and either twist it slowly or grasp the membranes with ring forceps to gently extract the membranes—a process commonly known as “teasing” out the membranes.

17. Palpate the fundus abdominally to assess if the fundus is firm. Routine vigorous massage to stimulate uterine contraction has not been shown to decrease the incidence of hemorrhage. Such massage may be indicated to express clots through the cervix if excessive bleeding is present, but it is not indicated if the estimated blood loss is normal.
18. Inspect the cord, placenta, and membranes as described in Appendix 30B (**Table 30-3).**
19. Show the placenta to the woman and family if interested.
20. Measure or weigh blood loss.
21. Weigh the placenta.
22. Send the placenta for pathology evaluation if indicated.

APPENDIX

30B

Inspection of the Umbilical Cord, Placenta, and Membranes

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Inspection of the umbilical cord, placenta, and membranes is performed as soon as the woman and her newborn are stable and comfortable. Any abnormal findings are documented in the maternal and neonatal records. In some situations, a photograph may be of value for documentation.

Inspection of the Umbilical Cord

Count the Number of Vessels

Wipe off the cut end with a gauze or towel if needed. Apply pressure; the apertures of the vessels will be visible. If vessels have collapsed, reclamp and recut the cord, then look for the vessels at the site of the new cut (**Figure 30B-1**).



Figure 30B-1 Counting the number of cord vessels.

Photograph courtesy of Carrie Klima, CNM, PhD.

Three vessels should be visible: two small arteries and one large vein. Any two-vessel cord should be submitted for pathology examination, and neonatal providers should be informed.

Measure the Length of the Cord

If the cord appears abnormally long or short, it should be measured. Remember to include the length of cord cut off the newborn's end when the cord was clamped and cut.

The normal length of a term umbilical cord is approximately 55 to 60 cm (range: 30–90 cm). A long cord, more than 75 cm, is associated with knots and fetal entanglement. Short cords, less than 32 cm, may be associated with limited fetal movement.

Inspect the Cord for Abnormalities

Look for knots, hematomas, tumors, cysts, edema, and the amount of Wharton's jelly (**Figure 30B-2**). The normal width of an umbilical cord is 1 to 2 cm. The width is affected by the amount of Wharton's jelly.



Figure 30B-2 True knot in the umbilical cord.

Photograph courtesy of Carrie Klima, CNM, PhD.

Inspect the Cord Insertion Site

Observe the insertion site for where the cord inserts in relation to the body of the placenta—central, eccentric, or marginal (Battledore placenta). Determine whether the cord is inserted directly into the placenta or is attached by exposed vessels (velamentous cord insertion) (**Figure 30B-3**). Centric and eccentric (off-center) insertions are variations of normal; however, velamentous and marginal insertions are unusual and associated with a risk of vasa previa should one of these vessels tear when the membranes rupture.



Figure 30B-3 Velamentous insertion of the umbilical cord.

Photograph courtesy of Tekoa L. King, CNM, MPH.

Inspection of the Placenta

Inspect the Placenta for Color, Shape, Size, and Consistency

Lay the placenta flat on a surface. Look at the fetal and maternal sides. If necessary, invert the membranes to see both surfaces.

The placental shape is usually round to slightly oval and approximately 2 to 3 cm in thickness. The fetal surface is smooth. The maternal surface is usually smooth with indentations at the edges of cotyledons, but may have gritty white areas of calcification. Generally, the quantity of calcifications has no clinical importance. A pale placenta is associated with immature neonates and anemia. With extended exposure to meconium, a placenta may be stained green.

Inspect the Smooth, Shiny Fetal Side of the Placenta

Inspect for distribution of vessels from the umbilical cord over the surface of the placenta. The fetal blood vessels traverse the surface of the fetal surface from the cord insertion toward the placental margin before turning into the parenchyma, toward the maternal surface. Inspect for cysts and to determine whether this is an extrachorial placenta (either a placenta circumvallata or a placenta marginata). If necessary, invert the membranes to visualize the entire fetal

surface. The fetal surface should also be examined closely for torn or intact blood vessels leading into the membranes so as to identify a missing or intact succenturiate (accessory) lobe.

Inspect the Maternal Side

Examine the placental margin for torn blood vessels leading into the membranes to look for evidence of a succenturiate lobe. A cotyledon missing from the main placental mass is identified by a defect in the placental mass with a rough surface where it tore away or a torn vessel at the edge of the placenta. This must be differentiated from a simple tear in the placenta without loss of tissue, which also leaves a rough surface. Differentiation is made by holding the placenta, maternal surface up, so that the cotyledons fall into place against each other (**Figure 30B-4**). A missing cotyledon is then evident because, as in a jigsaw puzzle, the surrounding pieces will not fit together smoothly. Observe for infarcts, cysts, tumors, and edema.



Figure 30B-4 Midwife examining the maternal side of the placenta. The placenta is intact.

Photograph courtesy of Carrie Klima, CNM, PhD.

Measure and Weigh the Placenta

Measuring and weighing the placenta are usually dictated by policy and are not always routinely performed. A term placenta usually weighs between 400 and 4600 grams. Regardless of the policy in place, if the placenta appears to be of abnormal size, weighing and referring for pathology examination are indicated. This information is then recorded in both the woman's and newborn's health record.

Inspection of the Membranes

Place the placenta maternal side down, and then place a hand inside the membranes on the fetal surface of the placenta and hold the membranes up to simulate the sac they once were (**Figure 30B-5**). If the membranes are ragged and do not form a sac, they may be incomplete. If a portion of the membranes is missing, membranes may still be within the uterus, although this is not a sensitive indicator of retained membranes.



Figure 30B-5 Midwife examining the fetal side of the placenta. The membranes are incomplete.

Photograph courtesy of Carrie Klima, CNM, PhD.

When inspection and documentation are complete, the placenta should be disposed of appropriately. In some cases, families may wish to keep the organ for culturally appropriate disposal, such as planting in their gardens. Others may desire to make impressions of it for a type of placental art. However, midwives should be aware that the placenta has bodily fluids and should be treated with universal precautions.

APPENDIX

30C

Manual Removal of the Placenta

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A retained placenta occurs in approximately 0.01% to 6.3% of women who have a vaginal birth.[1,2] Although there is no clear consensus on the length of time an unexpelled placenta warrants the diagnosis of retained placenta, the World Health Organization considers the placenta retained if it remains unexpelled longer than 30 minutes, but recommends waiting another 30 minutes before manually removing the placenta unless the woman is actively bleeding.[3] In the United States, the placenta is generally considered retained if not expelled at 30 minutes after the birth, and manual removal is recommended between 30 and 60 minutes if the woman is not actively bleeding.[2]

History

Retained placenta as defined by prolonged time.

Physical Examination

- Evaluation of progress of the third stage of labor, including signs of placental separation.
- Continual assessment of bleeding: Active bleeding may be apparent in the presence of an unexpelled placenta even if time since birth is not prolonged. Active bleeding requires prompt intervention.

Management Steps

1. Explain to the woman and others/family in attendance (with the woman's permission) the problem and the steps needed to resolve the problem.

2. Discuss options of pain management such as opioids, redosing of epidural, and/or nitrous oxide.
3. Alert the birth team (e.g., birth assistant, nurses) regarding the problem and plan for manual removal. Based on the birth site, another provider (e.g., physician, other midwife) with more experience may be immediately available and consulted as appropriate.
4. If an intravenous line is not already in place, insert one using a large-bore (at least 18-gauge) needle, in case a hemorrhage occurs and intravenous fluids are needed.
5. Verify that the newborn is being cared for appropriately. If the woman is holding the neonate, ask another responsible individual to hold the infant or place the newborn in a safe site.
6. Catheterize the woman's bladder as needed, as a full bladder may impede the effective uterine contractions needed after placental removal.
7. Change gloves or put on second pair so that the procedure is as sterile as possible. Gloves of a regular length can be used, but a longer length that overlaps a sterile gown may provide more protection from body fluids.
8. Reduce the diameter of the hand by squeezing the extended fingers close together, and gently insert one hand slowly and steadily through the vagina and past the cervix. The entire hand is inserted to facilitate the ability to perform the procedure. Once inserted, the hand should not be removed until the procedure is complete. Repeated insertion and removal of the hand increases pain and risk of infection for the woman.
9. Using the internal hand, sweep the margin of the placenta to find any area of separation. Once a point of separation is found, rotate the hand so the back of the hand is next to the uterine wall. This placement results in the hand being in a cup-like position and minimizes the risk of inadvertent uterine rupture.
10. Insinuate the fingers between the placenta and the uterus to establish a line of cleavage. Separate the placenta by sweeping back and forth from side to side, cutting through the decidua with the outer edge of the hand, fingertips, and first finger (**Figure 30C-1**).

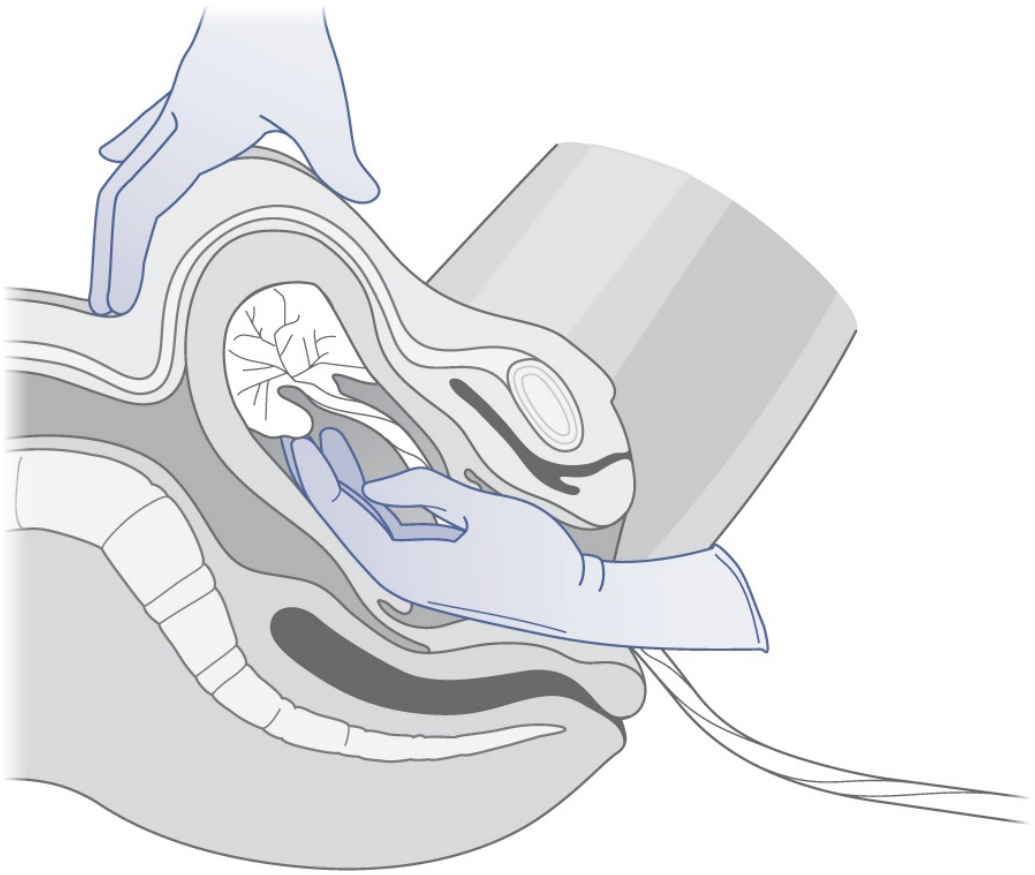


Figure 30C-1 Manual removal of the placenta.

11. Separate the entire placenta before attempting to extract it. If uncertain whether the placenta is totally separated, sweep the uterine wall again, feeling for and separating any remaining areas of attachment. Extraction while a section remains adherent increases the woman's risk of uterine inversion.
12. *Stop* the attempt to remove the placenta if it or its components are adherent. This finding is suggestive of a placental abnormality such as a placenta accreta. Immediate consultation/referral with a physician is required at this point.
13. Extract the placenta slowly after ascertaining that the placenta is not adherent; simultaneously continue to use the external hand to maintain fundal contraction.
14. Membranes may need to be slowly teased out, as described in Appendix 30A.
15. The placenta should be carefully inspected, as described in Appendix 30B. If the placenta does not appear complete, consider performing a uterine exploration to remove any retained tissue (Appendix 30F).
16. Maintain uterine contractility through uterine massage (as needed) and administration of a uterotonic agent. Continue close observation to rule out uterine atony.
17. After the placenta is removed:
    a. If the woman experienced excessive blood loss, assess her response by repeated laboratory evaluations, including hemoglobin or hematocrit values, and the woman's symptoms during the first 24 to 72 hours postpartum. Because tolerance of blood loss is contingent upon many factors, some women will be symptomatic (e.g.,

syncope) at higher levels than others. Management, including blood transfusions, will need to be individualized.

b. Antibiotic therapy is controversial and lacks strong evidence to either support or refute its use. Individualization is indicated, and consultation may be a reasonable action.

c. Document all procedures and the woman's response. Include consultations/referrals if appropriate.

References

1. Cheung WM, Hawkes A, Ibish S, Weeks AD. The retained placenta: historical and geographical rate variations. *J Obstet Gynaecol.* 2011;31:37-42.
2. Weeks AD. The retained placenta. *Best Pract Res Clin Obstet Gynaecol.* 2008;22:1103-1107.
3. World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. 2012. Available at: http://apps.who.int/iris/bitstream/10665/75411/1/9789241548502_eng.pdf. Accessed January 28, 2017.

APPENDIX

30D

Initial Management of Uterine Inversion

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A uterine inversion is an emergency. When a uterine inversion occurs, the risk for postpartum hemorrhage and disseminated intravascular coagulation (DIC) is high. The uterus may need to be repositioned when the woman is under general anesthesia. The most common symptom of uterine inversion is rapid maternal shock. Treatment for shock should be instituted immediately even before symptoms are evident.

1. Inform the nurse or other birth assistant of the diagnosis. Request emergency obstetrician and anesthesiology consultation and additional nursing support. Initiate emergency transfer steps if in an out-of-hospital setting.
2. Have a family member or someone else hold the newborn.
3. Explain to the woman and others in attendance that the uterus is inverted and briefly describe the steps needed to reposition the uterus.
4. Attempt to reposition the uterus immediately. Repositioning will be more successful if attempted as soon as the inversion is recognized and before the lower uterine segment and cervix contract to form a constriction ring.
    a. Do *not* remove the placenta if still attached. Removing the placenta while the uterus is inverted will increase the blood loss and increase the risk of complications.
    b. Manual repositioning is accomplished by placing one entire hand in the vagina.
        i. Initially place the fingertips on the sides of where the uterus has turned on itself so that the inverted fundus is in the palm of the hand (**Figure 30D-1**).

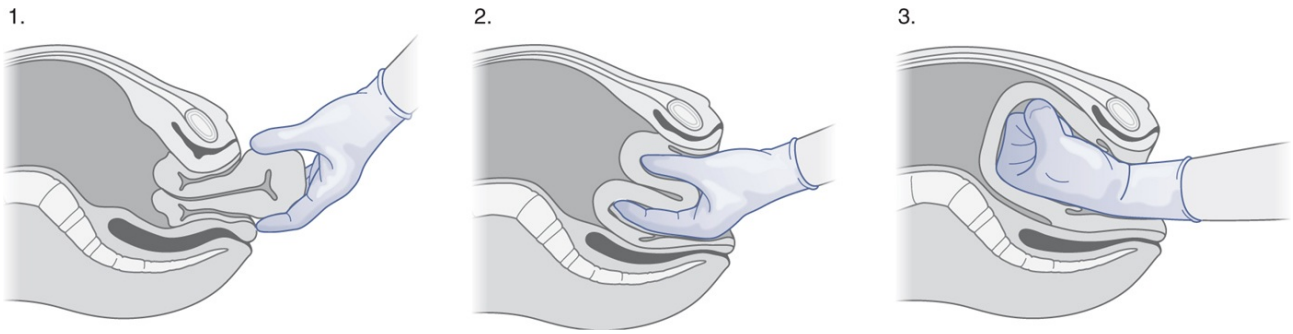


Figure 30D-1 Repositioning uterine inversion.

ii. Apply pressure with the palm of the hand on the fundus and with the fingertips on the uterine walls. Gently walk or slide the fingertips up the uterine walls so the fundus is folded back into its original position. Take care not to puncture or rupture the soft uterine wall.

c. Hold the fundus in place by forming the hand into a fist while waiting for the uterus to contract around the fisted hand. This puts tension on the uterine ligaments, which keeps the uterus in place, and the fist forms a ball that does not allow the fundus to fold in on itself.

d. Keep the hand in the uterus until a consulting physician is present and can take over the maneuver, even if it appears that the inversion has been corrected.

e. Use the abdominal hand to guard the uterus so it is lifted out of the pelvis, above the level of the symphysis, and hold in this position for several minutes.

5. Simultaneously:

a. Have the woman placed in Trendelenburg position.

b. Ask that an intravenous line be placed with a large-bore needle.

c. Discontinue uterotonics until the uterus is replaced.

d. Evaluate for signs of shock.

e. Request operating room and team preparation.

f. Request blood type and cross-match for 2 units of packed red blood cells, or initiate the postpartum hemorrhage protocol if one is in place.

g. Initiate quantitative evaluation of blood loss.

APPENDIX

30E

Management of Immediate Postpartum Hemorrhage

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Introduction

The management of a woman who is bleeding excessively during or immediately after the third stage of labor depends on the severity of bleeding, whether or not the placenta is expelled, and the available resources. The steps listed in this appendix take the reader through the process of assessing and managing a woman who has just expelled the placenta and whose bleeding is brisk but not life threatening. In practice, management will be customized for the woman and the situation. For example, if the woman is hemorrhaging and the uterus is not firm, bimanual compression may be the indicated first response. Conversely, if the bleeding is less severe, the clinician may check for lacerations before other steps are taken. Similarly, if the placenta is not expelled, the first step will be manual removal of the placenta. Multiple algorithms are available that outline recommended management steps for a woman who is hemorrhaging after birth. The reader is encouraged to know all possible management responses so that the response may be individualized as appropriate. Quantitative blood loss is the preferred method of determining the amount of blood lost during the third stage of labor and during/following a hemorrhage.

Management Steps

1. Maintain or reinstate a calm environment to reduce anxiety and facilitate management.
2. Palpate the woman's uterus, as approximately 80% of immediate postpartum hemorrhages occur secondary to uterine atony. External uterine massage may be the only intervention necessary.
3. If bleeding continues, proceed to the next steps.

4. Explain to the woman and others/family in attendance (with the woman's permission) that vaginal bleeding is excessive and explain the steps needed to resolve the problem.
5. Have a family member or other responsible adult hold the newborn. Alternatively, place the newborn in a safe site under observation.
6. Consult with other members of the healthcare team involved in the birth, including a physician if bleeding continues unabated. At this point, depending on the amount and rate of bleeding, ask for another practitioner to come to the bedside, or if in an out-of-hospital setting, initiate emergency transport.
7. Rule out bleeding from a laceration by efficiently performing an examination of the perineum, vulva, vagina, and cervix. Suture any active bleeding vessels appropriately.
8. If bleeding is not secondary to a laceration or the uterus is atonic, proceed to the next steps.
9. Catheterize the woman's bladder so a full bladder will not interfere with uterine contractility.
10. Order a uterotonic agent, if one was not administered earlier with release of the anterior shoulder of the newborn. Oxytocin (Pitocin) is the most commonly used agent, either 10–40 units in 1000 mL solution administered intravenously at a rapid rate (500 mL/hr), titrated to uterine tone, or 10 units given intramuscularly while intravenous access with a large-bore (usually 18-gauge) needle is initiated. If a uterotonic agent was administered with the birth of the neonate, consider a second agent (e.g., misoprostol [Cytotec], 15-methyl PG-F2a [Hemabate]). See **Table 30-8** for dose, route of administration, and side effects of uterotonic drugs used to treat postpartum hemorrhage. Continue administration of additional uterotonic agents to treat uterine atony.
11. If bleeding continues after the previous steps have been taken and the woman is in an out-of-hospital site, commence with an emergency transfer to a hospital. If the excessive bleeding is controlled quickly, the transfer can be cancelled.
12. If bleeding continues, initiate a second intravenous line with normal saline in preparation for blood transfusion and maintenance of intravascular volume.
13. Verify that a physician has been called; while waiting for emergency services arrival, proceed to the following steps.
14. Initiate *bimanual compression* (**Figure 30E-1**) of the woman's uterus to treat uterine atony.

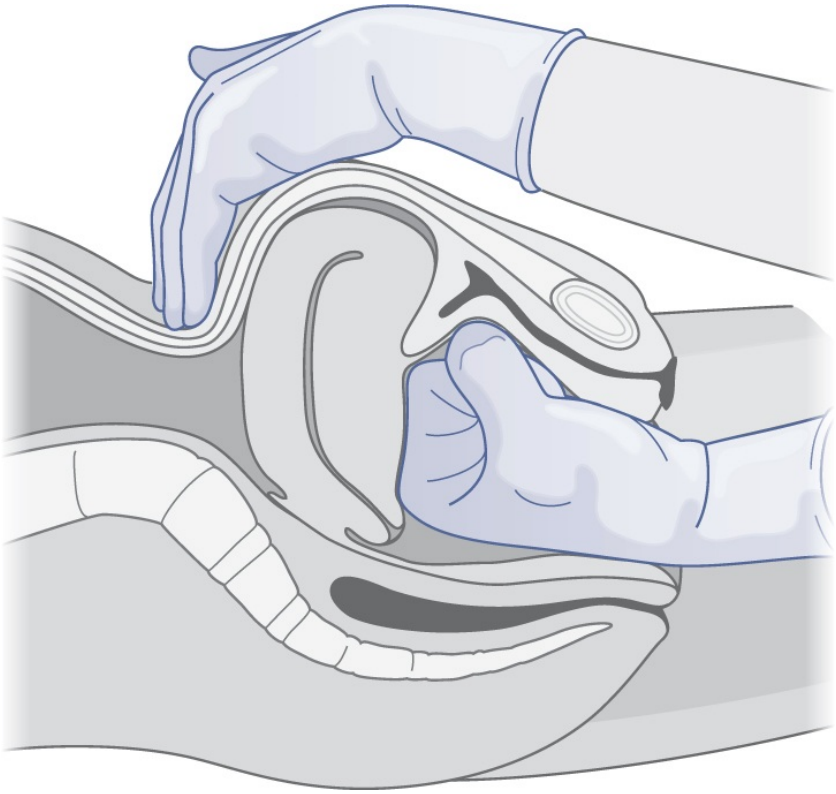


Figure 30E-1 Bimanual compression.

a. Extend the fingers of one hand and squeeze the extended fingers close together, gently insert this hand into the vagina, and close the fingers into a fist as the hand enters the vagina.
b. Position the fist palmar side up into the anterior fornix.
c. Direct pressure to the lower uterine segment/uterine corpus by applying pressure inward and upward against the anterior wall of the uterus.
d. Simultaneously, place the other hand externally on the abdomen and grasp the uterus between the two hands.
e. Apply pressure to the uterus trapped between the two hands by massaging it using pressure directed against the posterior wall of the uterus, primarily in the area of the fundus and corpus of the uterus with the abdominal hand. This compression places direct pressure on the bleeding uterine vessels while stimulating the uterus to contract, an action that will also provide continuous compression.
f. Continue bimanual compression until bleeding is controlled and uterine atony is resolved. The effectiveness of this intervention can be ascertained by momentarily releasing the pressure on the uterus and evaluating uterine consistency and bleeding at that moment.
g. Be aware that atony may seem to be resolved before it actually is, so bimanual compression should not be discontinued until the uterus is firm for several minutes. Removal of the hand and reinsertion increases pain/discomfort and risk of infection.
h. If bimanual compression provides a response but uterine atony does not resolve, continue compression until a physician is present.

15. If in a hospital, the blood bank should be notified and type and cross-match for 2 units prepared.
16. While waiting for a surgeon/obstetrician, engage other essential healthcare team members such as anesthesia personnel, nursing leadership, and other experienced providers available.
17. If retained products of conception are suspected to be the cause of the bleeding, proceed to performing an *intrauterine exploration*. Additional anesthesia or pain management methods may be needed at this point.
18. After bleeding has been controlled, discuss and explain the situation to the woman and her family, as the woman desires.
19. Continue to collect the blood lost and calculate the running total of loss.
20. Assess the woman's response to postpartum hemorrhage by repeat laboratory evaluations, including hemoglobin or hematocrit values, and the woman's symptoms during the first 24 to 72 hours postpartum. Because tolerance of blood loss is contingent upon many factors, some women will be symptomatic (e.g., syncope) at different hemoglobin levels than others. Management, including blood transfusions, will need to be individualized.
21. Antibiotic therapy following uterine exploration is controversial, lacking strong evidence to either support or refute its use. Individualization is indicated, and consultation may be a reasonable action.
22. Document all procedures and the woman's responses. Include any consultations/referrals if appropriate.

APPENDIX

30F

Intrauterine Exploration

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To perform an intrauterine exploration:

1. Change gloves or put on second pair so that the procedure is as sterile as possible. Gloves of a regular length can be used, but a longer length that overlaps a sterile gown provides more protection from body fluids.
2. Wrap one gauze sponge (e.g., 4 × 4) around two or four fingers, with the thumb stabilizing the gauze against the side of the first finger. The rougher surface of the gauze tends to be an effective modality to pick up placental fragments, ragged membranes, or blood clots. Use only one gauze so that it can easily be accounted for and to minimize the risk of leaving a gauze pad in the uterus or vagina.
3. Place the external hand on the woman's abdomen and grasp her uterus in a similar manner to that performed during bimanual compression, thereby stabilizing the organ.
4. Reduce the diameter of the internal hand by squeezing the extended fingers close together, and gently insert this hand slowly and steadily through the vagina and past the cervix. The entire hand is inserted to facilitate the ability to perform the procedure. Multiple insertions and removals of the hand increase pain and infection risk for the woman, so it is advisable to proceed carefully and intentionally.
5. Using the internal hand, sweep the inside of the uterus with the back of the hand while maintaining contact with the uterine wall so that the entire cavity is explored.
6. Remove the hand slowly from the uterus/vagina and unwrap, assess, and discard the gauze. If uncertain whether all the tissue was removed, repeat the procedure once for any remaining tissue or clots.
7. Maintain uterine contractility through uterine massage and administration of a uterotonic agent. Continue close observation to rule out uterine atony. An extended recovery period may be indicated.
8. Count the number of gauzes used and confirm that none remain in the vagina or uterus.
9. *Stop* the procedure at any time if portions of the placenta are felt to be adherent. This finding is suggestive of a placental abnormality such as a placenta accreta. Immediate

consultation/referral with a physician is required at this point, and preparation of the operating room team is indicated.

APPENDIX

30G

Sudden Postpartum Cardiovascular or Neurologic Emergencies

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Physical Examination

- Maternal consciousness
- Vital signs, including temperature
- Oxygenation (O_2 saturation)
- Skin color
- Evaluation of new findings (i.e., bleeding from unexpected sites)
- Continue monitoring uterine tone and vaginal bleeding

Management

- Initiate measures to obtain emergency assistance. If out of the hospital, implement the emergency transport protocol. If in the hospital, call for an emergency response team.
- Initiate a large-bore (18 gauge or larger) intravenous line if not already in place. If already in place, ensure patency, and consider placing a second line.
- Provide calm explanations to the woman and friends or family who are present at her birth.
- Assign someone to provide care to the neonate.
- Administer crystalloid, 0.9% sodium chloride, or lactated Ringer's solution rapidly if the woman is hypotensive.
- Apply oxygen at a 10–12 L/min flow rate via a nonrebreather face mask.
- Obtain blood pressure, pulse, temperature, and oxygen saturation levels frequently. If blood pressure is maintained, do not overload the woman with intravenous fluids—fluid overload can contribute to pulmonary edema.

- Be prepared to initiate cardiopulmonary resuscitation. Prepare for and assist with intubation and ventilation if needed.
- Position the woman in semi-Fowler's position.
- Order arterial blood gases, complete blood count, platelet count, fibrinogen, partial thromboplastin time, blood type and cross-match for 2 units of red blood cells, and chest X ray.
- Catheterize the bladder with an indwelling Foley catheter to drain to a urometer so output is monitored accurately.

CHAPTER

31

Birth in the Home and Birth Center

MARSHA E. JACKSON AND ALICE J. BAILES

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Introduction

Birth is profoundly affected by the environment in which it takes place. Ideally, every laboring woman and the team that supports and facilitates her efforts to birth will work together in an environment that is most comfortable and safe for the birthing woman. For many individuals, families, and providers, that safe, comfortable place to give birth is in the home or birth center.

The midwife who works in the home or birth center has an opportunity to develop the hands-on, low-tech skills that are the hallmarks of midwifery. Expertise in and fascination with normal birth make midwives keenly aware of how much they depend on the woman's natural abilities to complete the birth process herself. Although midwives in all settings finely tune their perception and interpersonal skills to amplify a person's abilities to birth, the opportunity to use these skills is increased in the home and birth center environments. The individuals who give birth in out-of-hospital settings are active in creating a network of support that enables them to optimize their health, and to give birth safely, effectively, and with great satisfaction.

As reviewed in the *History of Midwifery in the United States* chapter, midwifery originated in the home. Midwives later added the birth center concept. This chapter describes how the midwife works with those who choose home or birth center as the preferred birth site, and presents evidence-based guidelines for safe practice in these settings. As Varney explained in an earlier edition of this text:

> *If one were to place the control and responsibility assumed by the woman on a continuum, the continuum would stretch from the hospital delivery room at one end to home birth at the other. There are two important breaks in this continuum where power is transferred to the woman. The first break is when birth moves out of the hospital. The second break is when it moves from a freestanding out-of-hospital childbirth center to the home. In each instance the woman moves further away from external regulations and policies that are imposed on her childbirth experience.*[1(p536)]

Background

Until the mid-nineteenth century, healing arts in the United States were home-based, do-it-yourself types of assistance and included a variety of approaches.[2] Pregnant women in the United States anticipated giving birth at home, using their own power, without surgical or medical interventions. Surrounded by women attendants, they were the central figure during a social childbirth.[3,4] Attendants transmitted supportive midwifery skills, through direct empirical experience and apprenticeship.

As the twentieth century dawned, industrialization and urbanization had powerful effects in disrupting social organization and family life, and negatively affecting public health. State census officials began collecting maternal and infant mortality statistics and soon discovered that the U.S. data compared poorly to international data.[5] As midwifery became professionalized, midwives introduced education programs with home-based clinical practices and founded, owned, and staffed practices in both homes and birth centers. The data on the outcomes associated with these midwifery practices revealed good outcomes for midwife-attended births.[6] Nevertheless, a physician-led campaign that promoted physician care in hospitals for childbirth tipped public opinion toward birth in the hospital, where midwives were not permitted to practice.

By the late 1940s and early 1950s, most births in the United States occurred in hospitals. Pain medications sedated women during childbirth, and families were separated during and after the birth process. In the late 1950s and 1960s, a consumer backlash occurred, as many women and families rejected the hospital environment as too confining and disempowering. In turn, consumers, midwives, and some physicians began work to return birth to women's homes.[7-10]

The birth center concept was the culmination of an idea that had been evolving informally for many years. Some midwives may have preferred not to travel, or a laboring woman may not have had a suitable home in which to give birth. In such cases, midwives would designate a separate area of their home or a separate structure in which to attend births. Later, consumers who perceived hospital policies as too restrictive, yet did not want to give birth in their own homes, began to search for an alternative. Midwives were also in search of a place to attend physiologic births. The National Association of Childbearing Centers (NACC), now the American Association of Birth Centers (AABC), was established in 1983; this organization sought to create standards and promote a model facility integrated with the healthcare system, in which "low-tech, high-touch" care could be accessed.[11,12]

Some hospitals today advertise their labor and delivery suite as a birth center or as an alternative birthing center (ABC). These labels can be confusing, as they may be used to refer to both in-hospital and out-of-hospital centers. The birth centers discussed in this chapter are either freestanding facilities or, if within a hospital, are physically separate from the main hospital labor and delivery area. In most cases, they are administratively autonomous units and have specific policies and procedures for operating both the facility and the program for the childbearing families they serve.

Comparison of Home and Birth Center Settings

Both home and birth center practices share philosophical and administrative characteristics. The primary difference between home and birth center births is that birth in a birth center occurs within the professional's territory, to which the woman comes, whereas a home birth is within the woman's territory, to which the midwife goes.[1] Thus, the balance of power in each setting is different.

In the woman's home, the midwife and the assistant are the guests, and the woman is the host. Because the midwife is the one who travels to the woman, labor, birth, and postpartum care are not disturbed by travel that can cause intense discomfort to women in active labor or incur the risk of giving birth en route. When families give birth at home, there is little interruption in family routines for adults as well as for children, and bonding is uninterrupted. When the woman and the newborn are stable and remain undisturbed postpartum, the midwife and the assistant tuck the woman and her newborn into bed and quietly leave the home. Approximately 1% of women give birth at home in the United States. This percentage has been relatively stable since the 1960s, but has increased somewhat in the past 13 years.[13]

The birth center concept is a replicable model that has become a well-recognized choice for families. Birth centers provide an option for families who wish to avoid the hospital, but feel that home birth is not their birthplace of choice or is not available to them. According to the AABC:

> *The birth center is a homelike facility, existing within a healthcare system with a program of care designed in the wellness model of pregnancy and birth. Birth centers are guided by principles of prevention, sensitivity, safety, appropriate medical intervention, and cost-effectiveness. Birth centers provide family-centered care for healthy women before, during, and after normal pregnancy, labor, and birth.*[14](p1)

The Commission for the Accreditation of Birth Centers (CABC) provides a mechanism for external review of birth centers, which in turn gives guidance to professionals planning and practicing in birth centers.[15] Accreditation may enhance both family and practitioner confidence in the birth center choice. There is no comparable agency for home birth.

Safety During Birth in Home and Birth Center Settings

Essential Principles

To preserve safety at home and in the birth center, it is necessary to adhere to some essential principles:[16,17]

1. The woman must be committed to actively engaging in health promotion. When women are healthy and are experiencing normal pregnancy, they increase their chances for good outcomes for labor and birth. Some health problems preclude a home birth or birth center plan.
2. The place of birth must be planned before the onset of labor. Adequate planning for the place of birth includes determination that the woman's health and the pregnancy are within normal limits when labor begins and that family preparations for appropriate support are in place.
3. The attendant must be skilled and able to assess health status appropriately, provide vigilant care, and manage emergency complications should they occur. In addition to assisting with health promotion in the prenatal period, a midwife skilled in caring for women at home or in a birth center is vigilant in observation, communication, data collection, and care. This vigilance identifies both reassuring signs of normalcy and deviations from normal. If problems occur or persist at any time during the prenatal, intrapartum, or postpartum period, the midwife acts to correct the problem or, if necessary, works to stabilize the woman's condition and transfer her to hospital-based care.
4. There is a system in place to access consultation, hospitalization, and emergency transport. Clinical practice guidelines outline indications for consultation and a mechanism for transport to a hospital-based system. The midwife has established relationships with hospital-based practitioners to assure a smooth transition into the hospital should a transfer be indicated.

Outcomes of Birth in Home and Birth Centers

Data collection and research related to the safety and outcomes of home and birth center births have been conducted in many developed nations using varying research methods and populations.[18-21] Studies in the United States have demonstrated that planned home and birth center births for well women who are at term with a singleton fetus in vertex presentation, and who are experiencing a normal pregnancy, are at least as safe as—and, in some cases, safer than—their counterparts who give birth in the hospital.[22,23] Nonetheless, it is important to parse these data carefully. Analysis of this research is complex given the differing populations, risk status, provider type, and study methods included in the various investigations. A detailed bibliography of this body of evidence is included in the Resources section at the end of this chapter.

Maternal outcomes among well women who give birth at home or in a birth center are good, and generally better than the outcomes of women matched for demographic characteristics and

provider type who give birth in a hospital.[22-24] Rates of obstetric interventions and cesarean birth are lower for women who begin their labors planning to give birth in an out-of-hospital setting. Minimizing the use of technology may prevent some complications such as chorioamnionitis and increase the likelihood of normal birth; such technology is intentionally avoided during home and birth center births.[23]

The research findings with regard to newborn outcomes in home and birth center settings vary, and the clinical significance of these findings remains somewhat controversial. For example, studies of birth certificate data from the United States have found the rate of neonatal intensive care unit (NICU) admissions is lower for infants born at home, but the rates of Apgar scores of less than 7 at 5 minutes and early newborn seizures are higher.[22-24,25] Nevertheless, the absolute risk of newborn seizures is less than 1% in all settings; thus, comparing relative risks can convey a message of increased risk that is, in fact, not striking when absolute risks of rare outcomes are evaluated.[26]

Objective analysis of perinatal and neonatal mortality rates is also difficult. Some studies have reported perinatal mortality rates that include fetal deaths and deaths of infants to age 7 days, whereas others have used neonatal mortality rates that include only live births and measure deaths up to 28 days of life. Furthermore, because women with complications are transferred to a hospital and the intended birth site is not always known, newborn outcomes following hospital birth may capture some mother–newborn dyads for whom the family initially planned a home or birth center birth.

Two studies using data from women who planned a birth at home in the United States included an intention-to-treat analysis (i.e., outcome assigned on the basis of the intended site of birth so hospital transfers were included). Snowden et al. found the perinatal mortality rate (stillbirths and perinatal deaths) associated with home birth to be 3.9 per 1000 births.[25] Cheyney et al. reported an intrapartum death rate of 1.30 per 1000 births; early neonatal death rate of 0.41 per 1000 births; and the late neonatal death rate was 0.35 per 1000 births.[27] In the analysis by Snowden et al., the perinatal mortality rate among women with similar characteristics who gave birth in a hospital during the study period was 1.8 per 1000 births.[25] Aggregate data from birth centers in the United States have shown the intrapartum fetal mortality rate to be 0.47 per 1000 births and the neonatal mortality rate to be 0.40 per 1000 births for newborns who were born in a birth center.[23]

Overall, most studies of home birth in the United States and other developed nations have found a small but increased risk of adverse neonatal outcomes associated with home birth, primarily in nulliparous women, although the absolute increase in risk is quite low. The well-conducted, very large Birthplace in England study from the United Kingdom is a good example. For low-risk nulliparous women, home birth was associated with a small increased risk of a composite set of adverse neonatal outcomes: stillbirth after start of care in labor, early neonatal death, neonatal encephalopathy, meconium aspiration syndrome, brachial plexus injury, fractured humerus, or fractured clavicle. This composite measure found a rate of 9 adverse outcomes per 1000 newborns born at home versus 4 adverse outcomes per 1000 newborns born in hospital settings.[18]

In an effort to reach a consensus on the safety of birth outside of hospital settings,

professional organizations continue to discuss their conflicting positions. The American College of Nurse-Midwives (ACNM) states birth at home can be safe for essentially well women who experience an uncomplicated pregnancy and have assistance from a qualified provider.[28,29] The American College of Obstetricians and Gynecologists supports hospitals and birthing centers as the safest sites for birth, but state that their members respect women's right to make an informed choice about their planned birth site.[30] The World Health Organization's position is similar in recommending that women who are medically at low risk can choose home birth if they are attended by a skilled provider and have plans for transfer to a higher level of care if needed.[31]

Essential Elements of Birth Center and Home Birth

Table 31-1 outlines the essential elements for home and birth center births.

Table 31-1	Essential Elements of Birth Center and Home Births
Shared decision making in the client–midwife partnership	
A well-woman experiencing normal pregnancy and promoting her own health	
Clear recognition of the centrality of the birthing woman	
Increased authority for the birthing family and midwife	
Increased privacy available to the laboring woman	
Encouragement of family participation	
Optimal environment for physiologic birth	
Skilled personnel and equipment provided by the midwife	
Complex technologies deliberately kept at a distance	
Safety preserved through links within the community's healthcare system	
Independence in the midwifery practice	
Minimization of bureaucracy	
Home and birth center practices often owned by midwives	

Shared Decision Making

Shared decision making is an inherent component of midwifery care in general, but the process of shared decision making assumes an even more prominent role in the home or birth center setting. Early in the clinical relationship, the midwife introduces the concept of working in a client–midwife partnership, in which each party has a unique set of responsibilities. For more information about shared decision making, see the *Introduction to the Care of Women* chapter.

Authority, Centrality, and Privacy

Women have the right to own their births and to choose those with whom they wish to share their maternity experience. In the small and intimate environments provided in the home or birth center, it is easier for all to perceive the *authority* the woman has to make her choices and expect that her preferences will be honored. In this personalized milieu, distracting events are minimized, increasing the laboring individual's *centrality*. At home or in a birth center, the woman surrounds herself with only the people whom she chooses, meeting her needs for *privacy* or companionship.[32]

With the explicit recognition of her authority in the home and birth center settings, the laboring woman feels a sense of control over the birth's environment, management, and outcome. The woman's confidence in her providers' approach to care grows during the prenatal period, so the only surprises she will encounter in labor are the ones that labor brings. When all members focus only on the woman's centrality, the team may offer their skills, presence, and support for her all-important task of giving birth. They are not distracted as they

work to enhance the woman's comfort and help her use her energy efficiently. The woman's privacy is respected as she creates a team with as few as four persons (the woman and her birth partner, the midwife, and a second qualified practitioner/assistant) or as large as the home or birth center can accommodate.

At home or in a birth center, the environment communicates an expectation of normalcy and promotes physiologic birth. The woman's natural efforts are respected, and all who work with her are dedicated to the concept that birth is normal. It is expected that the woman has the ability to give birth, and that she can do so with physical, social, and psychological support and without the use of complex technology. She is not attached to equipment designed to do the job for her, so she is free to move around and to be in any position she chooses. A walk outdoors can invigorate her, occupy her attention, and promote labor progress. She can use hydrotherapy to promote relaxation. The private labor environment helps the women feel safe. She can make sounds without fear of being overheard by strangers close by. She has her own clothes to wear or not wear as she sees fit. Familiar food and drink that she and her family have provided are available to keep her well nourished and hydrated. Moreover, whether at home or in the birth center, women and their newborns are less likely to encounter pathogenic bacteria and invasive procedures, thereby reducing the risk of nosocomial infection.

In such a setting, emphasis is placed on labor as dynamic, progressing rapidly at some times and slowly at others. Ebbs and flows are normal. Encouraging activity to promote progress and quieting the environment to promote rest are simple tasks. In contrast, in the hospital environment there may be a need to make space available to accommodate other laboring women, so a woman may receive unnecessary interventions to speed labor. At home or in the birth center, attention can be focused on addressing the woman's needs, enabling her to progress or rest, expecting and accommodating plateaus of progress.

Client–Midwife Relationships

In every setting, the relationship between the midwife and the woman is vital. In the home and birth center, it is particularly important to specify the roles that each participant assumes as they share responsibility and work together in a partnership based on mutual trust. Women choose an attendant intentionally, searching for a good fit, and seeking a collaborative, nonhierarchical relationship. Goals and criteria for what is "normal" are clearly delineated. Collectively, along with the woman's support system, all team members share responsibility for both the process of care and the outcome. Working in this way, all promote clear and ongoing communication throughout the maternity cycle.

Midwives who choose home and birth center practice need a clear sense of their own strengths and limitations. They must be comfortable with sharing power and occupying a role that is not central. This requirement challenges midwives to sharpen their communication skills, to be open to discovering how to expand flexibility, and to know how to set and explain evidence-based limits and boundaries. When a woman plans to give birth in the home or the birth center, it is easier for her to see the connections between her actions and the outcomes. Her responsibilities for care are also clear, including recognition that her actions can make a difference.

Home and birth center midwives must be able to determine where philosophical gaps lie between themselves and their clients, and strategize to resolve them. These midwives must also feel comfortable taking a leadership role in the client–midwife partnership. Each midwife must be assertive as she describes the limits of her practice in the home or birth center environments.

Midwife–Consultant Relationships

If variations in the client's health state require consultation, collaboration, or referral, midwives need links to the community's healthcare system that they can activate to benefit the woman. Consultation may be provided by family physicians or obstetricians who are solo practitioners, a group practice, several different practices, or the house staff employed by the hospital. Some certified nurse-midwives (CNMs)/certified midwives (CMs) who attend home and birth center births maintain hospital privileges, facilitating seamless transfer into the hospital when necessary. Other home birth and birth center midwives elect not to have hospital privileges, but maintain consultation relationships with physicians or with hospital-based CNMs/CMs. For the midwife without hospital privileges, establishing a consultation agreement with hospital-based midwifery practices affords a means of access to the hospital, fostering a collegial relationship among midwives. Clients appreciate this arrangement because even if they transfer to the hospital, they still may have their birth attended by a midwife.

Before negotiations with a consulting practice take place, the midwife should write clinical practice guidelines and identify those situations for which consultation, collaboration, or referral would be needed. These guidelines are *not* a supervisory agreement. Many states have passed legislation repealing the requirement that midwives have written collaborative practice agreements with physicians or hospitals. Although writing guidelines is important, midwives and consultants must build a relationship based on open communication, mutual trust, and respect. Most consulting services do not see clients prenatally unless a problem is identified, but women may still want to meet with the consultant and should be encouraged to do so if desired.

Prenatal Care

During prenatal visits, the midwife works to enhance the woman's self-confidence and self-reliance. By giving reassurance regarding the normalcy of pregnancy's changes, and by communicating faith in her ability to give birth, the midwife assists the woman in amplifying her strengths and helps her to compensate for any limitations.

Introduction to the Practice

At the first prenatal visit, the midwife orients the family, introducing them to the services and philosophy of the practice. The midwife, the woman, and her family discuss their needs and explore how the woman envisions getting her needs met as a shared decision-making process. It can be a challenge to have these discussions while maintaining the family's confidence in the birth process. There should be ongoing communication and exploration of concerns throughout the maternity cycle. In this way, self-care is fostered, good decision making takes place, and the woman and her family can become empowered.[32-34] The midwife reviews the strong commitment to health promotion, to keeping birth normal, and to use of nonpharmacologic comfort measures for childbirth. This visit also provides an opportunity for both the woman and midwife to assess their abilities to work together. It is important that adequate time be allotted to cover all of these tasks.

Client Responsibilities During Pregnancy

As part of the introductory visit, the midwife provides an overview of the tasks that the woman will need to undertake to promote optimal health and to maximize the likelihood of having a normal pregnancy, with labor and birth occurring in the birth site she has chosen. Basic health promotion includes good nutrition, adequate rest and exercise, stress reduction, keeping scheduled appointments, and avoiding drugs, alcohol, and smoking. The woman's responsibilities also include assuring the participation of a support partner who will take primary responsibility to be available during pregnancy, labor and birth, and the postpartum period. When small children are to be present at the birth, an adult other than the primary support partner must be available to care for them.

Other responsibilities introduced during the initial visit will be addressed again later in the pregnancy, including breastfeeding preparation, attending childbirth education classes, touring the hospital or birth center, gathering birth supplies, and arranging for the services of a pediatric healthcare provider. Women may also be required to meet with the consultant service during the pregnancy, depending on the practice's guidelines.

Midwife Responsibilities During Pregnancy

The midwife's responsibilities are outlined during the initial visit to clarify the woman's expectations regarding the midwife's role. The midwife explains the concept of working in a client–midwife partnership, with each partner having a separate set of responsibilities. Throughout the maternity cycle, the midwife provides education, guidance, and health care,

with particular emphasis on signs of normalcy and signs of deviation from normal. The midwife makes 24-hour midwifery on-call services available to the woman. Finally, the midwife describes the referral and transport services available. Resources to cover remuneration to both the midwife and the consultant in case of transfer of the woman's care are also important factors to discuss.

Selection of the Birth Site

The advantages and disadvantages of all available birth site options are reviewed in detail during the first prenatal visit so the woman and the midwife can work together in considering the best birth setting, delineating what can and cannot be done in each site. When discussing birth site options, both positive and negative aspects of each site are explored. Fewer than 2% of women in the United States choose to give birth at home or in a birth center.[13] Thus, an understanding of the woman's motivation for her birth choice will enable the midwife and the woman to work together more effectively. The attitudes and concerns of the woman's support partner and/or family should also be included in this discussion.

Influences on choice of birth setting may include the philosophy of the woman, her support network, and the caregivers; preferences regarding technology; feelings about previous birth experiences; societal and family considerations; financial concerns; and the woman's health status.

Safety Criteria and Transfer to Hospital-Based Care

The issue of possible transfer of care if a complication occurs should be addressed at the initial visit and reviewed as needed during subsequent visits. These discussions include a review of the midwife–consultant relationships, hospital privileges, and planned sites for transfer if needed. Problems that can develop during pregnancy, labor, or birth, or in the immediate postpartum period, and their frequency of occurrence should be reviewed with the woman and her family. The importance of self-care and early intervention to promote health should be reemphasized. The midwife reviews the woman's health needs to assure the choice of an appropriate birth site, making the woman aware that some health conditions are best addressed in the hospital. **Table 31-2** lists conditions that may be present preconceptionally or may become evident during pregnancy, which suggest planning birth in a hospital setting is indicated.[28] **Table 31-3** identifies conditions that may develop during labor, birth, and the immediate postpartum period and require transfer to a hospital setting.[28]

Table 31-2 **Conditions That Are Indications for Planned Hospital Birth**[a,b]

Prior Pregnancy Conditions
Previous stillbirth or neonatal death related to an intrapartum event
Primary postpartum hemorrhage requiring additional procedures[c]
Prior cesarean birth
Shoulder dystocia with resulting injury

Current Pregnancy Conditions
Active preterm labor (before 37 0/7 weeks' gestation) or preterm, prelabor rupture of membranes
Essential or gestational hypertension[d]
Evidence of congenital fetal anomalies requiring immediate assessment and/or management by a neonatal specialist
Fetal growth restriction < 5th percentile
Insulin-dependent diabetes or gestational diabetes requiring pharmacologic management
Malpresentation: breech, transverse lie
Need for pharmacologic induction of labor
Post-term pregnancy > 41 6/7 weeks' gestation
Multifetal gestation
Oligohydramnios with additional complicating factors
Polyhydramnios
Placenta previa in the third trimester
Placental abruption
Preeclampsia[e]
Rh isoimmunization
Medical Conditions
Evidence of active infection with hepatitis, human immunodeficiency virus, genital herpes, syphilis, or tuberculosis
Psychiatric conditions that may affect intrapartum care management or maternal or neonatal transition following birth
Substantial medical conditions that have required acute medical supervision during the pregnancy and that could impact the birth, such as cardiac disease, epilepsy, thromboembolic disease, or hemoglobinopathy
Substance use/dependence

[a] This list is not exhaustive.

[b] Other obstetric or medical conditions may occur during pregnancy that warrant consultation, collaboration, or referral to determine the optimal site for the birth. Risk assessment for an individual woman may vary based on her prior medical, surgical, and obstetric history as well as resources available for hospital access within her community. Individual midwifery practice guidelines and/or client and/or midwife discretion will affect shared decision making about the selection of site of birth, and this process will be documented.

[c] Such as Bakri-balloon, dilatation and curettage, transfusion, and manual removal of placenta.

[d] Diagnosis of gestational hypertension is made following two blood pressure recordings > 140/90 mm Hg, taken 4 hours apart, after 20 weeks' gestation in a woman who was previously normotensive.[32]

[e] Diagnosis of preeclampsia is made following two blood pressure recordings > 140/90 mm Hg, taken 4 hours apart, after 20 weeks' gestation in a woman who was previously normotensive and has proteinuria. Dipstick reading of 1+ can be used if other techniques for detecting proteinuria are not available. Diagnosis can be made in minutes if blood pressure is 160/110 mm Hg or greater. Hypertension and thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema, and/or cerebral or visual symptoms may be used for the diagnosis in the absence of proteinuria.[32]

Reproduced with permission from American College of Nurse-Midwives. Clinical Bulletin No. 14: midwifery provision of home birth services. *J Midwifery Womens Health*. 2015;61(1):127-133.[28]

Table 31-3 **Intrapartum, Postpartum, and Newborn Conditions That Are Indications for Transfer from Home to a Hospital[a]**

Intrapartum Indications[b]
Malpresentation: breech, transverse lie identified during labor
Development of signs or symptoms of gestational hypertension[c] or preeclampsia[d]
Evidence of chorioamnionitis[e]
Evidence of fetal intolerance of labor or persistent Category II fetal heart tones[f] that are unresponsive to intrauterine resuscitation when birth is not imminent or in the presence of meconium
Need for pharmacologic augmentation of labor
Signs of placental abruption or unexplained increased vaginal bleeding
Postpartum Indications
Management of lacerations beyond the expertise of the attending midwife
Postpartum hemorrhage unresponsive to initial treatments
Retained placenta
Unexplained vaginal bleeding
Newborn Indications
Unstable health status

[a] Other obstetric or medical conditions may occur that warrant consultation, collaboration, or referral to determine the optimal site for the birth. Risk assessment for an individual woman may vary based on her prior medical, surgical, and obstetric history as well as resources available for hospital access within her community. Individual midwifery practice guidelines and/or client and/or midwife discretion will affect shared decision making about the selection of site of birth, and this process will be documented.

[b] When birth is imminent, careful consideration of the potential effect of transport on best practice management must be a priority consideration.

[c] Diagnosis of gestational hypertension is made following two blood pressure recordings > 140/90 mm Hg, taken 4 hours apart, after 20 weeks' gestation in a woman who was previously normotensive.[32]

[d] Diagnosis of preeclampsia is made following two blood pressure recordings > 140/90 mm Hg, taken 4 hours apart, after 20 weeks' gestation in a woman who was previously normotensive and has proteinuria. Dipstick reading of 1+ can be used if other techniques for detecting proteinuria are not available. Diagnosis can be made in minutes if blood pressure is 160/110 mm Hg or greater. Hypertension and thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema, and/or cerebral or visual symptoms may be used for the diagnosis in the absence of proteinuria.[32]

[e] Chorioamnionitis is defined clinically as maternal fever > 38°C (> 100.4°F) with maternal tachycardia (> 100 bpm) and/or fetal tachycardia (> 160 bpm).

[f] Guidelines for intermittent auscultation are detailed in American College of Nurse Midwives. Clinical Bulletin No. 13: intermittent auscultation for intrapartum fetal heart rate surveillance. *J Midwifery Womens Health*. 2015;60(5):626-632.

Reproduced with permission from American College of Nurse-Midwives. Clinical Bulletin No. 14. Midwifery provision of home birth services. *J Midwifery Womens Health*. 2015;61(1):127-133.[28]

The midwife's clinical judgment, skills, and resources influence decision making regarding the optimal birth setting for an individual woman. This assessment includes an evaluation of safety for the midwife. If the midwife feels the home is unsafe, the woman does not have documented prenatal care, or hospital resources are geographically too distant, a hospital setting should be recommended.

The midwife has ultimate responsibility for linking into the rest of the maternity care system,

even though some women arrange or lay the groundwork for consultation/hospital services.

Although urgent emergencies may occur in any birth setting, they are rare in the home and in the birth center.[23-25,27] Women need to explore their feelings regarding the possibility of a negative birth outcome regardless of their planned birth setting. They may not be ready to discuss this very sensitive issue in depth at the initial visit. Instead, introducing this subject during the first visit and revisiting it during subsequent prenatal visits is the best way to make sure this topic is fully explored.

Financial Considerations

Financial arrangements must be clear for both self-pay and insured clients, and discussed openly by all responsible parties. A written payment plan should be signed, which includes insurance information and a schedule for payments. Some women seek home and birth center services because they lack financial resources. The economically disadvantaged woman may benefit from the personalized, nonbureaucratic, respectful, one-to-one care associated with midwifery, which she may not encounter elsewhere in the healthcare system. As long as the woman can accomplish the needed preparation for the birth and the pregnancy remains normal, she is a good candidate for a home or birth center birth. Although an out-of-hospital birth may be less expensive if everything remains normal, it may be more expensive than a hospital birth if complications arise. To that end, women should be discouraged from seeking such services purely for financial reasons.

Initial Prenatal Visit

At the initial visit, it is especially important in home and birth center practice for the midwife to assess the client for predictors of normalcy and predictors of deviation from normal that might be evident in the woman's history and physical examination. Records from other healthcare providers should be requested if needed. When this information is obtained, an appropriate birth site may be selected. The woman and the midwife can identify factors that have the potential to develop into problems. Instituting prevention strategies early increases the likelihood that the woman will successfully give birth in her chosen birth site.

Calculation of an Accurate Due Date

Particular attention is paid to calculation of an accurate due date, because for most home and birth center practices there is a 5-week window of safety for the planned birth. A pregnancy is designated at full term from 37 weeks' gestation until 41 6/7 weeks' gestation. Births occurring at less than 37 weeks' gestation or more than 41 6/7 weeks' gestation are considered to be more safely accomplished in the hospital, but occasionally for women who are post-term, the window of safety can be expanded a few days with shared decision making, careful fetal surveillance, and appropriate consultation. When labor occurs before 37 weeks' gestation, the fetal lungs may not be adequately mature. In such a case, the woman whose preterm labor cannot be stopped is referred to the hospital, where staff and technology can give appropriate support to the newborn if needed. Likewise, women who begin labor after 41 6/7 weeks of

pregnancy are at increased risk for meconium staining and variant fetal heart rate patterns during labor, both of which have been implicated as causes of morbidity and mortality.[35]

Return Visits

During each return visit, the woman and the midwife identify reassuring signs of normalcy or identify problems through an interim history, physical examination, and laboratory and adjunctive studies. In the role of educator, the home and birth center midwife teaches the woman about symptoms that could indicate a deviation from normal. In this way, the woman is enabled to continue to take a principal role in early identification of problems, allowing for early intervention. For a woman to remain a candidate for a home or birth center birth, it is especially vital to intervene early, before uncorrectable problems develop. Women should know that if they encounter specific complications, they will lose their opportunity to have the birth they had planned—this knowledge can be a powerful motivator for women to engage in health promotion.

The Family's Preparation for Birth and the Postpartum Period

Planning for birth involves readying the participants and requires considerable time, thought, and energy. More preparation on the part of the family is required when birth occurs at home or in a birth center. **Table 31-4** lists family responsibilities in preparing for childbirth and the postpartum period.

Table 31-4	The Family's Preparation for Birth and Postpartum
Continuing commitment to a jointly created plan of maternity care	
Keeping the midwife informed of any life changes	
Reading material focused on maternity	
Attending childbirth education classes	
Writing an intrapartum birth plan	
Confronting and resolving fears	
Preparing prenatally for breastfeeding and pediatric care	
Preparing invited participants for their responsibilities	
Preparing siblings for their participation	
Paying fees for care and/or conveying insurance information	
Collecting and organizing specified supplies for home birth	
Obtaining and packing supplies to take to the birth center	
Understanding logistics for 24/7 availability of the second qualified practitioner	
Having a clean house	
Arranging for pet safety and care at the time of labor and birth	
Obtaining an infant car seat and assuring proper installation	
Making an accurate, detailed map to the home or checking global positioning system (GPS) accessibility	
Visiting an obstetric consultant, if needed	
Making emergency plans and posting them by each telephone and in the chart	
Having a reliable vehicle available with a full tank of gasoline	

Knowing the route to the hospital or birth center
Arranging doula participation if desired

As the due date nears, childbirth classes help the woman and her primary support partner learn about and focus on the upcoming birth. The classes that are most helpful emphasize the woman's inner strengths and are presented by an instructor who is knowledgeable about home and/or birth center births. The instructor communicates confidence that unmedicated birth is possible, given that few pharmaceutical options are available in the home or birth center. Instead, women will rely on techniques learned in class to enhance comfort and relaxation, to relieve pain, and to augment labor progress. Childbirth classes located in hospitals are often intended as an orientation to the hospital system and prepare the woman for labor and birth in that setting.

Writing a birth plan gives women and their partners an opportunity to discuss what is important to them for their birth. These plans detail preferences about the environment, social interaction, and comfort measures. Women and their partners identify the persons who will be present at the birth, the specific duties they may have, the role of siblings, religious or spiritual observances, and planned events with family and friends. Birth plans also describe coping measures learned from previous births, desired positions for labor and birth, and preferences for privacy and company at various points in labor. Plans for music, photography, and audio and/or video recording may be outlined.

To promote realistic thinking about possible transfer of care, families can be encouraged to write a hospital birth plan in addition to a home or birth center birth plan. This plan focuses on preferences regarding interventions and parental or family contact with the newborn in the presence of necessary medical interventions.

Most home and birth center practices anticipate that women will breastfeed their infants unless there are extenuating circumstances. Breastfeeding is an essential safety factor for women giving birth in homes or birth centers. Breastfeeding helps to keep the uterus contracted, thereby helping to control bleeding postpartum after the birth attendants leave the home or the family is discharged from the birth center. La Leche League and other breastfeeding support groups are invaluable resources for prenatal breastfeeding preparation and support. If breastfeeding problems are encountered, lactation consultants can be very helpful.

Persons who will be present for the birth also require preparation. If children will be present, they also need to be prepared for birth events, including sights and sounds. The children must be cared for by a designated adult other than the birth partner, so the woman and her partner can give their complete attention to managing labor. The designated adult's primary responsibility is attending to the children's needs and the woman's preferences at the time of the birth. This individual's plans for attendance at the birth may change as the children's and/or the woman's needs change; adults designated to care for the children should be prepared to leave the room where the birth is taking place if necessary. The children's support person also commits to caring for the children continuously until the woman and/or her partner return from the hospital if a transfer becomes necessary during or after the birth.

The people attending the birth should be selected carefully. It should be made clear that

people invited to attend the birth may be asked to leave if the woman seems to be distracted by their presence. Some participants may have fears about out-of-hospital births. Addressing their fears before the birth may change attitudes, and their attendance at the birth can be a transformative experience.

For women who plan to give birth in a birth center, bags should be packed and supplies ready by 36 weeks' gestation (**Table 31-5**). Supplies needed for home birth should be organized by 36 weeks' gestation and kept in or close to the room in which the woman intends to give birth (**Table 31-6**). A current electronic or paper copy of the woman's health record should also be available.

Table 31-5	**Family Supplies for a Birth Center Birth**
Nutritious, easy-to-prepare meals and snacks for the family, any support persons, and birth attendants	
At least 3 quarts of juice, energy drink, tea, and broths	
Comfortable clothing to wear during labor and extra clothing to wear home	
Extra pillows	
Music (CDs or electronic device)	
Camera and/or video camera	
Swim trunks/suit and extra towels for partner when water immersion is anticipated	
Entertainment for older children (e.g., books, games, videos)	
Nutritious postpartum meal for the new parents and birth attendants	
Hydrogen peroxide and ammonia for spot removal and laundry	
For the Newborn	
Clean thermometer	
Receiving blankets	
Petroleum jelly	
At least 5 disposable newborn-size diapers and baby wipes	
Baby clothing suitable for the weather	
Car seat, in place, adjusted to fit the newborn	

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Table 31-6	**Family Supplies for a Home Birth**
Most midwives request that the family assemble and organize supplies by 36 weeks' gestation. Some of the supplies are common household items; others can be purchased through a company that customizes birth kits to the specifications of the midwife or the family.	
Electronic or paper copy of chart to date	
Mattress cover (e.g., plastic drop cloth, shower curtain liner)	
Extra pillows covered with plastic, extra linen	
Soap for hand washing	
Food and fluids for labor, birth, and postpartum for the family, any support persons, and birth attendants	
Flexible straws	
Ice	

Clean towels
Antiseptic solution (e.g., povidone–iodine, benzalkonium chloride)
Wash cloths or 4 × 4 gauze pads
Flashlight with batteries and extra batteries
Mirror
Two-quart size bowls (for placenta)
Bulb syringe
Heating pad
Baby hats
Receiving blankets/soft towels
Thermometer
Petroleum jelly
Measuring tape
Peri pads and underpants
Squeeze bottle for perineal rinse
Disposable waterproof underpads
Baby supplies, including clothes, diapers, wipes, and car seat
Large plastic garbage bags
Plastic freezer bags
Two lightweight plastic containers for emesis
Paper towels
Tissues
Laundry detergent, ammonia, hydrogen peroxide, alcohol
Check photography supplies and charge batteries
Organize audio/music devices and other comfort enhancers

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Prenatal Home Visit

A home visit by the midwife and/or the assistant at approximately 36 weeks' gestation can provide assurance that the family is ready for the home or birth center birth. For home birth, a relatively clean house and pet care are obvious responsibilities of the family. Most pets can be present at births without causing any difficulties. The family should prepare a detailed map or ensure global positioning system (GPS) accessibility that can be tested for accuracy when this home visit is made. In addition, the family should make any needed parking arrangements for the midwife and the assistant.

Plans for cord blood preservation, placental encapsulation, or other requested services are discussed during this home visit. If a water birth is planned, the midwife reminds the family to test the hot water tank capacity before labor begins, to assure that all attachments are at hand and functioning properly, and to troubleshoot for any leaks. Information about equipment and safety requirements for water birth are included in the Resources section at the end of this chapter.

An emergency plan and important telephone numbers should be posted in the birthing room and in the woman's chart. The family should make a "practice run" to the backup hospital to

ensure that they know the fastest route, the traffic patterns, the location to park, and the point at which to enter the building should a transfer become necessary. The car that will be used for emergency transportation needs to be in good repair and have adequate fuel to get to the hospital. Having hospital admission forms completed online or a completed paper copy in the chart before labor begins will decrease stress if a transfer to the hospital becomes necessary.

The Intrapartum Period at Home or in the Birth Center

During the intrapartum period, the midwife's role is to be the guardian of the birth environment and the woman's energy to labor and give birth. The woman depends on the midwife's ability to assess for and promote continued normalcy. The midwife's ongoing presence provides physical and psychological labor support, as well as attention to comfort and progress using simple interventions only when necessary. Should any complication occur, the midwife must have the skills needed to manage, stabilize, and in many cases resolve the situation, consulting other resources if indicated. If complications cannot be resolved in the home or birth center setting, the midwife must have a plan to implement a transfer to the hospital.

Personnel and Equipment

Most home and birth center practices employ two intrapartum providers: the primary midwife and a second qualified practitioner or assistant. The assistant may be a second midwife, a registered nurse, an emergency medical technician, or even an individual preparing to ultimately attend a midwifery program. Labor is often long, and to meet the woman's needs, the midwife may need periodic rest. Inclusion of two caregivers assures adequate staff are available to meet the laboring family's needs for support and safety maintenance without compromising privacy. Some families desire the additional services of a doula.

The assistant provides support and technical labor, birth, and postpartum skills. Essential skills for the assistant include evaluation of the woman's physical and emotional needs and status during labor, the ability to monitor fetal and newborn vital signs, and certification in both adult cardiopulmonary resuscitation (CPR) and the Neonatal Resuscitation Program (NRP). In addition, the assistant should be able to provide support with breastfeeding and postpartum care. It is not necessary for the assistant to be a healthcare professional: Childbirth educators, La Leche League leaders, and doulas may also possess the additional skills needed.

The equipment needed to assist with normal birth is simple. Each time the midwife and the assistant attend a birth, they review the individual's chart and birth plan to focus attention on the individual needs. They organize and check the equipment (**Table 31-7**)[32] and review the skills needed to address any specific health issues. **Figure 31-1** shows the equipment needed for newborn resuscitation.

Table 31-7	Midwife Equipment for Birth at Home or in the Birth Center
Essential Supplies	
Current electronic or paper copy of chart available	***Newborn Supplies***
Paper or electronic forms for intrapartum, postpartum, and newborn charting	Heating pad for baby blankets
Birth certificate forms	Bulb syringe
Blood pressure cuff, stethoscope	Cord clamp or tape
Fetoscope/Doppler stethoscope	Baby scale
Gloves (sterile and nonsterile)	Measuring tape
Lubricant (sterile and nonsterile)	Vitamin K

Antiseptic wash
Urinary catheters
Suture with needles
Local anesthetic, syringes, and needles
Urine dipsticks
pH indicator paper
Intravenous fluids: lactated Ringer's and 5% dextrose in Ringer's lactate, normal saline
Intravenous catheters, long intravenous tubing, tape, antiseptic ointment, gauze pads
Blood collecting supplies: tourniquet, tubes, syringes, needles and Vacutainer
Sharps box and mechanism for hazardous waste disposal

Sterile Instruments

Scissors (2 pairs)
Large clamps (Kelly or Rochester-Ochsner)
Needle holder/driver
Ring forceps
Amniohook
Sterile 4 × 4 gauze pads
Erythromycin ophthalmic ointment

Resuscitation Supplies

Light source (e.g., flashlight)
DeLee suction trap or suction machine
Infant positive-pressure resuscitation bag and mask
Pulse oximeter adult/newborn
Laryngeal mask airway
Laryngoscope with infant blade (size 1)
Endotracheal tubes, sizes 3.0 mm and 3.5 mm, with stylets
Epinephrine
Feeding tube #8F and large syringe for stomach aspiration
Oral airways (infant, adult)
Small oxygen tank, oxygen mask and tubing

Postpartum Hemorrhage Control

Injectable oxytocin (Pitocin)
Injectable and oral methylergonovine maleate (Methergine)
Misoprostol tablets (Cytotec)

Extra Supplies

Adult positive-pressure resuscitation bag and mask
Antibiotics and 150-cc normal saline bags for intravenous administration of antibiotics
Camera
Electrolyte replacement drink (e.g., Pedialyte, Gatorade)
Flexible straws
Hot-water bottle or heating pad
Lidocaine jelly for topical use
Massage roller
Slow-cooker pot for hot perineal compresses

Newborn Supplies

Baby footprint stamp
Blood glucose monitoring sticks and/or glucometer
Cotton baby hats
Otoscope/ophthalmoscope

Personal Supplies

Apron or gown
Change of clothes
Protective eyewear
Toilet articles, personal medications
Mirror
Speculum

Sterile Instruments

Allis clamps
Combs for acupressure points
Culturette
Hemostats
Tissue forceps

Based on Bailes A. *The Home Birth Practice Manual.* 3rd ed. Washington, DC: American College of Nurse-Midwives; 2016.[32]



Figure 31-1 Newborn resuscitation equipment necessary for a home birth.

Note: The American Heart Association and American Academy of Pediatrics' Newborn Resuscitation Program algorithm is printed on a small card and can be used to guide the resuscitation process.

Photo courtesy of Marsha Jackson.

When labor begins, the family contacts the midwife who is the primary attendant and is responsible for coordinating care. During the initial labor call, the midwife gathers data about how the woman is experiencing her labor, her needs, and her support systems. The midwife, the woman, and her support network review choices and make appropriate management plans, taking into account the woman's perceptions and history. Plans may include a decision for the midwife to meet the woman for additional assessment. Maintaining contact between the midwife and the woman may be all that is necessary during the early phases of labor.

If the woman is comfortable at home and has the support she needs, she may want to continue her normal activities of daily living while emphasizing hydration, nutrition, and increased rest as needed. A variety of food choices should be provided by the family for the laboring woman, guests, and the professional staff. Friends and family may come to help with final preparation and management of other household activities, freeing the woman and her primary support partner to focus on the labor.

A woman planning a home birth can "double-make" her bed to protect the mattress. The bed can be made with a full set of linen and then completely covered with a plastic mattress cover, a drop cloth, or a shower curtain. A clean old sheet is placed on top of the plastic so that the bed may be used as desired for labor or birth. When the birth is completed, it is a simple matter to remove the top sheet and the plastic. Clean linen is then available in place for the woman's comfort postpartum.

The midwife or the assistant can perform the first physical assessment of labor in the woman's home, the birth center, or the midwife's office. The initial assessment should include a review of the prenatal record, a physical examination to assess presentation, vital signs of

the woman and fetus, uterine contractions, and maternal response to labor. If membranes are not ruptured, a pelvic examination might be part of the initial physical assessment to document cervical dilation, verify the presenting part, or provide information that the woman requests.

The time frame in which the midwife should be in close or continuous attendance can vary as labor progresses. As labor becomes active, the midwife and/or the assistant will be present to assess maternal well-being and fetal heart tones using intermittent ausculatation.[36] The midwife or the assistant also may augment the birth partner's support measures, give reassurance, and guide the woman to conserve her energy. The woman is free to move around and assume any position she chooses. The midwife may suggest positions that promote comfort, encourage progress, and maintain fetal well-being.

During the second stage of labor, it is not unusual for the woman to continue to change positions, moving from the bed to other locations within the home or birth center. The midwife must be alert, flexible, and able to anticipate the woman's movements. Sterile instruments should be placed on a portable sterile surface, allowing the midwife to move while following the woman's lead. If the woman is reluctant to change position, the midwife may use persuasive communication to suggest positions that will facilitate the birth.

As the fetal head emerges, the woman and/or her partner might reach down to complete the birth (**Figure 31-2**). Within the first few seconds after birth, the midwife or assistant will do a quick evaluation of newborn well-being, assessing the newborn's muscle tone, cord pulsations, and respiratory effort, and then assign a 1-minute Apgar score. The midwife and the assistant continue frequent newborn assessment for the first hour after birth. The newborn is kept skin-to-skin with the mother and covered with warm dry blankets, supporting newborn thermal regulation. The midwife is responsible for directing the assistant's care and continuing to observe the newborn for a successful transition from intrauterine to extrauterine life.



Figure 31-2 The mother reaches to complete the birth.

Photo courtesy of Sue Baelen for Wisewoman Childbirth Traditions.

It is normal for some newborns to latch on to the woman's breast immediately, whereas other newborns are more interested in exploring the change in their environment—looking, listening, nuzzling, licking, and taking their time to find the nipple and latch on. While the midwife is attentive to the progress of the third stage of labor, the assistant monitors the newborn's vital signs without disturbing family–infant bonding. The newborn can remain skin-to-skin in the birthing woman's arms while the assistant checks the vital signs every 30 minutes, or more frequently as indicated. Welcoming the newborn can take place as the family has planned. Siblings are often present at the birth or they can be invited in when the woman wishes.

Birth of the placenta is usually unhurried. If the woman needs or chooses to do so, she may squat or sit on the toilet to facilitate the completion of the third stage. The midwife may dispose of the placenta through a hazardous waste company. Some families desire to keep their placenta to bury under a plant. Other families may make arrangements for the placenta to be dried and encapsulated, although the latter may be controversial.

The Early Postpartum Period and Follow-Up

The first 2 to 4 hours postpartum is a special time for the new mother, her family, and friends to welcome and bond with the newborn. The midwife and the assistant in the home and birth center setting are participants during this time of welcome. Once it is assured that both maternal and newborn vital signs are within normal limits, and the amount of lochia, uterine position, and uterine consistency indicate the uterus is contracted, the midwife can leave the birthing room, facilitating a period of privacy and continued bonding time for the family. The woman and her newborn are likely to be physically stable after breastfeeding is established. Most often, eye prophylaxis and vitamin K administration are delayed until after this initial period of family bonding. Some families make an informed choice to waive these newborn procedures. Perineal suturing, if necessary, may also be delayed if the woman is not bleeding excessively; alternatively, it may be done immediately, according to the woman's preference.

While bonding continues, the midwife can attend to the usual responsibility of documenting the birth. It is not unusual for the midwife to spend this period actively working at jobs typically accomplished in the hospital by nursing, secretarial, housekeeping, and dietary staff. The midwife and the assistant assure proper nourishment for the woman, help with breastfeeding, assist the woman to shower and void, wash the instruments, and put the birth site in order. Quite often, family and friends who have attended the birth as guests become helpers during this postpartum family time.

As long as the newborn is adapting to life outside the uterus well, the physical examination is usually done after a period of bonding according to the family's preference. The midwife does a complete newborn physical examination, often using a warm, but unplugged heating pad. This examination provides an opportunity for teaching, as it usually takes place in the woman's bed and may be performed in the presence of excited children greeting the new sibling. Family members often participate in weighing, measuring, and dressing the newborn in the midst of friends taking photographs and a great deal of socializing and celebration.

After the midwife departs the home or the family leaves the birth center, it is recommended that the newborn and mother stay close together to aid in temperature stabilization, enhance maternal–newborn communication, and stimulate milk production. A newborn bath may be delayed for the first 24 to 48 hours after birth. The woman and newborn are assessed in the first 2 days either at a home or office visit or via a telephone assessment. Breastfeeding, the developing family relationships, adequacy of help in the home, infant caretaking abilities, and follow-up appointments with the newborn's healthcare provider are assessed at this visit.[1] During the 6-week postpartum recovery period, the family may be seen in the midwife's office to evaluate maternal involution and other maternal, infant, or family needs.

Framework for Clinical Management

Management strategies for selected clinical situations in the home and birth center can be divided into three categories: preventive, non-urgent, and urgent emergency. In-labor transfer rates to hospital-based care for women planning to birth at home or in a birth center range from approximately 7% to 24%.[18,20] The likelihood of transfer depends on multiple factors in addition to maternal or fetal clinical status, including distance to the collaborating hospital and collaborative protocols. Although a small fraction of women transfer to hospital care prior to labor, 84%[23] to 89%[27] of those who begin labor at home or at a birth center ultimately give birth at their preferred site. For the majority of the 10% of women who transfer to a hospital during labor, the reason for the transfer is failure to progress in labor (40%) and desire for pain relief medications or maternal exhaustion (20.5%).[27] Postpartum transfer occurs for approximately 1.5% of women who give birth at home and neonatal transfer occurs for approximately 1% of newborns.[27] Postpartum transfer rates from birth centers are similar, with 2.5% of women requiring transfer postpartum and 2.6% of newborns requiring transfer.[23] Most transports for hospital care are nonemergent. When Stapleton et al. reviewed transfers in a national birth center study (n = 13,030), they found that emergency transfer was required for fewer than 1% of women who gave birth in a birth center.[23]

Preventive management is the first line of defense to maintain safety during any birth. Ideally, health promotion during the antepartum period succeeds in bringing a well woman with a healthy pregnancy to term. Most women who choose home or birth center birth understand that to achieve their goal of giving birth in their preferred site, they must experience a pregnancy free of complications. Safety is most effectively maintained when strategies to promote health are actively implemented. Early identification of deviations from normal may be non-urgent, addressed with consultation and collaboration to resolve the deviation. Urgent emergency situations may require referral in addition to consultation and/or collaborative care. If the midwife determines that more personnel and complex technologies are needed, previously arranged plans are implemented for transfer of care to obtain interventions that are more safely provided within a hospital-based system.[15-23,28] During transfer, the midwife must utilize excellent decision-making and clinical skills, and promote good communication among all parties involved—the midwife, the woman, the family, and the consulting practice. Communication with the clinicians who will receive the woman is enhanced with a verbal report, along with a concise, complete chart that serves as an ambassador for the woman and the referring midwifery service.

Management of Non-Urgent Clinical Situations

Prolonged Labor

Prolonged labor due to maternal exhaustion in nulliparous women is the most common reason for transfer from home or a birth center into the hospital. The midwife's patience and attention to supporting the woman's efficient use of energy during labor are the midwife's most important preventive interventions. Adequate hydration and ingestion of nutritional sources of energy are essential. The midwife may encourage periods of rest when labor ebbs.

When slow progress does become a concern, it must be clearly understood that oxytocin (Pitocin) for the purpose of augmenting labor is not administered in the home or birth center. Exogenous oxytocin can cause tachysystole and variant fetal heart rate patterns that may indicate developing fetal acidemia if persistent. If labor progression cannot be accomplished by means of upright positions, adequate hydration, nutritional support, or nipple stimulation, then a hospital transfer may be indicated.

Sometimes when labor continues without progress, the woman becomes too fatigued to cope with labor pain and strategies for nonpharmacologic pain relief are no longer effective. If the woman's need for pain relief cannot be satisfied by the choices available in the home or birth center, a transfer to the hospital is indicated. A non-urgent transfer may take place in the family car as long as maternal and fetal vital signs are within normal limits. Women transferred for prolonged labor with their first birth can usually complete subsequent births at home or in birth center settings.

Prolonged Rupture of Membranes

In cases of prolonged rupture of the membranes, concern about infection is reduced but not eliminated at home or in the birth center. Pathogenic bacteria are not in abundance as long as the woman is not a carrier of group B *Streptococcus* (GBS), and avoiding vaginal examinations decreases the risk of infection. Nevertheless, monitoring for signs of infection remains critical. Setting a time limit before transfer to the hospital, if necessary, will depend on the midwife's preferences, the woman's preferences, and negotiations with the healthcare system, including consideration of maternity care and neonatal community standards.

Group B *Streptococcus* Colonization

Women who have vaginal colonization with GBS may give birth safely at home or in a birth center. If intravenous antibiotic prophylaxis is indicated, the antibiotic may be administered in these settings. Women who are colonized with GBS should discuss follow-up plans for the neonate's care with their pediatric care provider during the prenatal period. Families can be taught to monitor and record the newborn's vital signs, enabling them to convey information about the newborn's health to the pediatric care provider within 12 to 48 hours after birth.

Meconium Staining

Meconium-stained amniotic fluid does not always indicate a need to transfer the woman to the hospital. If meconium appears at any time after rupture of the membranes, the color, consistency, and amount of the meconium should be evaluated. Notification of the consultant depends on the consultation agreement and the characteristics of the meconium. Amniotic fluid with thick meconium indicates that the woman should be transferred to the hospital before the birth, if there is adequate time to do so safely.

If the fetal heart rate has normal characteristics that do not suggest development of fetal acidemia, and if labor is progressive and well established, proceeding with the home or birth center birth is appropriate. However, a discussion with the woman and her support partner regarding the midwife's assessment, possible consequences of meconium aspiration, and treatment options both in and out of the hospital should always be initiated and documented. A transport decision is made by combining the midwife's clinical judgment, skill and experience with consideration of the family's preferences. If the decision is made to remain at home or in the birth center for the birth, a DeLee suction trap and other resuscitation equipment should be readied.

Vaginal Birth After Cesarean Birth

Women who have experienced a prior cesarean birth may want to avoid having another cesarean. Some women who want to have a vaginal birth after a prior cesarean live in geographic areas where local hospitals do not provide vaginal birth after cesarean (VBAC) services.[37,38]

Both risks and benefits are associated with a trial of labor after cesarean (TOLAC) or an elective repeat cesarean birth (ERCB), as reviewed in the *Pregnancy-Related Conditions* chapter. If the woman has experienced a prior cesarean birth, the decision about which type of birth to have can be a complicated decision for families to make.

In brief, limited data indicate that approximately 87% of women who give birth at home or in a birth center and who have had a previous cesarean birth will experience a vaginal birth—a rate that is higher than the 74% successful VBAC rate among women who plan a VBAC in a hospital setting.[37-39,40] Although the successful VBAC rate may be higher in out-of-hospital settings, women who labor after a prior cesarean birth have an increased risk for uterine rupture and subsequent increase in fetal/neonatal morbidity or mortality.[37-41,42] Cox et al. examined birth outcomes among a large cohort of women who planned to give birth at home to identify differences in maternal and fetal-neonatal outcomes between women with a history of prior cesarean birth (n = 1052) and multiparous women in a comparison group who had not had a prior cesarean birth (n = 12,092).[37] A between groups comparison of neonatal outcomes for the same cohort of women revealed an almost four-fold increase in combined intrapartum and perinatal death in the prior cesarean birth group versus the comparison multiparous group (4.74/1000 births versus 1.24/1000 births; $P \leq 0.05$).[37] Studies of newborn outcomes for women who had a previous cesarean birth and labor at home have also found an increased incidence of newborn seizures (adjusted odds ratio [aOR], 8.53; 95% confidence interval [CI], 2.87–25.4; 0.19% versus 0.02%, respectively) and Apgar scores of less than 7 at 5 minutes (aOR, 1.5; 95% CI, 1.35–1.96; 2.68% versus 4.42%, respectively) when compared to a

similar cohort of newborns of women who gave birth in a hospital setting, although newborns in the hospital setting had higher rates of NICU admission (aOR, 0.40; 95% CI, 0.29–0.57; 1.11% versus 3.10%, respectively).[39]

A previous cesarean birth with no prior vaginal birth is among the conditions associated with the highest perinatal death rates (10.2/1000 pregnancies) following birth at home—a rate that is similar to the perinatal death rates for breech presentation (16.8/1000 pregnancies), twin gestation (14.5/1000 pregnancies), and preeclampsia (16.1/1000 pregnancies).[42] Nevertheless, the absolute risk of perinatal death is less likely for women who have a previous cesarean birth and a prior vaginal birth when compared to the perinatal death rate for nulliparous women (1.27/1000 pregnancies versus 3.43/1000 pregnancies, respectively).[42] Having had one vaginal birth appears to confer significant reduction in the risk for adverse outcomes.

Other studies of women who had a prior cesarean birth and labor at home or at a birth center in the United States have noted an increase in neonatal morbidity and mortality when compared to outcomes of women with a prior cesarean who give birth in a hospital setting.[37-39,41] ACNM and other organizations recommend a hospital as the preferred birth site for women who had a previous cesarean birth.[43]

Management of Urgent Emergency Clinical Situations

Variant Fetal Heart Rate Patterns

Variant fetal heart rate patterns, such as recurrent decelerations or tachycardia, that do not resolve with increased hydration, a change in maternal position, and/or a brief period of oxygen by mask indicate that the fetus is at increased risk for developing metabolic acidemia. A transfer to the hospital by ambulance is indicated in such a case to allow access to the needed personnel and technology to manage the problem if there is time to complete the transfer before the birth occurs. Similarly, if at any point during the labor, the midwife feels a need to monitor the fetus more closely than intermittent use of the fetoscope or Doppler allows, then the woman should be moved to the hospital rather than a fetal monitor moved to the woman. Intrapartum use of electronic fetal monitors is outside the Standards of the American Association of Birth Centers (AABC).

Malpresentation: Breech Presentation

Women diagnosed prenatally with a fetus in breech presentation or other malpresentation should have a shared decision-making discussion about external cephalic version, planned hospitalization for vaginal breech birth, or cesarean birth for breech. Breech presentation at the onset of labor is an indication for hospitalization, given the increased risk for adverse newborn outcomes.[42] Rarely, a woman in labor at home or in the birth center may present with a fetus in breech presentation that was not previously detected. If the birth is imminent, the midwife should manage the breech birth in place, but call 911 for extra personnel and transport resources should they be needed.

Postpartum Hemorrhage

A common fear regarding home or birth center birth is “What if the woman bleeds too much?” The reality is that postpartum hemorrhage requiring transport is rare, with its incidence being reported as between 0.2% and 1%.[23] In research that compared outcomes for a group of women giving birth in the home or a birth center with a low-risk group of women giving birth in the hospital, a greater incidence of hemorrhage was found in the hospital group.[19]

If postpartum bleeding is excessive and attributed to uterine atony, intravenous volume expanders with added oxytocin (Pitocin) can be initiated, methylergonovine maleate (Methergine) can be administered intramuscularly or orally, and/or misoprostol (Cytotec) can be administered orally or per rectum per standard protocols for drug, dosage, and timing. However, if a woman has a prenatal hematocrit of less than 30% or a hemoglobin of less than 10 g/dL, she may be unable to tolerate even a normal postpartum blood loss; in this circumstance, prenatal transfer to hospital-based care may be considered.

If immediate or delayed postpartum blood loss results in symptoms of hypovolemia such as dizziness, weakness, or difficulty in ambulation, transfer to hospital-based care is needed because blood and blood products are not available in home and birth center settings. If the

woman is stabilized at home but subsequently experiences reduced self-care abilities or delayed lactogenesis II, she may require hospitalization for evaluation and treatment. It is important to remember that women who give birth at home or in a birth center will not have continuous professional postpartum support beyond 3 to 12 hours postpartum.

Newborn Complications

"What if the baby doesn't breathe?" is the number one fear that the general population has regarding home and birth center birth. The reality is that resuscitation is rare when a well woman experiences a healthy pregnancy, has an unmedicated and uncomplicated intrapartum course,[44] and has an uncomplicated birth at home or in a birth center. Nevertheless, as noted earlier, Neonatal Resuscitation Program certification is a critical skill for both the midwife and the second qualified practitioner who attend home and birth center births. When a need for resuscitation occurs in these settings, both the woman and the newborn may need medical support at the same time, so having two qualified providers present is essential. In the large majority of cases, the resuscitation equipment and skills provided by home and birth center midwives are usually successful in stimulating the newborn's respiratory effort. If this does not occur, the goal is to stabilize the newborn and provide adequate oxygenation until transport to a neonatal intensive care unit is completed.

Transfer to Hospital-Based Care

If problems require hospital-based care, the midwife becomes the link between the family and the hospital that will provide the necessary intervention that all wished to avoid. Intrapartum transfer is stressful for all concerned—the family, the midwife, and the hospital receiving the family—and good communication among everyone involved is critical. Notifying the receiving providers as far in advance as possible helps the transfer go smoothly and enables the woman to get the care she needs in a timely fashion.[45] Transfer is usually accomplished by car for non-urgent indications. Management of complications that may occur during labor, birth, or the early postpartum period depends on written clinical practice guidelines, characteristics of the woman and her support system, the network of health system support, and the midwife's ability to perform clinically.[28] Other factors that influence decision making may include the CNM's/CM's level of experience, distance to the hospital, and road conditions. The transfer summary is a simple, easy-to-use form that conveys pertinent data to the receiving staff (**Figure 31-3**). It is also a quality management tool that enables accurate statistical collection of practice data.

Name _____

Date	Time	
		Reason for transfer:
		Review with client and family:
		Name of consultant:
		Name of hospital:
		Consultation and plan:
		Transport called (if applicable):
		Arrival of transport vehicle (if applicable):
		Mode of transport:
		Mother: T ____ P ____ R _____ BP ____ Condition _____
		Baby: FHT _____ T _____ P _____ R _____ Condition _____
		Departure from home/birth center (circle one)
		Arrival at hospital/accompanied by:
		Receiving provider:
		Type of intervention:
		Initiation of treatment:
		Birth information: SVD Inst. Asst. C/S Apgar ___/___
		Outcome summary:
		CNM departure from hospital:
		Breastfeeding status:
		Release for hospital records sent:
		Hospital records received:

Signature _____

Figure 31-3 Transfer summary.

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In most situations, the problem necessitating transfer is identified as it develops over time. Unfortunately, transfers often occur when labor has been long, and both the woman and the midwife are fatigued. When a hospital transfer becomes necessary, women who had planned a

home or birth center birth must cope with two concerns: worrying about their health and/or their fetus/newborn's health, and experiencing the feelings (perhaps grief) associated with the loss of their planned home or birth center birth. The client and her family may be disappointed, and sometimes they may blame the midwife for their difficult situation.

Providing anticipatory guidance during prenatal visits can reduce the family's distress if transport becomes a reality. Reminding families late in pregnancy of the possibility that transfer could be necessary, reviewing practice statistics, and describing actual transport experiences may be helpful. Discussion may include a review of not only the decision-making process, but also consultation services, the trip to the hospital, and the admission process. Women need help to understand that if they are going to the hospital, they are going because they need the technical interventions.

During the process of shared decision making at the first visit, and again around 36 weeks' gestation, women should be made aware of the possibility that their fetus/newborn could be injured or die during the birth process regardless of their chosen birth setting. Although most people hesitate to focus on this frightening issue, women need to ask themselves what they would do and how they would cope. They also need to explore how they would respond to family or friends who may not have been supportive of their decision to give birth away from a hospital. In such circumstances, the midwife needs to be prepared to offer additional support and counseling for the woman and her family. Some women, as well as the practitioners who serve them, may benefit from professional counseling to process unexpected outcomes.

Promoting a Smooth Transfer of Care

Best practice guidelines for transferring from a planned home birth to a hospital setting have been developed by the Home Birth Summit.[45] The guidelines include model practices for midwives and receiving hospital staff as well as recommendations for policy development.[45]

Management dilemmas may occur particularly if the woman's birth plan or desires conflict with accepted medical community standards. It may be difficult to convince a consultant to implement a plan that may be safe, but unpopular in the medical community. Both the midwife and the consultant may be caught in the middle. Families may pressure their midwife to continue care despite complications that arise during the labor or birth. This conundrum has the potential to threaten the relationship with the consultant, and it can create a climate of disrespect at the hospital that could cause clinical harm for that family. Such a choice also has the potential to negatively affect the home or birth center practice, creating barriers for clients and the practitioners who serve them and, therefore, adversely affecting future birth options in the community. Many times a midwife may be tempted to be led by the heart instead of the head. Wish management does not work. Learning to say "no" is not just important—it is critical. Midwives must be brutally honest with themselves and not be pressured by clients to practice outside of their clinical practice guidelines and circle of safety. Care must be taken not to jeopardize the entire practice for one client.

Business Aspects of Home and Birth Center Settings

The legal status of home birth and birth center settings varies in different states within the United States. In a few states, it is illegal for midwives to attend home births. Furthermore, regulatory requirements that must be met by midwives who attend home births vary by state. Birth center regulation is also inconsistent from state to state. Midwives practicing in the United States are encouraged to investigate the legal requirements for home birth and birth center operation in the states in which they live and practice. ACNM has staff specializing in professional services and legislative liaisons to keep members aware of practice and political issues. AABC also has staff who assist midwives who plan to open and operate birth centers. To break down barriers, safeguard the profession, and protect the healthcare rights of women and their families, home and birth center midwives have to be both alert and proactive.

All midwifery practices need to address business and regulatory issues. Many of the same professional and legal documents that are necessary when starting a hospital practice are also necessary for a home or birth center practice. Birth center and home birth practices are often midwifery owned and operated. A self-employed independent midwife has more control over clinical, business, and administrative decisions. These practices tend to be small, minimizing bureaucracy and simplifying communication and policy. Such a small organization is more responsive to the needs of the woman, her family, and the midwife when compared to a larger institution. On the downside, there is much more responsibility, time involved, and financial risk in self-employment.[32]

Administrative support is helpful for a home or birth center practice, so that the midwife can continue providing clinical services. The person in charge of billing and collections is key to the practice's financial survival. His or her interpersonal skills and commitment to the practice help keep the service financially sound. Salaries for clinical staff should amount to approximately 50% of the income generated by the practice. Even in established practices, it is not uncommon for home birth or birth center midwives to have lower salaries than their hospital-based colleagues.

Many home birth and birth center midwives are participating providers on health insurance panels, and are reimbursed by Medicaid. Some insurance companies require accreditation through the Commission for Accreditation of Birth Centers for birth center–based midwives to become participating providers. ACNM has developed materials to help midwives develop business and negotiation skills.

Financial arrangements with consulting practices should be clear. Consultants may decide not to charge a consultation fee to the midwife for providing services, but rather charge the woman only for services actually rendered. By assuring that the woman pays the consulting practice promptly, the midwife fosters a good consultation relationship. Sometimes when consultants serve home and birth center practices, those consultants may benefit from referrals not only from the midwifery practice, but also from client networks and sympathetic childbirth educators. Conversely, hospital-based colleagues may refuse cross-coverage for the consultants.

A system for data collection and analysis should be put in place before a practice opens. Accurate data collection and analysis facilitates evaluation, quality management, validation of

effective practice, peer review, and research. Practice data should be collected regularly and analyzed at least annually. ACNM, AABC, and Midwives Alliance of North America (MANA) have web-based data collection applications. When presented clearly and succinctly, statistics can also serve as a public relations tool conveying important information to families about outcomes and transfers.

Midwives working in home and birth center practices need to be astutely aware of who wields power. These midwives are practicing outside of the mainstream maternity system. Although these midwives work with less bureaucracy and more clinical freedom, their practice is subject to increased scrutiny and they lack the protective institutional layer that a hospital may provide. ACNM has staff specializing in professional services and legislative liaisons to keep members aware of practice and political issues. To break down barriers, safeguard the profession, and protect the healthcare rights of women and their families, home and birth center midwives have to be both alert and proactive.

Conclusion

Midwives have increased access to the important resources of time, place, and social interaction when working in the home or birth center environment, and can promote authority, centrality, and privacy for the women they attend. In the hospital, the staff may become inured to and unconscious of interruptions:

> *Studies found that a woman with a low risk delivery giving birth to her first child in a teaching hospital could be attended by as many as 16 people during 6 hours of labor and still be left alone for most of the time. Routine, though unfamiliar, procedures, the presence of strangers, and being left alone during labor and/or delivery caused stress, and stress can interfere with the course of birth by prolonging it.*[31](p10)

As long as the birth remains normal, midwives limit their role to helping the woman be comfortable, validating the efforts the woman makes to give birth, and, at times, augmenting and redirecting the partner's supportive efforts.

Within the home and birth center environment, continuity of care takes on a new meaning for the midwife. Midwives have more freedom to offer one-to-one care and to be a continuous presence during labor. The midwife carefully selects interventions and utilizes them only when the woman needs them and without interrupting the flow of labor. Being aware of and encouraging changes in place, time, and social interactions are special tasks of the midwife in the home and birth center settings. In these small and personalized environments, midwives maximize opportunities to practice "the art of doing nothing well."[46]

Resources

Organization/Author	Description	Webpage
American Association of Birth Centers (AABC)	Includes information about birth centers, position statements, information of standard for birth centers, and resources for health outcomes in birth centers.	http://www.birthcenters.org
American College of Nurse-Midwives (ACNM)	A Model Practice Template for Hydrotherapy in Labor and Birth.	http://onlinelibrary.wiley.com/doi/10.1111/jmwh.12587/epdf
	Home Birth Resources	http://www.midwife.org/Home-Birth-Transfer-Guidelines
Commission for Accreditation of Birth Centers (CBAC)	Information on which birth centers are CBAC accredited, requirements for accreditation, and so on.	https://www.birthcenteraccreditation.org/find-accredited-birth-centers/
Home Birth Summit	Best Practice Guidelines: Transfer from Planned Home Birth to Hospital.	http://www.homebirthsummit.org/wp-content/uploads/2014/03/HomeBirthSummit_BestPracticeTransferGuidelines.p
Midwives Alliance of North America (MANA)	Professional organization for certified professional midwives and midwives who care for women exclusively at home or in birth centers.	https://mana.org
Vedam S, Stoll K,	Home Birth:	http://midwifery.ubc.ca/files/2013/11/HomeBirth_AnnotatedGuideToTheLiteratur

Schummers L, Fulton C	An Annotated Guide to the Literature.

References

1. Varney H. *Varney's Midwifery*. 3rd ed. Sudbury, MA: Jones and Bartlett; 1997.
2. Starr P. *The Social Transformation of American Medicine: The Rise of a Sovereign Profession and the Making of a Vast Industry*. New York, NY: Basic Books; 1982.
3. Litoff JB. *American Midwives 1860 to the Present*. Westport, CT: Praeger; 1978.
4. Wertz R, Wertz D. *Lying-In: A History of Childbirth in America*. New Haven, CT: Yale University Press; 1989.
5. Hetzel AM. History and organization of the Vital Statistics System. National Center for Health Statistics. 1997. Available at https://www.cdc.gov/nchs/data/misc/usvss.pdf. Accessed September 19, 2017.
6. Laird MD. Report of the Maternity Center Association Clinic, New York, 1931–1951. *Am J Obstet Gynecol*. 1955;69(1):178-184.
7. Kline W. Back to bed: From hospital to home obstetrics in the city of Chicago. J His Med Allied Sci. 2018;73(1):29-51.
8. Stewart D, Stewart L. *Safe Alternatives in Childbirth*. Marble Hill, MO: NAPSAC International; 1976.
9. Ventre FI. The lay midwife. *J Nurse Midwifery*. 1977;22(4):32-35.
10. Myers-Ciecko JA. Evolution and current status of direct-entry midwifery education, regulation, and practice in the United States, with examples from Washington state. *J Nurse Midwifery*. 1999;44(4):384-393.
11. Ernst K, Bauer K. Birth centers in the United States. American Association of Birth Centers; 2017. Available at: http://c.ymcdn.com/sites/www.birthcenters.org/resource/collection/028792A7-808D-4BC7-9A0F-FB038B434B91/Birth_Center_in_the_United_States.pdf. Accessed September 18, 2017.
12. Phillippi JC, Alliman J, Bauer K. The American Association of Birth Centers: history, membership, and current initiatives. *J Midwifery Womens Health*. 2009;54(5):387-392.
13. MacDorman MF, Mathews TJ, Declercq E. *Trends in Out-of-Hospital Births in the United States, 1990–2012*. NCHS Data Brief, No. 144. Hyattsville, MD: National Center for Health Statistics; 2014.
14. Ernst K, Bauer K. *The Birth Center Experience*. Perkiomenville, PA: American Association of Birth Centers; 2016. Available at: https://c.ymcdn.com/sites/www.birthcenters.org/resource/collection/028792A7-808D-4BC7-9A0F-FB038B434B91/The_Birth_Center_Experience.pdf. Accessed September 19, 2017.
15. Commission for the Accreditation of Birth Centers. Indicators of compliance with standards for birth centers: Reference Edition 1.0 (effective 06/15/16). Available at: https://www.birthcenteraccreditation.org/wp-content/uploads/2014/02/CABC_IndicatorsRefEd_2016-0615v1.1.pdf. Accessed September 19, 2017.
16. Jackson ME, Bailes A. Home birth with certified nurse-midwife attendants in the United States: an overview. *J Nurse-Midwifery*. 1995;40:493-506.
17. Sakala C, Corry M. *Evidence-Based Maternity Care: What It Is and What It Can Achieve*. New York, NY: Milbank Memorial Fund; October 2008.
18. Birthplace in England Collaborative Group, Brocklehurst P, Hardy P, et al. Perinatal and maternal outcomes by planned place of birth for healthy women with low risk pregnancies: the Birthplace in England national prospective cohort study. *BMJ*. 2011;343:d7400. doi: 10.1136/bmj.d7400.
19. Hutton EK, Reitsma AH, Kaufman K. Outcomes associated with planned home and planned hospital births in low-risk women attended by midwives in Ontario, Canada, 2003–2006: a retrospective cohort study. *Birth*. 2009;36(3):180-189.
20. Janssen PA, Saxell L, Page LA, Klein MC, Liston RM, Lee SK. Outcomes of planned home birth with registered midwife versus planned hospital birth with midwife or physician. *CMAJ*. 2009;181(6-7):377-383.
21. de Jonge A, van der Goes BY, Ravelli AC, et al. Perinatal mortality and morbidity in a nationwide cohort of 529,688 low-risk planned home and hospital births. *BJOG*. 2009;116(9):1177-1184.
22. Cheng Y, Snowden JW, King TL, Caughey AB. Selected perinatal outcomes associated with planned home births in the United States. *Am J Obstet Gynecol*. 2013;209(4):325.e1-325.e8.
23. Stapleton SR, Osborne C, Illuzzi J. Outcomes of care in birth centers: demonstration of a durable model. *J Midwifery Womens Health*. 2013;58(1):1-12.
24. Wax J. Maternal and newborn morbidity by birth facility among selected United States 2006 low risk births. *Am J Obstet Gynecol*. 2010;202:152.e1-5.
25. Snowden JM, Tilden EL, Snyder J, Quigley B, Caughey AB, Cheng YW. Planned out-of-hospital birth and birth outcomes. *N Engl J Med*. 2015;373:2642-2653.
26. DeClercq E. The absolute power of relative risk in the debates on repeat cesareans and home birth in the United States. *J Clin Ethics*. 2013;24(3):215-224.
27. Cheyney M, Bovbjerg M, Everson C, Gordon W, Hannibal D, Vedam S. Outcomes of care for 16,924 planned home births in the United States: the Midwives Alliance of North America Statistics Project, 2004 to 2009. *J Midwifery Womens Health*. 2014;59(1):17-27.
28. American College of Nurse-Midwives. Clinical Bulletin No. 14: midwifery provision of home birth services. *J Midwifery*

Womens Health. 2015;61(1):127-133.
29. American College of Nurse-Midwives. Planned home birth. Position statement. December 2016. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000251/Planned-Home-Birth-Dec-2016.pdf. Accessed November 3, 2017.
30. American College of Obstetricians and Gynecologists. Committee Opinion No. 697: planned home birth. *Obstet Gynecol.* 2017;129:e117-e122.
31. World Health Organization, Maternal and Newborn Health/Safe Motherhood Unit. *Care in Normal Birth: A Practical Guide*. Geneva, Switzerland: World Health Organization; 1996.
32. Bailes A. *The Home Birth Practice Manual.* 3rd ed. Washington, DC: American College of Nurse-Midwives; 2016.
33. Nieuwenhuijze M, Low LK. Facilitating women's choice in maternity care. *J Clin Ethics*. 2013;24(3):276-282.
34. Avery MD. *Supporting a Physiologic Approach to Pregnancy and Birth: A Practical Guide*. Ames, IA: John Wiley & Sons; 2013.
35. Murphy PA, Fullerton J. Outcomes of intended home births in nurse-midwifery practice: a prospective descriptive study. *Obstet Gynecol.* 1998;92:461-470.
36. American College of Nurse-Midwives. Clinical Bulletin No. 13: intermittent auscultation for intrapartum fetal heart rate surveillance. *J Midwifery Womens Health*. 2015;60(5):626-632.
37. Cox K, Bovbjerg ML, Cheyney M, Leeman LM. Planned home VBAC in the United States, 2004–2009: outcomes, maternity care practices, and implications for shared decision making. *Birth*. 2015;42(4):299-308.
38. Boucher D, Bennett C, McFarlin B, Freeze R. Staying home to give birth: why women in the United States choose home birth. *J Midwifery Womens Health*. 2009;54(2):119-126.
39. Tilden EL, Cheyney M, Guise JM, et al. Vaginal birth after cesarean: neonatal outcomes and United States birth setting. *Am J Obstet Gynecol.* 2017;216:403e1-403e8.
40. National Institutes of Health Consensus Development Conference statement: vaginal birth after cesarean: new insights. March 8–10, 2010. *Obstet Gynecol.* 2010;115(6):1279-1295. Available at: https://consensus.nih.gov/2010/images/vbac/vbac_statement.pdf. Accessed September 19, 2017.
41. Lieberman E, Ernst EK, Rooks JP, Stapleton S, Flamm B. Results of the national study of vaginal birth after cesarean in birth centers. *Am J Obstet Gynecol.* 2004;10(5)4:933-942.
42. Bovbjerg ML, Cheyney M, Brown J, Cox KJ, Leeman L. Perspectives on risk: assessment of risk profiles and outcomes among women planning community birth in the United States. *Birth*. 2017;44:209-221.
43. American College of Nurse Midwives. Clinical Bulletin No. 12: care of women desiring vaginal birth after cesarean. *J Midwifery Womens Health*. 2011;56(5):517-525.
44. Weiner GM, Zaichkin J, Kattwinkel J, et al. *Textbook of Neonatal Resuscitation*. 7th ed. Elk Grove Village, IL: America Academy of Pediatrics and American Heart Association; 2016.
45. Home Birth Summit. Best practice guidelines: transfer from planned home birth to hospital. Available at: https://www.ftc.gov/system/files/documents/public_comments/2014/04/00173-90022.pdf. Accessed August 16, 2017.
46. Kennedy HP. The essence of nurse-midwifery care: the woman's story. *J Nurse-Midwifery.* 1995;40:493-507.

PART VI

Postpartum

CHERYL TATANO BECK

A wealth of knowledge is readily available in this postpartum section for midwives to assist them in providing optimal care to new mothers. The *Anatomy and Physiology of Postpartum* chapter clearly identifies the complex processes a woman's body goes through to achieve physiologic adaptations after giving birth. Every system is involved in restoring a woman's body to its nonpregnant state: cardiovascular, hematologic, respiratory, renal, hepatic, gastrointestinal, musculoskeletal, integument, metabolism, and endocrine. The comprehensive material in this chapter provides midwives with an excellent foundation to provide optimal postpartum care—the topic that is the focus of the *Postpartum Care* chapter. In this chapter, postpartum care is addressed in an extremely organized progression, beginning with the first hour after birth, moving to the initial postpartum period (first 72 hours), and finally in the mid-postpartum period (from discharge to 6 weeks after birth). The *Postpartum Complications* chapter then provides midwives with up-to-date vital information on the diagnosis and treatment of an array of complications that women may experience after giving birth, such as endometritis, preeclampsia/eclampsia, secondary postpartum hemorrhage, and thrombophlebitis. The last chapter in this section, *Breastfeeding and the Mother–Newborn Dyad*, describes the health benefits for breastfeeding women and infants, barriers and facilitators of breastfeeding, contraindications, pharmaceuticals and breastfeeding, a systematic assessment, and problems related to breastfeeding.

As noted at the very beginning of this *Postpartum* section, a mother's physical well-being can influence her mental well-being. In the postpartum period, women's physical and mental well-being are intricately intertwined. Although perinatal mental health disorders are also addressed in the *Mental Health Conditions* chapter to provide a comprehensive summary of the mental health care provided by midwives, this topic is an especially critical consideration during care of women in the postpartum period.

Over the past few decades, postpartum depression has become more widely recognized as a perinatal mental health disorder. Nevertheless, traumatic childbirth and the post-traumatic

stress disorder (PTSD) that can develop as a result of such trauma are all too often overlooked. An estimated 45% of postpartum women have reported experiencing a traumatic birth.[1] In addition, in a recent meta-analysis Yildiz et al. reported the mean prevalence rate of PTSD related to birth trauma as 4.0% in community samples and 18.5% in high-risk samples.[2] Just as in the general population, comorbidity of depression and PTSD can exist in women during the postpartum period. The physical complications addressed in this section certainly should be considered in addition to the impact on women's mental health outcomes as described in the *Mental Health Conditions* chapter.

Furthermore, the relationship between postpartum complications and mental health outcomes is complex. In the Listening to Mothers II U.S. national survey, the presence of more physical problems since birth was significantly related to elevated post-traumatic stress symptoms in mothers.[3] Postpartum complications and distressing symptoms are strongly associated with PTSD and depression.

It may not be readily apparent to a midwife that a woman perceives her birth to have been traumatic. Just like beauty, traumatic childbirth is in the eye of the beholder.[4] How a woman perceives her birth may be quite different than how that birth is viewed by the midwife who was present at the birth. It is critical that midwives identity as soon as possible those mothers who are struggling with the aftermath of birth trauma so that interventions can be started quickly. Traumatic childbirth can have long-term chronic consequences. The minutes or hours during the birthing process that a woman perceives as traumatic can be likened to a pebble dropped into the water, resulting in ripples spreading out.[5] Some of these ripples resulting from traumatic childbirth can negatively impact mothers' breastfeeding experiences, the anniversary of their birth trauma, and their subsequent births.

Traumatic births can impact not only the women who give birth, but also the midwives who care for them. There can be a cost to clinicians who care for patients who have been traumatized. This so-called secondary traumatic stress has been well documented in midwives.[6] A birth affects every individual present, and those effects can be lifelong. As we care for women during the postpartum period, we also need to remember to care for ourselves.

References

1. Alcorn KL, O'Donovan A, Patrick JC, Creedy D, Devilly GJ. A prospective longitudinal study of the prevalence of posttraumatic stress disorder resulting from childbirth events. *Psychol Med*. 2010;40:1849-1859.
2. Yildiz PD, Ayers S, Phillips L. The prevalence of posttraumatic stress disorder in pregnancy and after birth: a systematic review and meta-analysis. *J Affect Dis*. 2017;208:634-645.
3. Beck CT, Gable RK, Sakala C, Declercq ER. Posttraumatic stress disorder in new mothers: results from a two-stage U.S. national survey. *Birth*. 2011;38:216-227.
4. Beck CT. Birth trauma: in the eye of the beholder. *Nurs Res*. 2004;53:28-35.
5. Beck CT. Middle range theory of traumatic childbirth: the ever-widening ripple effect. *Glob Qual Nurs Res*. 2015;1-13.
6. Beck CT, LoGiudice J, Gable RK. A mixed methods study of secondary traumatic stress in certified nurse-midwives: shaken belief in the birth process. *J Midwifery Womens Health*. 2015;60:16-23.

CHAPTER

32

Anatomy and Physiology of Postpartum

TEKOA L. KING

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Introduction

Physiologically, the postpartum period begins with expulsion of the placenta and continues through to the involution of the uterus and return of other reproductive organs to a nonpregnant state. Postpartum is not a return to the prepregnancy state because pregnancy itself has some lasting effects on a woman's body.

Classically, the postpartum period is considered to last between 6 and 8 weeks. This time period is not sharply delineated, however, and varies among women. Among breastfeeding women, the anatomy and physiology of the mammary glands will continue to be different than in nonpregnant, nonlactating women for the duration of lactation and beyond.

A woman's ability to complete the important tasks of bonding and caring for her new infant(s)—or, in the case of a stillbirth or other loss, the task of grieving and healing—are affected to a significant degree by her physical well-being. Her physical well-being can also influence her risk for developing postpartum depression, post-traumatic stress disorder, or another perinatal mood disorder.

Anatomy and Physiology of Lactation

Ramsay's ultrasound studies of lactating breasts "in action" have profoundly changed current knowledge of the process of milk production and release.[1,2] The parenchyma includes the milk ducts—structures that have been described as "tangled tree roots." Random and intertwined, the milk ducts often intersect as they reach the nipple/areolar complex. The 9 to 10 ducts open onto the surface of the nipple and the lobular alveolar structure. The presence of "lactiferous sinuses" where the ducts terminate under the nipple are no longer considered to be distinct anatomic structures. The stroma includes the connective tissue, fat (adipose) tissue, blood vessels, lymphatics, and Cooper's ligaments. The glandular tissues (lactocytes, alveoli, and supporting structures) where the milk is formed are interspersed throughout the breast, with more than 65% of the glandular tissues being located in a 30-mm radius from the base of the nipple.[1] During lactation, the breast weighs an average of 600–800 grams.

The functional milk-making unit is the alveolus. The lactocytes—that is, the alveolar epithelial cells—convert nutrients from the maternal circulation into various milk components and secrete milk into the lumen of the alveolar sac (**Figure 32-1**). The alveoli are surrounded by myoepithelial cells that contract under the influence of oxytocin, causing milk to be released into the ductules and ducts and to move through pores toward the nipple. The Montgomery glands just under the areola secrete volatile compounds that elicit specific infant behaviors, including increased sleepiness, head turning, and appetite response; the secretion of these compounds has been proposed as one of the stimuli that promote the so-called breast crawl, which refers to the observation that the newborn may spontaneously crawl toward the breast after birth if placed skin-to-skin on the mother's abdomen and chest.[3]

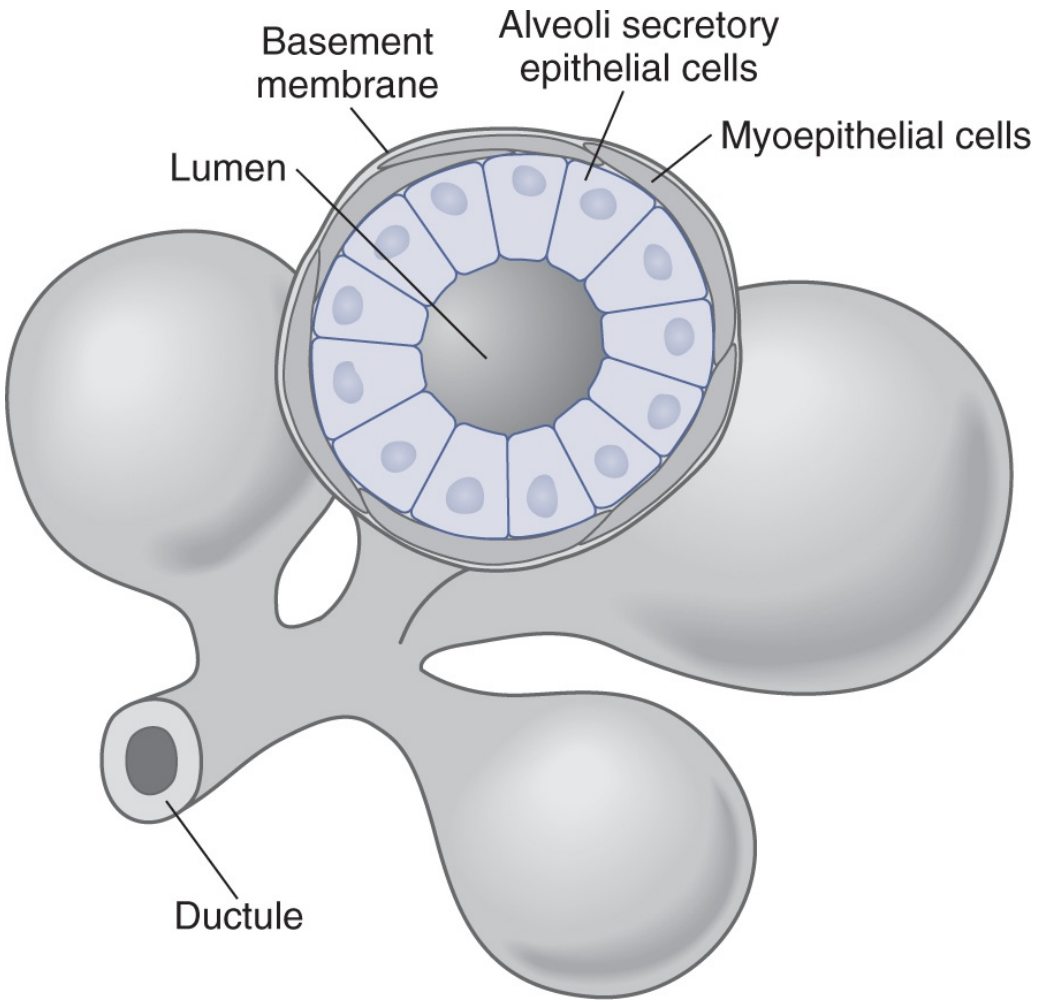


Figure 32-1 Anatomy of the alveoli of the breast.

Lactogenesis

Lactogenesis, which refers to the ability to secrete milk, occurs in four stages (**Table 32-1**). *Lactogenesis I* (secretory initiation) starts during the second half of pregnancy, whereas *lactogenesis II* (secretory activation) occurs in the first postpartum days. *Lactogenesis III* refers to the time when lactation is fully established. *Lactogenesis IV* refers to the mammary gland involution that occurs when breastfeeding ceases.

Table 32-1 Stages of Lactogenesis

Stage	Description	When This Stage Occurs
Lactogenesis I	Cytologic and enzymatic differentiation of alveolar cells; uptake of the substrates for milk begins; colostrum production begins with small fat droplets and proteins.	Begins at approximately 16 weeks' gestation and continues through a few days or even weeks after birth.
Lactogenesis II	Lactose secretion begins and fluid volume increases. Colostrum is essentially diluted by lactose and fluids, becoming "mature" milk.	Occurs approximately 30 to 72 hours postpartum; may be delayed as long as 96 hours after cesarean birth.

Lactogenesis III	Ongoing copious secretion of milk in response to milk removal from the breast by the baby or other means.	Ongoing until weaning and involution.
Lactogenesis IV	Involution of mammary gland and apoptosis of alveolar cells.	Begins when milk removal from the breast diminishes or ends. Some milk may be produced for many weeks or longer after overt production ends.

Lactogenesis I: Development During Pregnancy

By the end of the first trimester, the alveoli epithelial cells (lactocytes) begin secreting proteins and fat (colostrum), placental lactogen, progesterone, estrogen, and prolactin. In the second and third trimesters, the duct system expands and the number and activity of the lobules increase. Veins become increasingly visible on the skin surface of the breast, and women may observe some colostrum leaking from their nipples. By 16 to 18 weeks' gestation, colostrum secretion has begun in preparation for the subsequent onset of copious secretion of milk. Patterns of breast growth during pregnancy vary, and are unrelated to the woman's nutritional status.

Lactogenesis II: Onset of Copious Milk Production

The sudden drop in the plasma progesterone level that occurs once the placenta is expelled triggers the second stage of lactogenesis, which features the onset of copious milk secretion ("milk coming in") and the subsequent delivery of milk to the infant. The lactose secretion in the lactocytes, in combination with osmosis of fluid from the interstitial spaces, causes milk volume to rapidly increase, beginning approximately 30–40 hours after birth.

Lactogenesis II involves both milk synthesis and milk ejection/release (or milk production), with at least two different hormones working in tandem to coordinate these functions. Suckling stimulates the secretion of prolactin by the anterior pituitary. Plasma concentrations of prolactin increase rapidly once suckling starts. The increased prolactin, in turn, initiates milk secretion by the alveolar cells. Suckling also causes the posterior pituitary to release oxytocin, which causes the myoepithelial cells to contract, ejecting the milk from the alveoli and lobules into the ducts.

Oxytocin plays many complex roles in women. It has been called the "tend and befriend" hormone because oxytocin is associated with a calming effect on the lactating woman and infant, stabilization of maternal blood pressure, lower cortisol levels, increase in trust and facial recognition, and decrease in anxiety and aggressive behaviors.[4-6]

Several risk factors may lead to delayed onset of lactogenesis II beyond 72 hours postpartum, including obesity, diabetes, polycystic ovary syndrome, and other biologic conditions that are associated with increased androgen levels.[7-12] Delayed onset of lactogenesis II is also associated with cesarean birth, retained placental fragments, ovarian theca lutein cysts, hypothyroidism, certain types of breast surgery, and severe maternal anemia.[9] The actual etiology of delayed lactogenesis II in women with these conditions is not fully understood and may be multifactorial. For example, women with obesity are at higher risk

for interventions that delay lactogenesis, such as cesarean birth.[8]

Although infant factors such as prematurity or newborn illness that disallows early suckling can negatively affect the normal onset of lactogenesis II, early milk expression (within 1–2 hours after birth) can ameliorate the situation and provide nutrition for the infant.[13] Newborns with an ineffective or weak suck, palate abnormalities, tongue-tie, and congenital heart defects may not be able to effectively remove colostrum; when the infant has any of these conditions, expression of milk should begin within the first 1–2 hours to support normal lactogenesis and milk intake by the infant.[6-8]

Lactogenesis III: Ongoing Milk Synthesis

Lactogenesis III, or galactopoiesis, is the ongoing maintenance of milk production, which includes a switch to autocrine or local control of lactation once lactation is fully established. The mechanism is as follows: Nipple stimulation via infant suckling triggers prolactin release from the anterior pituitary and stimulates the hypothalamus to produce oxytocin. The oxytocin is released from the posterior pituitary.[14] Prolactin stimulates the lactocytes to produce milk, and oxytocin stimulates contraction of the myoepithelial cells surrounding the alveoli, which causes milk ejection into the lactiferous ducts (**Figure 32-2**). Oxytocin is also released through sensory pathways when the mother sees, feels, touches, smells, or hears stimuli that remind her of the infant and breastfeeding.[15]

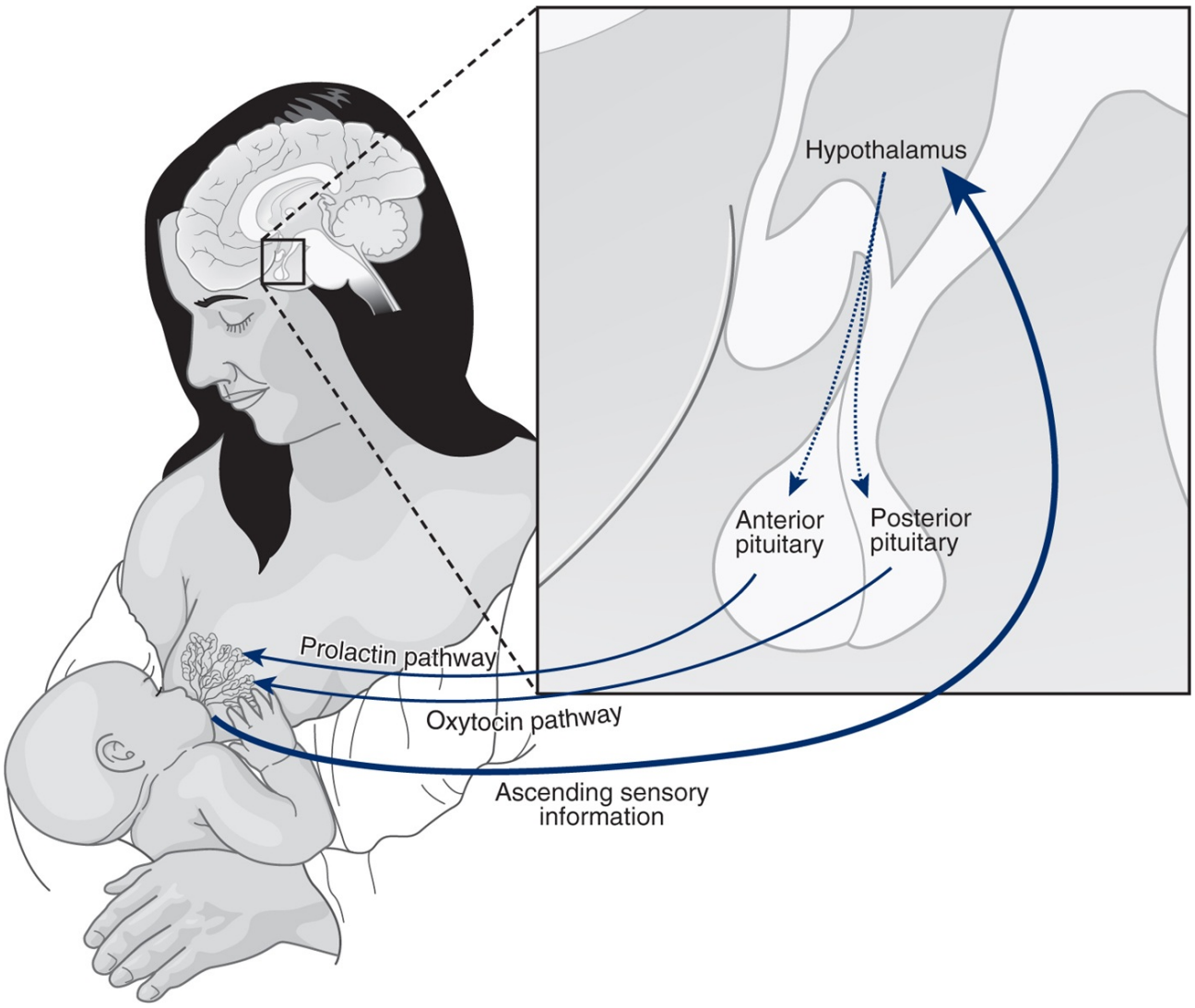


Figure 32-2 Autocrine control of lactation: Infant suckling triggers a nerve impulse that terminates in the hypothalamus. The hypothalamus then (1) manufactures oxytocin and signals the anterior pituitary to release oxytocin and (2) signals the anterior pituitary to release prolactin. Oxytocin is released first and thus the myometrial cells around the alveoli contract and eject premade milk. Prolactin initiates formation of breast milk.

In addition, milk removal stimulates milk synthesis. If milk is not removed from the breast, secretion first slows and then ceases over a period of days.[16] When the milk is not removed, inflammatory pressure causes synthesis and release of a milk protein known as the feedback inhibitor of lactation. This protein downregulates the cell surface prolactin receptors on the lactocytes.[17] In this way, the rate of milk synthesis and the total daily amount of milk produced are closely related to the degree of "emptiness" of the breast, such that milk production varies during the day and night, and from one breast to the other.[18]

The composition of breast milk is one of the more fascinating aspects of this biology, as it varies according to the individual infant's needs. The primary components of breast milk are lactose, oligosaccharides, fats, proteins, minerals, and other bioactive agents such as immune globulins that provide passive immunization. These compounds have multiple functions in addition to providing nutrition. For example, breast milk has bacteriostatic, anti-inflammatory,

and immune-modulating properties in the infant's intestine. Likewise, breast milk stimulates gastrointestinal motility and enhances the maturity of the infant's intestinal tract. Other components of breast milk include neutrophils, lymphocytes, epithelial cells, and macrophages. The macrophages are capable of pinocytosis and chemotaxis, while the lymphocytes direct some immune reactions and development of cell-mediated immunity in the infant.

In brief, breastfeeding has important short-term and substantial long-term benefits for the health of the newborn and infant. Some of these benefits reflect physiologic actions of breast milk, whereas others may be more likely the indirect effects of the maternal–infant interactions. The interested reader is referred to recent reviews, [19-27] and the *Breastfeeding and the Mother–Newborn Dyad* chapter for more in-depth reviews of the benefits of breastfeeding.

Lactogenesis IV: Involution

The process of mammary involution occurs after lactation ceases. Most of the anatomic studies of mammary involution have been performed on mice, so the exact chain of physiologic events that occur in humans is not definitively known.[28] When the infant no longer suckles at the breast, prolactin levels fall in the mother and the effects of prolactin on breast milk production end. The early stages of involution appear to be reversible if suckling resumes. Otherwise, the mammary gland involutes, or regresses through the process of mammary epithelial cell apoptosis (cell self-destruction). Any remaining secretion is absorbed, and adipose tissue from the lactocytes is redeposited within the breast. In most cases, involution takes approximately 6 weeks after milk removal ceases, although the human breast never fully returns to a prepregnant condition.[28]

Gradual weaning invokes a different process of inhibition of lactation and mammary involution that does not involve acute milk stasis, wherein a progressive autocrine inhibition of milk production occurs in response to gradually decreased intake by the infant.[28] Regardless of whether a woman breastfeeds her child, some of the mammary epithelium that was produced during pregnancy is retained. The breasts, therefore, never return to their prepregnant state.

Reproductive Organ Involution

Immediately following birth, the uterus is approximately 20 centimeters in length, weighs approximately 1 kg, and is in a retroverted position.[29] The uterine fundus can be palpated as a firm mass in the abdominal midline several centimeters below the umbilicus in the first hours after birth. In the first few days postpartum, the uterus lies in a slightly retroverted position that "drapes" over the sacral promontory, and is easily palpable in the abdomen.[30] As the uterus contracts further, it undergoes a rotation of 100–180° along the axis of the internal os and assumes an upright position by postpartum day 7.[30] By the end of the first week postpartum, the uterus weighs approximately 500 grams and may still be palpable above the symphysis pubis. By 10 to 14 days postpartum, the uterus weighs approximately 300 grams; at this point, it has typically taken on its nonpregnant anatomic position in the pelvis, which in most women is in an anteverted inclination, and is no longer able to be palpated abdominally (**Figure 32-3**).

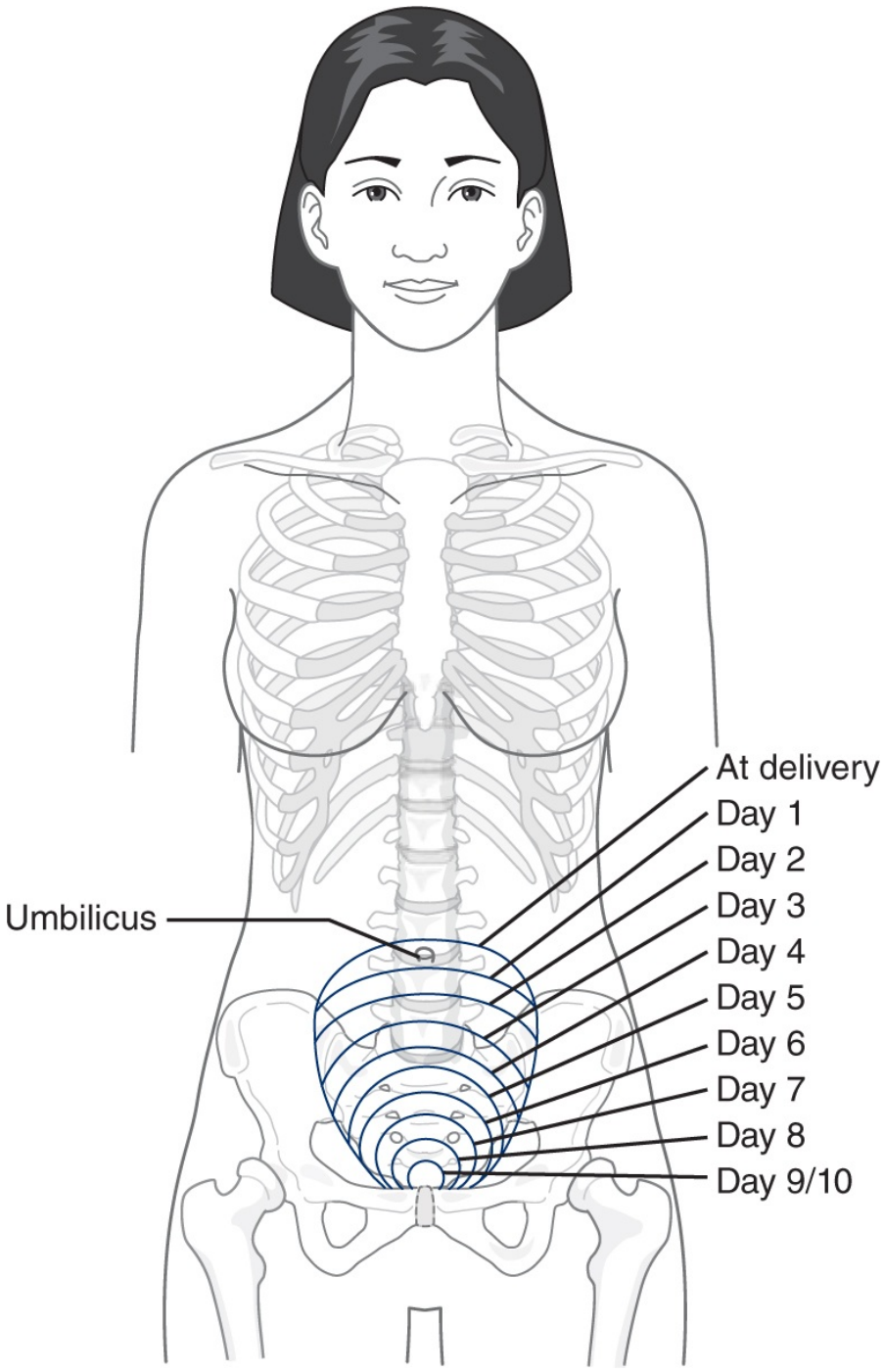


Figure 32-3 Postpartum involution of the uterus. The fundal height descends toward the pelvis at a rate of approximately 1 centimeter per day.

Postpartum Hemostasis

Following placental separation and expulsion from the uterus, a series of events must occur to ensure maternal hemostasis at the placental attachment site. This series of events begins prior to placental separation, with oxytocin-induced contractions of the myometrium that cause a permanent and progressive shortening of the muscle fibers. In addition to promoting the separation of the placenta from the uterine wall, this shortening and retraction of muscle fibers

compresses the maternal arteries that served as the vascular supply to the placenta.[31] As the myometrium contracts, the uterine walls press against each other, which may also promote hemostasis.

The mechanical compression caused by myometrial contractions is the primary mechanism of hemostasis in the immediate postpartum period. In addition, a clotting response is initiated at the time of placental expulsion that further contributes to hemostasis at the placental attachment site. Uterine atony during the first hour following delivery can lead to massive maternal hemorrhage, especially if the contractions are inadequate.

During pregnancy, changes in the coagulation and fibrolytic systems promote coagulation via an increase in platelet activity, a decrease in fibrinolysis, and an increase in coagulation factors. This “hyper-coagulable” state encourages and supports adequate coagulation at the placental attachment site and protects against hemorrhage following birth. In brief, during pregnancy (and particularly in the third trimester) the woman experiences an increase in a number of clotting factors (I, II, VII, VIII, IX, and XII), a decrease in protein S levels, and a progressive fall in the activity of activated protein C.[32] Postpartum, these clotting factors promote coagulation of the sheared maternal vessels at the placental attachment site. However, while the clotting factors protect a woman from hemorrhage, they also predispose her to thromboembolic events.[33,34] The relative proportions of coagulation factors appear to return to their nonpregnant state by 3 weeks postpartum, but certain clotting cofactor levels, such as protein S levels, may not return to normal levels until 8 weeks postpartum or later.[33,35]

Uterine Involution

Uterine involution is the process of uterine contraction, myometrial cell shrinkage, and reestablishment of the endometrium. The uterus involutes by approximately 50% in the first 24 to 48 hours postpartum and then gradually diminishes to the nonpregnant size over the next 6 to 8 weeks.[29] This process takes longer in multiparous women than in primiparous women.[36]

During involution, the uterus loses mass secondary to myometrial cell autolysis. The result is a marked decrease in the size—rather than the number—of myometrial cells. Approximately 90% of the excess cellular protein is broken down by proteolytic enzymes that are released by myometrial cells, endothelial cells of uterine blood vessels, and macrophages.

The uterine decidua is divided into multiple layers (strata) during pregnancy. Postpartum, the placenta separates at the spongy portion of the layer known as the decidua basalis, most of which is sloughed off with the placenta or in the early postpartum period. In the second and third postpartum days, the remaining decidua becomes differentiated into two layers, identified as the superficial and basal layers. The superficial layer is formed through leukocyte invasion and protects the desquamated endometrium from infection. This layer becomes necrotic and is sloughed off. In contrast, the basal layer remains intact and gives rise to the new endometrium through proliferation of the remaining glandular and stromal tissue. By 1 week postpartum, the endometrial surface has become epithelized; by 2 to 3 weeks postpartum, the endometrium has regenerated and resembles a nonpregnant endometrium, excluding the placental implantation site. The remaining thrombosed vessels at the placental implantation site take approximately 6 weeks to heal through an “exfoliation” process of sloughing infarcted and necrotic tissue,

followed by a process of endometrial remodeling.

Lochia

The postpartum vaginal discharge that begins immediately at birth and continues for approximately 4 to 8 weeks is termed lochia. The mean total duration of lochia is 33 days.[37–39] Lochia is further described as lochia rubra, lochia serosa, and lochia alba based on its changing color.[39] This color results from the changing composition of the tissue that is sloughed and expelled in the postpartum endometrial restoration process.

Initially, the lochia consists primarily of blood; as a result, *lochia rubra* is red or brownish red in color. As the uterine bleeding decreases at approximately 3 to 5 days postpartum, leukocyte infiltration occurs and the placental implantation site undergoes exfoliation. At this point, the discharge contains some blood but primarily wound exudate and leukocytes; therefore, *lochia serosa* is a pinkish brown color. The median duration of lochia serosa is 22 days.[37,38] Some women will experience a transient increase in bleeding 7 to 14 days postpartum secondary to sloughing of the placental eschar (scab) at the placental site. Toward the end of the uterine involution process, the endometrium has been primarily restored. *Lochia alba* is composed predominantly of leukocytes and some decidual cells and is white or yellowish white in color.

Cervical and Vaginal Involution

During labor, the maternal cervix undergoes remodeling to become soft and dilated; it also shortens so that it is not palpable as distinct from the lower uterine segment. The first two stages of remodeling are described in more detail in the *Anatomy and Physiology During Labor and Birth* chapter. After the birth of the newborn, the cervix begins to reconstitute. At this point, it may appear edematous and have sustained lacerations. Internally, the extracellular matrix is repaired and the epithelial barrier restored during this process.[40] By the end of the first week postpartum, the endocervical canal has reappeared and the os is only approximately 1 centimeter dilated. The external cervical os usually does not return to its previous nonpregnant form with the dimple shape characteristic of most cervices of nulliparous women, but rather takes the form of a slit (**Figure 32-4**). The cervical endothelium also undergoes remodeling during the postpartum period, which has been associated with a high incidence of spontaneous regression of high-grade cervical intraepithelial lesions.[41]

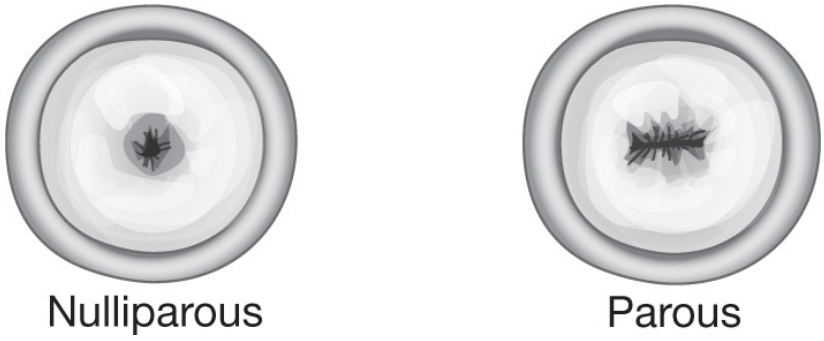


Figure 32-4 Postpartum anatomic changes in the cervix.

The vagina is initially edematous and bruised after a vaginal birth and has lost its prepregnant tone and rugae. The vaginal rugae return at approximately 3 to 4 weeks postpartum, but the prepregnant vaginal tone may never be completely restored. The vaginal epithelium is usually healed by 6 to 10 weeks postpartum. Following a woman's first vaginal birth, the hymeneal ring is replaced by hymeneal tags and the vaginal opening becomes visible rather than closed by the labia minora.

Wound Healing

Some women experience perineal lacerations during the process of vaginal birth, or from the use of forceps, vacuum, or cutting of an episiotomy. Identification, categorization, and repair of perineal and vaginal lacerations are described in the appendices of the *Second Stage of Labor and Birth* chapter. The wound healing process is divided into three phases: hemostasis and inflammation, granulation and proliferation, and remodeling.[42]

Hemostasis and Inflammation

The hemostatic process begins immediately at the time of injury and continues for a few days. First, platelets adhere to damaged tissue and combine together to form a platelet plug. Fibrinogen is attracted to this platelet plug, where it is subsequently cleaved into fibrin via the action of thrombin. Fibrin is an elastic, insoluble protein that forms an interlacing fibrous network that stabilizes the area of blood vessel injury. Platelets also release growth factors that call fibroblasts to migrate to the area, as well as cytokines that mediate the initial inflammatory response.

The inflammatory response begins within the first 6 to 8 hours following the perineal injury.[42] Polymorphonuclear neutrophils (PMNs) from blood vessels in and surrounding the injured area migrate to the area of injury. These white blood cells work to rid the area of bacteria, damaged cells, and cell debris. The PMNs also assist in recruiting monocytes to the area, which then differentiate into macrophages and cleanse the area of foreign cells as well as damaged and dead cells via the process of phagocytosis. Unlike the PMNs, which are part of the initial inflammatory response and are short-lived, macrophages can live for months. After approximately 3 or 4 days, in addition to their work in ridding the area of debris and foreign and dead cells, the macrophages begin to release key growth factors that trigger the growth of new tissue in the area. During this phase, the woman may experience swelling, burning, or pruritus in the area in which the laceration occured.

Granulation and Proliferation

Approximately 5 to 7 days after injury, the fibroblasts that were recruited in the initial inflammatory response migrate to the area of the wound and begin to lay down the extracellular matrix and collagenous tissue in the area that serve as the “framework” or scaffold for the rebuilding of the new tissue. Revascularization (angiogenesis) is essential to feed the new (granulation) tissue with nutrients and oxygen. New vessels arise as branches from intact vessels in the area. Regrowth of the epithelial layer occurs as epithelial cells migrate to this layer from the edges of the injured area or from adjacent tissues. This process can take up to 4 to 5 weeks.

As the granulation tissue grows to fill the wound, the area will take on a red or pebbled pink appearance, particularly if the tissue is healing via secondary intention (not approximated by sutures). This new tissue is highly vascular and friable. As part of health education, the woman should be advised to keep the area clean and to blot the area dry after urinating/defecating or

after washing. Blotting is preferred instead of wiping, to avoid damaging this new tissue. Women who have sutures in place should be made aware that, depending on the suturing material, it may take 6 weeks or longer for the sutures to be fully absorbed.

Remodeling

During the last phase of wound healing, the collagen tissue undergoes remodeling to become similar to the prebirth tissue; this phase can last for years. Many of the new blood vessels created during the proliferative stage of wound healing begin to regress. Wound contracture occurs secondary to the action of specialized contractile fibroblasts known as myofibroblasts. The collagen scar tissue, and particularly the initial scar tissue that has not undergone remodeling, will feel thicker and less distensible—a change that may contribute to postpartum dyspareunia. As the healing process continues, some women describe the scar tissue as transitioning from feeling painful to feeling numb. With time, the tissue is likely to regain most of its elasticity and sensation. The scar tissue is not as strong or elastic as normal tissue, however, and there is an increased risk of lacerations along the scar tissue with subsequent births.

Postpartum Physiology in Nonreproductive Organs

Pregnancy results in anatomic and/or physiologic changes in every organ system of the body, all of which are altered again during the puerperium (**Table 32-2**).[43-53] These physiologic changes may disappear over time depending on the organ involved and the underlying health of the individual woman. Because of their importance and profound changes, the postpartum changes that affect the cardiovascular and endocrine systems are described here in detail.

Table 32-2 Postpartum Maternal Physiologic Adaptations

Postpartum Change	Timing of Return to Nonpregnant Status	Clinical Notes
Cardiovascular		
Auto-transfusion of 10% to 15% of blood volume secondary to removal of the placenta and shifting of extracellular fluid into the intravascular system. Stroke volume and cardiac output increase by 80%.	Immediate	Women with hypertension, preeclampsia, or cardiac disease have an increased risk for pulmonary edema or cardiac failure in the first 24–48 hours postpartum.
Cardiac output is increased secondary to the increased flow of blood back to the heart from the loss of the uteroplacental circulation, decreased pressure from the pregnant uterus, and mobilization of extracellular fluid.	Cardiac output increases during the first 48 hours after birth, then slowly returns to normal by 6–12 weeks postpartum.	Women with some types of cardiac disease can be at risk during the postpartum period for cardiac failure, pulmonary edema, and other cardiac abnormalities.
Plasma volume slowly returns to nonpregnant values.	2 weeks postpartum	Women have a natural diuresis and diaphoresis in the initial postpartum period.
Hematologic		
Impaired fibrinolysis normalizes.	24–48 hours	
WBC levels normalize.	6 days	WBCs are elevated in labor and gradually return to normal in the puerperium.
Coagulation factors normalize.	4–6 weeks	An increased risk for thromboembolic events is present for the first several weeks postpartum.
Platelet function normalizes.	12 weeks	
Respiratory		
Removal of the weight of the gravid uterus at birth relieves intra-abdominal pressure and allows for elevation of the diaphragm, so that normal excursion of the diaphragm is reestablished.	Immediate	The dyspnea related to pregnancy usually resolves rapidly after birth. Therefore, complaints of shortness of breath or chest pain in the postpartum period should be evaluated immediately, especially because of the increased risk of thromboembolic events in the postpartum period.
Decrease in tidal volume back to normal.	Immediate	
Loss of placental progesterone production, a mediator of many		

respiratory changes of pregnancy, leads to a rapid return of normal nonpregnant respiratory parameters.		
Return of normal RV and ERV that were decreased during pregnancy.	RV returns immediately; ERV takes several months to return to nonpregnant values.	
Renal		
Gradual return to nonpregnant values of GFR, renal plasma flow, plasma creatinine, BUN, and creatinine clearance, all of which were altered during pregnancy.	2–3 months postpartum	Alterations may reflect changes of pregnancy that have not returned to nonpregnant levels.
Mild proteinuria may develop in the first few postpartum days.	3–5 days	New-onset proteinuria may be related to normal postpartum changes. However, evaluation for preeclampsia should occur if new-onset proteinuria is noted in the first 2–3 weeks postpartum.
Natriuresis (sodium excretion) and diuresis (increased urine production) lead to a decrease in blood volume, causing it to return to nonpregnant levels.	3 weeks or less	Oxytocin promotes reabsorption of water. The decrease in oxytocin contributes to postpartum diuresis. Urine production in postpartum days 2–5 can reach 3000 mL. In addition to increased urine production, many women will experience episodes of diaphoresis in the first postpartum week.
Gradual return of bladder tone and normal size and function of the bladder, ureters, and renal pelvis, all of which were dilated during pregnancy.	6–8 weeks, although it may take longer	Changes of pregnancy and trauma, including interventions, during the birth of an infant can lead to an increased risk of urinary tract infections and urinary retention during the postpartum period.
Hepatic		
Liver function indices (AST, ALT, LDH) that increased during labor return to normal.	3 weeks or less	Interpretation of liver function tests drawn in the postpartum period must reflect the knowledge that alterations from pregnancy and/or labor may not have returned to nonpregnant levels.
Alkaline phosphate, which increased during pregnancy, returns to normal.	6 weeks	
Gastrointestinal		
Gastric tone and motility remain decreased for 2–3 days postpartum.	Bowel movements resume 2–3 days postpartum; normal GI function and bowel patterns return in 1–2 weeks postpartum	Decreased GI motility in the postpartum period can lead to abdominal distension, constipation, and, in severe cases, ileus.
Musculoskeletal		
Ligaments and muscles are lax, so the abdominal wall remains soft and flaccid in the immediate postpartum. Separation of the rectus muscle (diastasis recti abdominis) may be present.	Variable	The recovery of abdominal muscular tone and resolution of diastasis recti abdominis are somewhat related to activity/exercise.

Musculoskeletal		
Muscles and associated tissues that were stretched during childbirth gradually regain tone.	Variable, but can take months to years	Stretching and injury of the pelvic and perineal musculature and associated structures increase the risk of urinary and anal incontinence as well as pelvic organ prolapse. Prevention of these complications includes avoidance of episiotomy and performance of postpartum pelvic exercises.
Integument (Skin and Hair)		
Hyperpigmentation (including linea nigra and melasma), striae gravidarum (stretch marks), varicosities, and other skin changes of pregnancy fade or regress.	6 months or less	Many skin changes do not disappear completely. Striae commonly progress from red to silvery white in color in approximately 3 months.
With return of estrogen levels to nonpregnant levels, hair growth slows and follicles enter into a hair-shedding phase, which results in a period of alopecia or postpartum telogen effluvium.	Normal hair growth is reestablished by 4–6 months postpartum for most women	Reports of hair loss in women in the postpartum period are common, and the provider can offer reassurance. Women with excessive hair loss leading to bald patches or with hair loss lasting longer than 6 months should be evaluated for thyroid dysfunction or nutritional deviations.
Metabolism		
Demand for nutrients and hormone levels drop abruptly. Maternal metabolism of nutrients begins to return to normal. Maternal insulin resistance seen during pregnancy begins to resolve shortly after removal of the placenta.	Fasting glucose plasma levels and plasma free fatty acid levels return to prepregnant levels in the first 3–5 days for women with normal glucose metabolism in pregnancy; triglyceride levels normalize within 2–3 weeks	Insulin resistance resolves nearly immediately following birth, which may require medication adjustment for women with Class A2, GDM. Maternal glucose values should be back to normal by the 6-week postpartum visit. Women with GDM should be screened for continued glucose intolerance at 6–12 weeks postpartum.
The increase in intestinal calcium absorption seen during pregnancy resolves to nonpregnant levels of calcium absorption.	6 weeks in nonlactating women	In women who are lactating, increased calcium needs for breast milk are supported by increased reabsorption of calcium from the skeleton. This process is associated with a reversible decrease in maternal bone density.
Endocrine		
The thyroid-binding globulin level decreases, which leads to a drop in T_3 and T_4.	First 3–4 days	After giving birth, women are at increased risk for Graves' disease, postpartum thyroiditis, and thyroid storm. Diagnosis can be complicated.
Endocrine		
FSH and LH levels are low	2 weeks Nonpregnant values return by 4–6 weeks in nonlactating women	Ovulation is highly unlikely in the first 2 weeks postpartum. Return of FSH and LH levels to normal is related to lactation status; ovulation occurs earlier among nonbreastfeeding women than those who lactate.
Prolactin and oxytocin	Fall to normal nonpregnant values in 7–14 days if not lactating	See the *Breastfeeding and the Mother–Newborn Dyad* chapter for a review of lactogenesis II. Pituitary disorders can cause an alteration in prolactin levels that interfere with lactogenesis.

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; ERV, expiratory reserve volume; FSH, follicle-stimulating hormone; GDM, gestational diabetes; GFR, glomerular filtration rate; GI, gastrointestinal; LDH, lactic dehydrogenase; LH, luteinizing hormone; RV, residual volume; WBC, white blood cell.

Cardiovascular and Hematologic Changes

A series of significant and rapid cardiovascular changes occur in the immediate postpartum period—namely, loss of blood, auto-transfusion of 10% to 15% of blood volume secondary to removal of the low-pressure fetal–placental unit,[47] and mobilization of extracellular fluid back into the maternal circulation. These dramatic fluid shifts make the postpartum period a period of maternal cardiovascular instability and a time of increased risk for adverse outcomes, including pulmonary edema, cardiac failure, and death, in women with preexisting cardiac disease, hypertension, or preeclampsia.

In the first 10 to 15 minutes after the newborn's birth, maternal stroke volume and cardiac output are increased approximately 80% above pregnancy levels.[52] This effect is hypothesized to be due to increased cardiac preload that results from auto-transfusion of utero-placental blood back into maternal systemic circulation and improved venous return with decompression of the vena cava secondary to removal of mechanical pressure from the gravid uterus. In the first postpartum hour, stroke volume and cardiac output remain elevated and heart rate decreases, but mean arterial blood pressure is unchanged.[52]

Mobilization of extracellular fluid back into circulation in the first few postpartum days also contributes to increased maternal blood volume. This acute increase in blood volume helps compensate for the normal blood loss of parturition. Following the sudden increase in cardiac output immediately after birth, the woman's blood volume declines to prepregnant levels over a period of approximately 2 weeks. Normal return of cardiovascular function depends in part on a physiologic diuresis in the first week postpartum. Without normal excretion of the extracellular fluid into the intravascular system, there is an increased risk of pulmonary edema, particularly among women with cardiac disease or preeclampsia.

The increase in stroke volume first during pregnancy and then during the postpartum period leads to a temporary increase in the size of the left atrium of the heart.[48] This physiologic ventricular hypertrophy resolves more slowly than the increase in stroke volume and cardiac output. Left ventricular size does not return to normal until approximately 6 months postpartum.[48]

The blood loss that occurs during birth causes a marked decrease in the woman's red blood cell (RBC) volume. In addition, a period of hemodilution arises in the first postpartum week due to the increase in plasma volume that occurs as interstitial fluid is mobilized. The loss of RBCs combined with this hemodilution leads to a decrease in hemoglobin and hematocrit in the first postpartum week. A woman's hemoglobin and hematocrit gradually return over a 4- to 6-week period to their prepregnant values as plasma volumes and RBC production return to normal.

Endocrine Changes

An abrupt drop in the blood levels of multiple hormones—including human placental lactogen (hPL), human chorionic gonadotropin (hCG), estrogen, and progesterone—occurs when the placenta is expelled. This event sets in motion a series of endocrine alterations, including changes in glucose and lipid metabolism and the initiation of lactogenesis II.

With decreasing levels of circulating estrogen, the thyroid-binding globulin level (which was elevated in pregnancy) also drops. This change, in turn, leads to less production of triiodothyronine (T_3) and thyroxine (T_4) and a gradual return to nonpregnant thyroid function. Nevertheless, thyroid dysfunction is more common in the postpartum period than in other times during a woman's life. The incidence of thyroid dysfunction is 7% to 10% in the postpartum period versus 3% to 4% in nonpregnant women.

Postpartum thyroid dysfunction, including postpartum thyroiditis, is often transient. Postpartum thyroiditis—a thyroid disorder that manifests during the first postpartum year—is thought to be an autoimmune disorder that is unmasked in the postpartum period due to the rebound in immunologic function that follows pregnancy.[49] In approximately 23% of women, thyroid dysfunction will become permanent.[50]

The marked changes in the maternal hormonal milieu in the postpartum period are postulated to be the cause of postpartum mood disorders, including postpartum depression. Studies that have evaluated the link between hormonal changes and the development of depression have yielded contradictory results and, in general, have not found a consistent link between hormone levels and postpartum depression.[51] Significant methodological difficulties arise when trying to identify a causal link between any given biologic factor and the development of mood disorders, particularly at a time when all physiologic and psychosocial systems experience significant changes. However, lack of evidence does not necessarily mean that no such link exists. Currently, hormone levels cannot be used to effectively predict the development of postpartum mood disorders or to determine the need for treatment.

Resumption of Pituitary Function and of Menstruation and Ovulation

Throughout pregnancy, the hypothalamic–pituitary–ovarian (HPO) axis is suppressed and, as a result, production of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) is also suppressed. Following birth, FSH and LH levels remain low for the first 2 postpartum weeks, but then rise gradually. Ovarian production of estrogen and progesterone, therefore, also remains low during the first few postpartum weeks.

Pituitary function returns to nonpregnant levels by 4 to 6 weeks postpartum. However, return of nonpregnant levels and patterns of gonadotropin-releasing hormone (GnRH), FSH, and LH secretion are related directly to lactation status. In breastfeeding women, the return of ovulation and menstruation varies and reflects both the duration and frequency of breastfeeding. Elevated levels of prolactin, along with other endocrine alterations related to frequent and regular infant suckling, disrupt the pulsatile release of GnRH, which in turn suppresses the LH surge. This cascade of events culminates in the suppression of ovulation. Even once ovulation is resumed, women who are breastfeeding are more likely to experience

nonfertile ovulation. This breastfeeding-related suppression of ovulation and menstruation is referred to as “lactational amenorrhea.”

In nonlactating women, the return of ovulation occurs sometime between postpartum days 45 and 94.[53] As many as 70% of these ovulations may be fertile, and in 20% to 70% of women, ovulation precedes the first menses.[53] Therefore, appropriate contraceptive counseling is essential for postpartum women since many—particularly those who are not breastfeeding—may ovulate and be fertile prior to their first postpartum visit. For women who desire to defer another pregnancy for a period of time, timely initiation of the woman’s chosen method of contraception is an important management intervention.

Weight Loss in the Postpartum Period

Weight loss following pregnancy is a common concern for women. Aside from the immediate weight loss that occurs directly related to the birth of the baby, expulsion of the placenta, and loss of fluid in the first postpartum week, the physiology of weight loss (which is beyond the scope of this chapter) does not appear to differ in the postpartum period from the physiology of weight loss at other times in a woman’s life. The rate and amount of weight loss in the postpartum period seem to be determined by the same factors that determine weight loss at any point in a woman’s life, including existing weight/body mass index (BMI), diet, age, and activity level.[54] Furthermore, studies indicate that pregnancy weight retention is not a significant determinant of excessive weight. A study of 7000 women who were followed from the beginning of one pregnancy to the beginning of the next pregnancy found that the average increase in weight between the first pregnancy and the next was 3.4 kg.[55]

In recognition that humans tend to gain weight as they get older, additional studies that controlled for age found an increase of only 0.3 to 0.5 kg between pregnancies.[55] These findings suggest that interventions to reduce rates of maternal obesity should focus not only on the loss of weight gained in pregnancy, but also on primary prevention of obesity and weight management at a preconceptional visit.

Conclusion

The postpartum period is a time of remarkable and rapid physiologic adjustment. Knowledge of postpartum physiology is necessary to determine the difference between normal and abnormal progression during this period. Moreover, although the vast majority of women in the postpartum period have a normal course of recovery, it is important to teach all women about signs of danger and worrisome symptoms in the postpartum period. Finally, despite a relatively rapid change from the physiology of pregnancy to a nonpregnant physiologic state, it is important to remind the woman that it is quite normal for her not to feel completely back to "herself" during the weeks and even months following the birth of a newborn. The postpartum period is a profound role transition and life-changing event that cannot be described via a review of anatomic and physiologic changes.

References

1. Ramsay DT, Kent JC, Hartmann RA, Hartmann PE. Anatomy of the lactating human breast redefined with ultrasound imaging. *J Anat*. 2005;206(6):525-534.
2. Geddes DT. Inside the lactating breast: the latest anatomy research. *J Midwifery Womens Health*. 2007;52(6):556-563.
3. Doucet S, Soussignan R, Sagot P, Schaal B. The secretion of areolar (Montgomery's) glands from lactating women elicits selective, unconditional responses in neonates. *PLoS One*. 2009;4(10):e7579.
4. Jonas W, Nissen E, Ransjo-Arvidson AB, Matthiesen AS, Uvnas-Moberg K. Influence of oxytocin or epidural analgesia o personality profile in breastfeeding women: a comparative study. *Arch Womens Ment Health*. 2008;11(5-6):335-345.
5. Bell AF, Erickson EN, Carter CS. Beyond labor: the role of natural and synthetic oxytocin in the transition to motherhood. *Midwifery Womens Health*. 2014;59(1):35-42.
6. Galbally M, Lewis AJ, Ijzendoorn M, Permezel M. The role of oxytocin in mother–infant relations: a systematic review of human studies. *Harv Rev Psychiatry*. 2011;19(1):1-14.
7. Lind JN, Perrine CG, Li R. Relationship between use of labor pain medications and delayed onset of lactation. *J Hum Lactat*. 2014;30(2):167-173.
8. Jevitt C, Hernandez L, Goer M. Lactation complicated by obesity. *J Midwifery Womens Health* 2007;52(6):606-613.
9. Hurst NM. Recognizing and treating delayed or failed lactogenesis II. *J Midwifery Womens Health*. 2007;52(6):588-594.
10. Leeners B, Rath W, Kuse S, Neumaier-Wagner P. Breast-feeding in women with hypertensive disorders in pregnancy. *J Perinat Med*. 2005;33:553.
11. Vanky E, Isaksen H, Moen MH, Carlsen SM. Breastfeeding in polycystic ovary syndrome. *Acta Obstet Gynecol Scand*. 2008;87:531.
12. Carlsen SM, Jacobsen G, Vanky E. Mid-pregnancy androgen levels are negatively associated with breastfeeding. *Acta Obstet Gynecol Scand*. 2010;89:87.
13. Parker LA, Sullivan S, Krueger C, Mueller M. Association of timing of initiation of breastmilk expression on milk volume and timing of lactogenesis stage II among mothers of very low-birth-weight infants. *Breastfeed Med*. 2015;10(2):84-91.
14. Kent JC. How breastfeeding works. *J Midwifery Womens Health*. 2007;52(6):564-570.
15. Stuebe AM, Grewen K, Meltzer-Brody S. Association between maternal mood and oxytocin response to breastfeeding. *J Womens Health (Larchmt)*. 2013;22(4):352-361.
16. Kent JC, Mitoulas LR, Cregan MD, Ramsay DT, Doherty DA, Hartmann PE. Volume and frequency of breastfeedings and fat content of breast milk throughout the day. *Pediatrics*. 2006;117(3):e387-e395.
17. Wilde CJ, Addey CV, Bryson JM, Finch LM, Knight CH, Peaker M. Autocrine regulation of milk secretion. *Biochem Soc Symp*. 1998;63:81-90.
18. Neville MC, Allen JC, Archer PC, et al. Studies in human lactation: milk volume and nutrient composition during weaning and lactogenesis. *Am J Clin Nutr*. 1991;54(1):81-92.
19. Victora CG, Bahl R, Barros AJ, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet*. 2016;387(10017):475-490.
20. American Academy of Pediatrics. Breastfeeding and the use of human milk. *Pediatrics*. 2012;129(3):e827-e841.
21. Giugliani ERJ, Horta BL, Loret de Mola C, Lisboa BO, Victora CG. Effect of breastfeeding promotion interventions on child growth: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:20-29.
22. Horta BL, Loret de Mola C, Victora CG. Breastfeeding and intelligence: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:14-19.
23. Lodge CJ, Tan DJ, Lau MXZ, et al. Breastfeeding and asthma and allergies: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:38-53.
24. Bowatte G, Tham R, Allen KJ, et al. Breastfeeding and childhood acute otitis media: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:85-95.
25. Hassiotou F, Geddes DT. Immune cell–mediated protection of the mammary gland and the infant during breastfeeding. *Ad Nutr*. 2015;6(3):267-275.
26. Ip S, Chung M, Raman G, et al. *Breastfeeding and Maternal and Infant Health Outcomes in Developed Countries*. Rockville, MD: Agency for Healthcare Research and Quality; 2007.
27. Victora CG, Horta BL, Loret de Mola C, et al. Association between breastfeeding and intelligence, educational attainment and income at 30 years of age: a prospective birth cohort study from Brazil. *Lancet Global Health*. 2016;3(4):e199-e205.
28. McNally S, Stein T. Overview of mammary gland development: a comparison of mouse and human. *Methods Molec Biol* 2017;1501:1-17.
29. Langer JE, Oliver ER, Lev-Toaff AS, Coleman BG. Imaging of the female pelvis through the life cycle. *Radiographics*. 2012;32(6):1575-1597.
30. Diniz CP, Araujo Júnior E, Lima MM, Guazelli CA, Moron AF. Ultrasound and Doppler assessment of uterus during puerperium after normal delivery. *J Matern Fetal Neonatal Med*. 2014;27(18):1905-1911.

31. Weydert JA, Benda JA. Subinvolution of the placental site as an anatomic cause of postpartum uterine bleeding: a review. *Arch Pathol Lab Med*. 2006;130:1538-1542.
32. O'Riordan MN, Higgins JR. Haemostasis in normal and abnormal pregnancy. *Best Pract Res Clin Obstet Gynaecol*. 2003;17(3):385-396.
33. Bremme KA. Haemostatic changes in pregnancy. *Best Pract Res Clin Haematol*. 2003;16(2):153-168.
34. Brenner B. Haemostatic changes in pregnancy. *Thromb Res*. 2004;114(5-6):409-414.
35. Dahlman T, Hellgren M, Blomback M. Changes in blood coagulation and fibrinolysis in the normal puerperium. *Gynecol Obstet Invest*. 1985;20:37-44.
36. Paliulyte V, Drasutiene GS, Ramasauskaite D, Bartkeviciene D, Zakareviciene J, Kurmanavicius J. Physiological uterine involution in primiparous and multiparous women: ultrasound study. *Obstet Gynecol Int*. 2017;2017:6739345. doi:10.1155/2017/6739345.
37. Chi C, Bapir M, Lee CA, Kadir RA. Puerperal loss (lochia) in women with or without inherited bleeding disorders. *Am J Obstet Gynecol*. 2010;203(1):56.e1-56.e5.
38. Fletcher S, Grotegut CA, James AH. Lochia patterns among normal women: a systematic review. *J Womens Health (Larchmt)*. 2012;21(12):1290-1294.
39. Sherman D, Lurie S, Frenkel E, Kurzweil Y, Bukovsky I, Arieli S. Characteristics of normal lochia. *Am J Perinatol*. 1999;16(8):399-402.
40. Timmons BC, Mahendroo M. Processes regulating cervical ripening differ from cervical dilation and postpartum repair: insights from gene expression studies. *Reprod Sci*. 2007;14(8 suppl):53-62.
41. Ueda Y, Enomoto T, Miyatake T, et al. Postpartum outcome of cervical intraepithelial neoplasia in pregnant women determined by route of delivery. *Reprod Sci*. 2009;16(11):1034-1039.
42. Guo S, DiPietro LA. Factors affecting wound healing. *J Dent Res*. 2010;89(3):219-229.
43. Sminakis KV, Chasan-Tabe L, Wolf M, Markenson G, Ecker JL, Thadhani R. Postpartum diabetes screening in women with a history of gestational diabetes. *Obstet Gynecol*. 2005;106:1297-1303.
44. Gaberšček S, Zaletel K. Thyroid physiology and autoimmunity in pregnancy and after delivery. *Expert Rev Clin Immunol*. 2011;7(5):697-706.
45. Chiarello CM, Falzone LA, McCaslin KE, Patel MN, Ulery KR. The effects of an exercise program on diastasis recti abdominis in pregnant women. *J Womens Health Phys Ther*. 2005;29(1):11-16.
46. Hellgren M. Hemostasis during normal pregnancy and puerperium. *Semin Thromb Hemost*. 2003;29(2):125-130.
47. Duvekot JJ, Peeters LL. Maternal cardiovascular hemodynamic adaptation to pregnancy. *Obstet Gynecol Surv*. 1994;49(12):S1-S14.
48. Mone SM, Sanders SP, Colan SD. Control mechanisms for physiological hypertrophy of pregnancy. *Circulation*. 1996;94:667-672.
49. Stagnaro-Green A. Postpartum thyroiditis. *J Clin Endocr Metab*. 2002;87(9):4042-4047.
50. Alexander EK, Pearce EN, Brent GA, et al. 2017 guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2017;27(3):315-389.
51. Le Donne M, Mento C, Settineri S, Antonelli A, Benvenga S. Postpartum mood disorders and thyroid autoimmunity. *Front Endocr*. 2017;8:9.
52. Ouzounian JG, Elkayam U. Physiologic changes during normal pregnancy and delivery. *Cardiol Clin*. 2012;30(3):317-329.
53. Glasier JE. A return of ovulation and menses in postpartum nonlactating women: a systematic review. *Obstet Gynecol*. 2011;117:657-662.
54. Schauberger CW, Rooney BL, Brimer LM. Factors that influence weight loss in the puerperium. *Obstet Gynecol*. 1992;79(3):424-429.
55. Gore SA, Brown DM, Smith West D. The role of postpartum weight retention in obesity among women: a review of the evidence. *Ann Behav Med*. 2003;26(2):149-159.

CHAPTER

33

Postpartum Care

Ira Kantrowitz-Gordon

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Introduction

The end of pregnancy initiates physiologic changes, as many organ systems return to their nonpregnant states. The uterus involutes, cardiac output and blood volume decrease, and estrogen and progesterone levels fall. Lactogenesis II begins and may continue for years in women who chose to breastfeed. Yet the postpartum period is so much more than just these physical changes—birth transforms the social and emotional context for the woman and her family.

Definition of the Postpartum Period

Pregnancy comes to a definitive end with the delivery of the placenta and membranes. At this time, the woman begins the physiologic transition to the nonpregnant state. Traditionally the postpartum period, or puerperium, has been defined as lasting 6 weeks: By 6 weeks after giving birth, most women have completed the last of the physiologic transitions, which include uterine involution, cessation of lochia, and establishing lactation. This is a somewhat arbitrary—and an arguably short—time frame for the transition to parenting. An alternative definition of the postpartum period is the "fourth trimester," which suggests a transition that lasts approximately 3 months.

While "recovery" implies healing from illness or trauma, the postpartum transition, as a normative life experience, is more than that. With the arrival of the newborn, family members assume new roles both within the family and in the community. For many families, employment status may change for either parent. As definitions of "family" have expanded in recent decades, recognition that there are considerable variations in a family's adjustment after childbirth has become more widespread. Despite the significance of these changes, women and families may receive less attention and support during the postpartum period than they did during pregnancy and the intrapartum period. As a consequence, the postpartum period is a time of both opportunity and challenges to health.

Postpartum Care in the United States

Standard postpartum care in the United States today includes the traditional model of an office visit with the prenatal care provider at 4 to 6 weeks postpartum, although women who were followed prenatally in a group setting such as the CenteringPregnancy usually have a group reunion at 1 to 2 months. Thus, after hospital discharge, many women do not have contact with their healthcare provider until the 6-week postpartum office visit, and a significant number of women do not attend this visit. It is unclear why this specific time frame was selected, and in some centers, it has been replaced by an earlier examination at 2 to 3 weeks. An additional office visit at 1 to 2 weeks postpartum has been proposed to bridge the gap in care from discharge until 6 weeks, and to support breastfeeding, contraception, and the psychosocial transition postpartum. The financial pressures of clinical practice deter many providers from providing this extra visit, however, as there often is no additional reimbursement for it.

In developed countries, there is a wide variation in the amount of financial support provided to women after childbirth. In the United States, unpaid parental leave is guaranteed for 12

weeks under the Family and Medical Leave Act, but only approximately 56% of women are eligible for this job protection.[1] For many families, taking time off to care for the woman or infant in the postpartum period places the family at significant financial risk. This economic challenge in the United States stands in contrast to policies in most other developed countries, where paid maternal leave extends from 14 weeks (e.g., Singapore and Switzerland) to longer than a year (e.g., Japan and Finland). In some countries, there is a similar range of paid paternal leave.[2]

Cultural Perspectives of the Postpartum Period

The first essential perspective for provision of effective postpartum care is understanding the woman in her social and cultural context so that the care provided is culturally sensitive. Throughout history, in all parts of the world, and across cultures, the postpartum period has been acknowledged as a special or vulnerable time during which women follow distinct rituals. For example, Mexican–American immigrant families may practice the *cuarentena*, or 40 days of postpartum recovery.[3] During this time, the woman's body is considered open and vulnerable to illness. Practices to protect the woman include sexual abstinence, wrapping the abdomen in tight garments (*fajas*), avoiding strong foods and cold substances, and remaining at home as much as possible.

In many parts of Asia, the new mother remains at home and rests for 30 to 40 days after giving birth—a practice often referred to as "doing the month."[4] This period of prolonged rest protects the woman from exposure to illness and conserves warmth that she needs for caring for her newborn. To maintain warmth, the woman may eat special foods, avoid showering, and stay in bed. Often a female family member will stay with her for the first few months to do the cleaning and cooking so that the new mother can rest. Similarly, Chinese women may follow the practice of *zuo yuezi*, or "sitting the month."[5,6] During the first month postpartum, they are cared for by their mothers, mothers-in-law, or grandmothers; avoid cold foods; and keep themselves warm by wearing long clothing and staying indoors. In addition to its protective qualities, sitting the month is seen as a way to show respect to elders' beliefs and to traditions that have been observed for millennia.[6] Vietnamese women also view the postpartum state as a cold state (*duong*) and, therefore, protect themselves through warmth (*am*). Cultural practices include warm water for bathing and stimulating lactation, staying indoors, and consuming foods that have warm qualities, such as meat, eggs, ginger, and wine.[7] The postpartum cultural practices in southeast Turkey are surprisingly similar to those of both the Asian and Latin cultures. These practices include avoiding cold foods, and remaining at home on reduced activity for 40 days.[8] A common characteristic of all these practices is the provision of additional social support to the woman after childbirth.

When a woman is geographically distant from her extended family, it may be difficult to maintain cultural practices.[9] Knowledge about other cultures and practices is a necessary beginning for midwives, but it is also important to incorporate this knowledge into clinical practice with curiosity and humility, so that women and families are treated as *they* wish to be treated. To accomplish this balancing act requires an ongoing process of clinical inquiry and communication with women to provide care that is culturally congruent—that is, care that fits with the family's values and meanings.[10,11] Questions that can help the midwife conduct a cultural assessment are listed in **Table 33-1**. Resources for providing culturally congruent midwifery care can be found at the end of this chapter.

Table 33-1	**Postpartum Cultural Assessment**
Consider the following questions when caring for women and families postpartum: Is the postpartum period viewed as a normal physiologic process, a wellness experience, a time of vulnerability	

and risk, or a state of illness? What does the postpartum experience mean to the woman?

Is the postpartum period a time for privacy or socialization?

What kinds of support are the new family given during the postpartum period, and who gives that support?

Are there culturally expected practices during the postpartum period regarding diet, nutrition, treatments (including pharmacologic therapies), and activity?

What maternal precautions or restrictions are necessary during the postpartum period?

How is postpartum pain managed?

What are the patterns regarding care of the infant and relationships within the nuclear and extended families? Are there expected patterns for feeding and caring for the infant?

Beyond Gender

Parenting is a gendered, socially constructed experience. Lesbian, gay, bisexual, queer (LGBQ) and transgender and gender nonconforming (TGNC) parents may have different psychosocial experiences of childbirth, and little is known about their postpartum experiences. These families may experience difficulty interacting with the healthcare system due to heterocentric practices in language use and history forms, discriminatory practices, and lack of support from the extended family.[12] Assumptions about gender-based parenting roles may not be valid. For example, either female parent may choose to breastfeed in lesbian couples. Transgender men may experience the physical aspects of childbirth differently than do other parents.[13] Due to the additional stresses facing these families, a supportive environment, healthcare team, and social network are important for birth and postpartum care.[14] The midwife should use an approach of cultural humility and inquiry in caring for trans parents, and use language that supports their preferred pronoun and gender identity. General healthcare issues germane for LGBT and TGNC individuals is briefly reviewed in the *Midwifery: Clients, Context, and Care* chapter.

The First Hour Postpartum: The Fourth Stage of Labor

The term *fourth stage of labor* refers to the first postpartum hour following placental expulsion. Immediately following the expulsion of the placenta, a number of maternal changes occur as the physical and emotional stresses of labor and birth resolve and postpartum recovery and bonding begin. Close observation and frequent assessment as described in **Appendix 33A** are needed during the fourth stage of labor, as this is a time of both physical vulnerability to emergent complications and heightened sensitivity as the woman and family are introduced to the newborn. This section presents a brief overview of maternal–infant behaviors during the initial first postpartum hours and evidence-based practices that support attachment and the formation of the family.

Maternal–Infant Bonding and Attachment

The theory that there is a biologically determined critical period immediately after birth during which maternal–newborn bonding takes place was first proposed by Klaus and Kennell in the 1970s.[15] These researchers found that skin-to-skin contact between the woman and her newborn in the first hour after birth resulted in more attachment behavior by a woman toward her infant up to a month after birth. This seminal work by Klaus and Kennell set the stage for the plethora of subsequent research that has profoundly influenced the care of women, newborns, and families in the postpartum period. Emotional attachment between parents and their newborn has lifelong implications.

During the fourth stage of labor, the woman experiences profound physical and psychological changes as she recovers from the act of giving birth in the setting of an intense hormonal milieu and first meets the newborn. Partners, grandparents, siblings, and other family members or support persons present at a birth also experience heightened awareness and possible psychological vulnerability. Factors such as a traumatic birth, previous life traumas, or newborn illness can all influence an individual's response to childbirth, attachment to the newborn, and development of identity and role as a mother or primary caretaker.

Definitions of parent–infant attachment have historically included concepts such as bonding, love, attachment, or an instinctual connection. The initial development of attachment between parents and a newborn takes many forms; the response of one parent may differ from the other. More importantly, the process is bidirectional and includes a great deal of interactive and reciprocal behaviors. The infant's primary behavior is seeking physical proximity. The mother's (or other primary caregiver's) behavior demonstrates responsiveness to the newborn's cues. For example, animal and human studies of bonding in the first postpartum hour have found that healthy newborn infants undergo a predictable series of behaviors immediately after birth, including crawling toward the mother's breast, rooting and suckling, and looking at the mother's face.[16] It is theorized that this behavior is stimulated by maternal odor and voice recognition.[17]

Goulet et al. used concept analysis methodology to examine the vast literature on attachment to develop a clearer understanding and definition of this process.[18] In their analysis, these authors identified three key attributes in the mutual relationship between parents and newborns.

- *Proximity*. Physical closeness allows interaction with multiple senses (e.g., hearing, touch, sight, and smell), facilitates a strong emotional connection, and creates an awareness of the infant's individuality and needs that are different from the parent's needs.
- *Reciprocity*. Parents and newborns interact in a responsive and adaptive manner. Parents become sensitive to the infant's cues and body language in their caregiving. The infant contributes to the interaction by reinforcing the parents' behaviors.
- *Commitment*. Feeling and acting responsibly to ensure the safety, growth, and development of the infant place the infant in a central position in the family and identify the parenting role as an important part of the adult's identity.[18]

An important caveat to bonding theory is that much of this work is based on animal studies, wherein immediate imprinting of the infant to the caregiver is biologically species specific. Superimposing the results of animal studies on human behavior may result in beliefs that have no scientific basis. In addition, the underlying premise that this sensitive period is necessary for the maternal–newborn relationship has been criticized. Attachment bonds form over time and involve a complex interplay of neural circuitry, hormones such as oxytocin, and interactive behavior.[19] Overemphasis on the first hour may be harmful in situations where it is missed, such as when the medical condition of the woman or the newborn requires physical separation of this dyad. Fortunately, there are countless opportunities to develop attachment, as demonstrated by parents who adopt children at a variety of ages. Continuing reciprocal responses, strengthening of the relationship, and changing roles are processes that occur throughout life, including during the postpartum period.

Caregiver interventions during the fourth stage of labor, including those related to medical, psychosocial, and environmental conditions, can either facilitate or interfere with these important processes. Despite incomplete knowledge about the exact neurobiology involved in maternal adaptation, a great deal of research has been conducted that has identified short-term and long-term effects of specific care practices such as skin-to-skin contact and early breastfeeding with newborn self-latching.

Early Continuous Skin-to-Skin Contact

Early skin-to-skin contact involves placing a naked newborn prone on the woman's abdomen or chest immediately or soon after birth. The infant is dried and the area of the infant's body that is not touching the mother's skin is covered with a blanket to assist in maintaining warmth. A cap may be applied to the dried head of the newborn to assist with temperature maintenance. The vital signs of the newborn and woman are monitored with the infant on the maternal chest. Other procedures are delayed for at least 1 hour or until after the first successful breastfeeding.

Continuous maternal–newborn contact and skin-to-skin touching have been found to improve maternal affectionate behavior, regulate the newborn's behavior, and increase the duration of breastfeeding.[20] Early contact also increases maternal sensitivity and the infant's self-regulation and irritability at 1 year after birth.[21] Moore et al. conducted a meta-analysis of 46 randomized controlled trials (RCTs) of early skin-to-skin contact (n = 3850 mother–infant

dyads) and found that skin-to-skin contact improved breastfeeding and breastfeeding duration at 1 to 4 months (relative risk [RR], 1.24; 95% confidence interval [CI], 1.07–1.43), although differences in study methodology and risks of bias casts some uncertainty on the strength of these findings.[21] Positive trends in improved summary scores for maternal affectionate love/touch, maternal attachment behavior, and shorter duration of infant crying were also noted in the group that experienced early skin-to-skin contact.[21]

In the first 2 hours after cesarean birth (**Figure 33-1**), neonates who were held in skin-to-skin contact with the father demonstrated less crying, became calmer, and were more drowsy than newborns left in a crib.[22]



Figure 33-1 Skin-to-skin contact and breastfeeding immediately after cesarean birth.

Courtesy of Tekoa L. King, CNM, MPH.

Oxytocin and cortisol are likely important mediators of early parent–infant interactions. Premature infants have better cardiorespiratory stability when exposed to skin-to-skin contact with their mothers.[21] Increased oxytocin levels have been demonstrated after skin-to-skin care in preterm infants[23] and after cesarean birth,[24] which suggests a physiologic mechanism for increased attachment and breastfeeding success in these contexts. Oxytocin levels in mothers and fathers have been shown to correlate with affectionate behaviors and touch at 6 months

postpartum.[25] Newborns who experience skin-to-skin care may attain heart rate stability and have lower salivary cortisol levels when compared to infants who do not share skin-to-skin contact with their mothers immediately after birth.[26] Given the strength of these findings, hospitals in the United States are increasingly instituting skin-to-skin contact immediately after birth for all women including in the operating room and recovery room following a cesarean birth.[27]

Newborn Self-Latching

When healthy term infants are placed in skin-to-skin contact during the first hours following birth, they frequently crawl and find the areola by themselves, and most will initiate suckling spontaneously.[16] Supporting newborn interaction with the parents may enhance the newborn's ability to self-regulate his or her behavior over the long term (i.e., improved sleep, social-interactive behavior, and language development).[16] The wide-ranging positive effects of unrestricted maternal–newborn contact reinforce the importance of protecting the family in the first hours after birth from unnecessary intervention.

Inclusion of Significant Others

Involvement of the woman's partner, newborn's father, or other support persons during the fourth stage of labor has been found to help the woman during the postpartum period,[28] increase the partner's emotional involvement with the neonate,[29] and increase the sense of security experienced by the partner.[30] Men and lesbian partners have expressed feeling helpless, useless, anxious, out of place, and vulnerable during birth; including family members in the experience is an important component of family-centered midwifery care.[31,32]

For all these reasons, it is important to assess and address the partner's needs during the fourth stage of labor to facilitate the transition to parenthood. Strategies include anticipatory guidance and explanations to the woman and her significant others. Support via midwifery presence and information has been found to enhance the birth experience of the partner.[33] Engaging partners in activities such as cutting the cord and skin-to-skin contact has been found to enhance their emotional involvement with the neonate.[29] Additional activities may include encouraging continued support for the woman during required genital tract repair, engaging in observation of the neonatal transition, and assisting with initial breastfeeding.

Early Breastfeeding

Evidence that breastfeeding improves health outcomes for women and their infants is overwhelming; this evidence is reviewed in detail in the *Breastfeeding and the Mother–Newborn Dyad* chapter. Initiation of breastfeeding in the first few hours after birth is recommended, as the infant is alert and awake during this time period. Activities during the fourth stage of labor that have been found to promote early breastfeeding include skin-to-skin contact between the newborn and mother following birth and maintaining this contact for the first few hours of life.[34] If the woman and her neonate must be separated for medical

indications, skin-to-skin contact in the intensive care nursery, early effective breast milk expression, and support from peers and staff can be implemented and are very important to the promotion of breastfeeding in neonatal units.[35]

Initial Postpartum Period

The first 72 hours after birth is labeled as the early puerperium period. The frequency with which the midwife evaluates the woman and newborn during this period will vary depending on the birth setting and individual circumstances. In most settings, the midwife assesses the woman at least once daily. Each visit includes a chart review; interval history, including physical, psychological, and emotional changes since the birth; physical examination; and observation of family interactions. Care of women during this period includes treatment of pain and common discomforts, immunizations as needed, and health education.

Health Record Review

The woman's prenatal record and record of labor and birth are reviewed prior to all postpartum examinations. A review of the infant's status is part of this pre-assessment because the newborn's health status may directly influence the management plan for the woman. A review of the health record includes assessment of the woman's vital signs, laboratory results, medication use, and comments from other caregivers. Previous orders and progress notes are reviewed as well. Medications that were temporarily stopped during the pregnancy or labor should be evaluated and a decision about restarting them made.

Time elapsed since birth, in hours or days, is ascertained to identify expected physical findings. When entering notes in the health record, it is important to indicate precise time information. For example, postpartum day 1 may denote that the woman is within 24 hours postpartum or between 24 and 48 hours postpartum. Since indicating postpartum days may generate confusion based on time of birth and calendar days, specifying how many *hours* have elapsed since the birth is preferred.

Interval History

The initial assessment of maternal physical well-being starts with eliciting information about nutrition, bladder and bowel function, activity level, and a description of pain or discomfort if present. Information about the infant feeding method, infant behavior, and sleep–wake cycles should be collected even if the midwife is not the primary provider for the newborn.

When a home visit is conducted, the midwife should arrange the visit to coincide with those times when the infant is likely to be awake if possible. This enables an evaluation of the parent–newborn interaction and the parents' skill and comfort in handling the newborn, which may indicate areas of learning needs. Examples of criteria for assessment of parent–newborn interactions are listed in **Table 33-2**.[36] Cultural factors need to be considered when evaluating the parent–newborn interactions.[37]

Table 33-2	Clinical Assessment of Parent's Interaction with Newborn
Uses gentle touch with newborn	
Positions infant for face-to-face interaction	
Directs gaze toward newborn when interacting	

Makes visual contact
Displays range of facial expressions
Verbalizes to newborn
Makes positive, affirming, loving comments to the newborn; avoids making disparaging comments
Modulates interaction in response to newborn's cues
Elicits verbalizations and behaviors from the newborn and responds contingently
Responds to newborn's distress

Modified with permission from Fowles ER, Horowitz JA. Clinical assessment of mothering during infancy. *J Obstet Gynecol Neonatal Nurs.* 2006;35(5):662-670.[36]

Physical Examination

The physical examination focuses on the physiologic changes that occur after birth, and includes assessment of the heart, lungs, breasts, abdomen, perineum and anus, and lower extremities. The postpartum examination procedure with expected findings is detailed in **Appendix 33B**.

Care of Women During the Early Postpartum Period

Specific components of care for women during the early puerperium can vary secondary to individual needs. In many hospital settings, routine or standing orders for the first postpartum days are used as a standard template for all women, but it is imperative that clinicians consider whether these orders are appropriate for the specific woman. The orders may need to be individualized—for example, by modifying pain management routines, discontinuing intravenous fluids, or requesting a referral to a lactation consultation. The midwife should be certain the topics outlined in **Table 33-3** are addressed if written orders are prepared, or that they are considered in the care of women delivering in out-of-hospital settings.

Table 33-3 Postpartum Orders and Plans

Topic	Details/Options
Hydration	Oral hydration preferred; should specify when to discontinue any intravenous hydration
Nutrition	Consider food allergies, cultural preferences, and diabetes
Activity level	Generally unrestricted; first time out of bed with assistance after epidural anesthesia, cesarean birth, or postpartum hemorrhage
Routine postpartum medications	RhoGAM if needed, vitamins and iron, stool softeners or laxatives, analgesics, immunizations, and sleep medications as needed
Preexisting medications	Continue or reinstate medications used prenatally, such as thyroid replacement and antidepressants, per the plan that was made prenatally. Consult with a physician colleague as indicated.
Perineal care	Ice packs, topical medications, peri-bottle
Management for inability to void	Peppermint oil essence in commode or toilet room, catheterization if necessary

Breast care	Purified lanolin cream or hydrogel for nipple soreness, breast pump for engorgement, binder/supportive bra for women who are not breastfeeding
Laboratory tests	Hemoglobin and hematocrit if symptoms of anemia or hypovolemia; Rh screen of the newborn if the woman is Rh negative
Immunizations	Rubella and varicella for nonimmune women; influenza if needed; HPV series can be resumed if discontinued during pregnancy; Tdap if not received during pregnancy
Contraception	Prescription for self-administered contraceptives; DMPA before discharge in selected clients
Discharge if in a hospital setting	Depends on family readiness and status of newborn
Follow-up	Postpartum clinics, home visits, 2 or 6 weeks in office

Abbreviations: DMPA, depot medroxyprogesterone acetate; HPV, human papillomavirus; Rh, Rhesus factor; RhoGAM, Rh immune globulin; Tdap, tetanus–diphtheria–acellular pertussis.

Rhesus Alloimmunization

Most women experience a small transfer of fetal blood into the maternal circulation (fetomaternal hemorrhage) during childbirth, primarily at the time of placental separation. If the fetal blood type is Rh D positive and the maternal blood type is Rh D negative, the fetal red blood cells that enter the maternal circulation are identified in maternal circulation as a foreign antigen. The fetal cells stimulate an immune response (alloimmunization) that produces immunoglobulin M (IgM) and immunoglobulin G (IgG) antibodies, which are directed against Rh D–positive red blood cells. In a subsequent pregnancy with a Rh D positive fetus, these antibodies can cross the placenta and bind to fetal red blood cells, causing hemolytic disease of the fetus and newborn. Approximately 17% of RH D–negative women who are not immunized will become alloimmunized.[38]

Anti-D immune globulin (RhoGAM) is a hyperimmune plasma derivative that binds fetal Rh D–positive cells and prevents the maternal production of anti-D antibodies.[39] Administration of anti-D immune globulin within 72 hours following birth reduces the risk of alloimmunization quite significantly (RR, 0.12; 95% CI, 0.07–0.23) and is recommended for all Rh D negative women who have a newborn with Rh D–positive blood who are not already sensitized.[40]

The newborn's blood type usually is tested on a small sample from the umbilical cord. If blood typing of the newborn's cord blood is not available, the benefits of Rh immune globulin administration to Rh D negative women outweigh the theoretical risks of unnecessary administration. Administration after 72 hours may be protective, and anti-D immune globulin should be given even up to 14 days after the birth, as no studies have been conducted that address the latest period for which the agent is effective. Because anti-D immune globulin is a blood product, it has theoretical risks of infection; however, current technologies screen the product for all known viruses, such as human immunodeficiency virus (HIV). Midwives should discuss its benefits and risks carefully with women, especially with women who object to the use of blood products.

The standard 300-mcg dose of anti-D immune globulin is sufficient to treat a fetal–maternal hemorrhage containing 15 mL of red blood cells or 30 mL of whole blood. While rare, it is important to know when the fetal–maternal hemorrhage exceeds this amount, because

additional anti-D immune globulin will be needed if the amount of hemorrhage is larger than 30 mL of whole blood. Therefore, a Rosette test is performed on the mother's blood to ascertain whether Rh-positive blood is detectable in the maternal blood. If the Rosette test is positive, a Kleihauer–Betke test is performed to determine the amount of fetal hemoglobin present. In some facilities, a Kleihauer–Betke test is the only test ordered because it entails the quantitative measurement of fetal red blood cells in maternal blood; the test results are used to determine if and how much additional Rh immune globulin should be administered.

Immunizations

The early postpartum period is an opportunity to provide women with protection against several infections. Women who received part of the human papillomavirus (HPV) vaccine series prior to becoming pregnant can complete that series postpartum. All women should be offered seasonal influenza vaccine regardless of their pregnancy status and should be offered this vaccine postpartum if the woman did not receive it during her pregnancy. Booster vaccination with tetanus–diphtheria–acellular pertussis (Tdap) should be administered to all pregnant women in the third trimester. This applies to all women, even if they received the vaccine prior to pregnancy or during a previous pregnancy.[41] The Tdap vaccine may be given in the postpartum period if it was not administered to the woman during pregnancy. Vaccination of all family members with a one-time dose of Tdap will also protect the infant from acquiring pertussis.[41]

Live-virus vaccines against rubella and varicella that are contraindicated during pregnancy should be offered to nonimmune women in the immediate postpartum period. The woman is not likely to be pregnant at this time, and no increased risk of congenital anomalies has been demonstrated if conception occurs within 1 to 3 months following vaccine administration. Nevertheless, it is recommended to delay conception at least 1 month after live-virus vaccine administration.

Common Discomforts in the Early Postpartum Period

Pain and fatigue are the most common discomforts experienced by most women the first several days after giving birth. Pain can be secondary to perineal lacerations or abdominal incisions, breast pain from engorgement, nipple pain related to infant suckling, uterine cramping, and hemorrhoids. Unmitigated pain can interfere with a woman's ability to care for her infant, delay recovery, and increase the risk for postpartum depression and for persistent pain.[42]

Treatment of Pain in the Early Postpartum Period

Both non-pharmacologic and pharmacologic treatments for pain can be used to manage breast or perineal pain. Individual medications usually are chosen based the severity of pain, mechanism of action, any known drug allergies, and compatibility with breastfeeding. Given the wide inter-individual variability in postpartum pain, an individualized stepwise approach

using a multimodal strategy is recommended.[43] Medications that are commonly prescribed to treat pain during the early puerperium are listed in **Table 33-4**.[43,44]

Table 33-4 Medications Commonly Prescribed to Treat Pain in the Early Postpartum Period

Drug: Generic (Brand)	Formulations	Indications and Dose	Clinical Considerations	Lactation
Acetaminophen (Tylenol)	325, 500 mg	*Mild to moderate pain* 500, 650, 1000 mg every 6 hours	Maximum: 4 g/day acetaminophen from all sources	Safe
Ibuprofen (Motrin, Advil)	200, 400, 600 mg	*Mild to moderate pain* 400 to 600 mg every 6 hours	Take with food Maximum: 3200 mg/day Avoid with hypertension All NSAIDs have a ceiling effect such that larger doses do not increase pain relief	Safe
Ketorolac (Toradol)	IM: 15, 30, 60 mg PO: 10 mg Should not be given PO as initial dose	*Short-term treatment for postoperative pain* 60 mg IM × 1 dose, then first PO can be 20 mg, followed by 10 mg PO every 4–6 hours	Reduces postoperative opiate use Maximum: 40 mg/day Do not use for women with renal impairment Do not shorten dosing interval to less than 4 hours	Safe
Acetaminophen (Tylenol)/ codeine No. 3	300/30 mg	*Moderate pain* Tylenol No. 3 every 4–6 hours	Take with food May cause excessive sedation in women and newborns who are rapid metabolizers	FDA advisory to avoid codeine in breastfeeding women if possible. If needed, use smallest effective dose for less than 4 days. Observe newborn for sedation, excessive sleepiness, and poor feeding.
Hydrocodone/ acetaminophen (Norco)	Hydrocodone/ acetaminophen combination as 5/325 mg, 7.5/325 mg, or 10/325 mg	*Moderate pain* 1–2 tablets every 4–6 hours	Maximum: 4 g/day acetaminophen from all sources	
Hydrocodone/ acetaminophen (Vicodin)	Hydrocodone/ acetaminophen combination as 5/300 mg	*Moderate pain* 1–2 tablets every 4–6 hours	Maximum: 4 g/day acetaminophen from all sources	
Vicodin ES	7.5/300 mg			

Abbreviations: IM, intramuscular; NSAIDs, nonsteroidal anti-inflammatory drugs; PO, oral.

Based on American College of Obstetricians and Gynecologists. Practice advisory on codeine and tramadol in breastfeeding women. 2017. Available at: https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-on-Codeine-and-Tramadol-for-Breastfeeding-Women. Accessed December 28, 2017[43]; Fahey JO. Best practices in management of postpartum pain. *J Perinat Neonat Nurs*. 2017;31(2):126-136.[44]

Women who have experienced an uncomplicated vaginal birth may not require medication

stronger than ibuprofen (Advil) 600 mg taken orally every 6 hours as needed for either uterine cramping or perineal pain.[44] Nonsteroidal anti-inflammatory drugs (NSAIDs) appear to be more effective than acetaminophen or aspirin, probably because NSAIDs have both analgesic and antiinflammatory effects.[45] NSAIDs have a ceiling effect which means that increasing the dose beyond a certain point will increase the incidence of side effects but does not increase the analgesic effect. The ceiling dose for ibuprofen appears to be at approximately 400 mg per dose. NSAIDs transfer into breast milk in very small amounts are considered safe for lactating women.

Women who need stronger pain relief are usually treated with a hydrocodone–acetaminophen combination analgesic, but some cautions are warranted. The active metabolite of codeine and hydrocodone is morphine, which is lipophilic and crosses into breast milk. Individuals who have a genetic polymorphism that involves duplication of the enzyme CPY2D6 are ultra-rapid metabolizers of drugs such as codeine that are metabolized by CYP2D6. In 2006, the adverse effects of codeine use by persons who are ultra-metabolizers first became evident in a case report about a woman who took codeine for postpartum pain. Her ultra-rapid metabolism of the drug resulted in her infant receiving a large amount of morphine via breast milk; the infant died of morphine overdose.[46] Subsequently, the U.S. Food and Drug Administration (FDA) issued a warning about the use of codeine by breastfeeding women that noted the possibility of morphine toxicity in breastfeeding infants. In 2017, the FDA expanded the Drug Safety Communication to recommend that breastfeeding women and young children should not use codeine or tramadol.[47]

The frequency of CYP2D6 ultra-rapid metabolizers varies by ethnicity. Overall, in the United States, the prevalence is 4% to 5%.[48] Only 0.5% of persons with a Chinese ancestry have duplicates of this enzyme, but the prevalence is as high as 29% in persons from Ethiopia.[48] Although genomic tests that can detect this status are available commercially, they are costly and not recommended for general screening.

Frequent breastfeeding the first days following birth is a critical step in establishing lactation, but some women may need analgesic medications that are in the opioid-strength range during this period. Recognizing that the health benefits of breastfeeding are significant and the incidence of morphine overdose in breastfeeding newborns is small, the strategies listed in **Table 33-5** are recommended.[49-51] These guidelines, developed by the Motherrisk Program in Canada,[49] have been shown to reduce the incidence of newborn sedation following maternal use of codeine.[50]

Table 33-5 Strategies for Safe Use of Medications That Contain Codeine During Breastfeeding

Do not prescribe codeine-containing medications if the woman is known to be an ultra-rapid metabolizer for CYP2D6.
Consider prescribing an opioid analgesic other than codeine or tramadol if possible. Several randomized controlled trials have found that nonsteroidal anti-inflammatory drugs are equivalent to codeine with regard to the degree of pain relief reported. The American Academy of Pediatrics' Committee on Drugs states that morphine, hydromorphone, and butorphanol are preferred agents for breastfeeding women who require opioids for pain relief.
If codeine is prescribed:

Use the lowest possible dose that is effective,

Use the medication for the shortest duration possible, and no longer than 4 days. Central nervous system depression worsens in the newborn after 4 days, probably secondary to accumulation of morphine. After 4 days of use, the dose should be decreased or a different medication used.

When codeine causes central nervous system sedation (grogginess, somnolence), the degree of sedation is consistent between the mother and the breastfed infant. Therefore, if the mother shows signs of sedation, the newborn should be examined as well.

Caution women to avoid self-administering additional doses.

Teach women taking these medications about the danger signs in the newborn, which include increased sleepiness (sleeping more than 4 hours at one time), breastfeeding difficulties, limpness, or breathing difficulties.

Notify the infant's care provider that the woman is taking a codeine-containing medication.

Consider genomic testing for individual situations.

Based on Madadi P, Moretti M, Djokanovic N, et al. Guidelines for maternal codeine use during breastfeeding. *Can Fam Physician.* 2009;55:1077-1078[49]; Kelly LE, Chaudhry SA, Rieder MJ, et al. A clinical tool for reducing central nervous system depression among neonates exposed to codeine through breast milk. *PLoS One.* 2013;8(7):e70073.[50]

Populations at risk for being ultra-rapid metabolizers are identified and more information on genomic testing is presented in the *Pharmacotherapeutics* chapter. In addition, women should be instructed that the maximum dosage from all medications containing acetaminophen should not exceed 4000 mg in 24 hours.

Breast Engorgement

Breast engorgement occurs as lactogenesis II starts. This stage begins between 48 and 72 hours after birth in most women, but may not appear until up to 7 days postpartum.[52] It is theorized that engorgement of the breasts is due to a combination of milk accumulation and stasis as well as increased vascularity and congestion. This combination results in further congestion by virtue of lymphatic and venous stasis. Engorgement occurs as the milk supply increases and lasts approximately 24 to 48 hours. The breasts become distended, tense, and tender to the touch. The skin is warm to the touch, with visible veins, and is taut across the breasts. The nipples are firmer and may become difficult for the infant to grasp. Although engorgement is not an inflammatory process, the increased metabolism associated with milk production may cause a mildly elevated temperature. However, a fever of 38.0°C (100.4°F) or higher suggests the presence of mastitis or another infection, as discussed in the *Postpartum Complications* chapter. Women who begin breastfeeding immediately after the birth, nurse frequently on both breasts, and avoid the use of supplements or pumping to express milk for bottle-feeding, are less likely to experience painful engorgement.

Effective treatments for engorgement include application of cold packs, increasing the frequency of nursing, and use of mild analgesics.[53] Application of chilled cabbage leaves has been suggested based on the theory that a substance in the cabbage leaves called sinigrin will relieve engorgement. Studies have not found that cabbage leaves have a therapeutic effect, although application of anything cold will cause vasoconstriction and a reduction in engorgement pain.[53,54]

When a woman is not breastfeeding, comfort measures for engorgement are targeted toward pain relief and cessation of milk production. There are no medications approved for lactation suppression in the United States. Nevertheless, several strategies may be implemented to help with the discomfort and inhibit milk production: the use of good breast support by a supportive bra or breast binder, application of ice packs, and the use of analgesics. These women should use a bra or breast binder to firmly support the breasts, and may be more comfortable if the bra is also worn at night while sleeping. No attempt should be made to express milk from the breasts, either manually or by using heat, as this will simply promote milk ejection (i.e., let-down reflex) and further milk production. For example, the woman should avoid standing in a warm shower and letting the water run over the breasts. When using ice for comfort, caution should be used to not apply the ice directly to the skin. Engorgement will improve within 24 to 48 hours, although the presence of breast milk may take several weeks to resolve completely.

Nipple Pain

Nipple pain and trauma are very common in the initial days postpartum. Nipple pain can lead to discontinuing breastfeeding if untreated. Although most women will experience painful nipples at the onset of nursing that gradually resolve spontaneously, some women will experience blistering, severe pain, or nipples that develop cracks and bleeding. The differential diagnosis includes abnormalities in the way the newborn is positioned or sucking, superficial or ductal fungal infection, and Raynaud's disease. Assessment, diagnosis, and treatment of nipple pain are reviewed in more detail in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

Anhydrous lanolin has historically been recommended for treatment of nipple pain and nipple trauma. More recently, studies have shown that application of breast milk and protection with a breast shield is more effective in healing trauma and mitigating pain than is application of lanolin, which must be wiped away before breastfeeding.[55,56]

Uterine Cramping

Uterine cramping postpartum, commonly referred to as *afterpains*, is caused by the continuing sequential contraction and relaxation of the uterus. Painful cramping is more common in multiparous women and occurs most often during breastfeeding in the first few days after birth. Infant suckling stimulates secretion of oxytocin by the posterior pituitary, which then causes the uterus to contract. Having an empty bladder will lessen uterine cramping because a full bladder displaces the uterus upward, which is associated with more painful contractions. Use of heating pads or lying prone with a pillow or blanket roll under the lower abdomen also may relieve the discomfort of afterbirth pains. NSAIDs are generally very effective for treating uterine cramping and are generally needed for 48 to 72 hours at most.

Perineal Pain

Perineal pain can be treated with nonpharmacologic topical agents, oral analgesics, or topical anesthetics. A helpful toileting technique includes rinsing with warm water from a peri-bottle

during urination, wiping front to back, and patting any incision area dry instead of rubbing or wiping. If perineal discomfort does not progressively improve with time, reexamination should be performed to rule out pathology, including infection.

Ice packs or cold gel packs are most useful for reducing swelling and numbing the perineum in the first 24 hours. All ice packs should be wrapped in a soft covering, with the absorbent side out, for protection against freezing injury; they should not be applied for more than 20 minutes.

Witch hazel compresses may be applied to the perineum or anal area and held in place by the sanitary pad. A 4 × 4 gauze soaked in witch hazel or a ready-to-use commercial witch hazel product may be used. There are no studies that have evaluated the effectiveness of witch hazel. Similarly, the few studies that have compared topical anesthetics or topical anti-inflammatory creams have demonstrated no significant difference between the topical medications and cooling agents.[44,57,58]

When analgesics are needed for perineal pain, NSAIDs provide effective pain relief for most women. Opioid analgesics should be reserved for women whose pain is not well controlled with moderate doses of an NSAID.[44] The constipation that can be caused by opioids can exacerbate perineal pain.

Hemorrhoids and Constipation

Pushing during the second stage of labor can cause hemorrhoids to become edematous and traumatized. The nonpharmacologic comfort techniques listed in **Table 33-6** may be used in combination, except that cold and warm options should not be used during the same time span.

Table 33-6	Comfort Measures for Women with Hemorrhoids
Ice packs	
Cold sitz baths	
Warm water compresses	
Warm sitz baths	
Hemorrhoidal medications:	
Ointments	
Analgesic or anesthetic spray	
Witch hazel compresses (Tucks pads)	
Hydrocortisone suppository	

In addition, a mild stool softener can prevent painful bowel movements that occur when the stool is hard. Increased fluid intake and a high-fiber diet also will promote normal function. Stool softeners or laxatives should be discontinued if the stools become too soft or liquid. Suppositories should be avoided if the woman has sustained a third- or fourth-degree laceration.

Postoperative Care Following Cesarean Birth

While the goals of postpartum care are similar after cesarean birth, some unique circumstances must be considered. In the immediate postoperative period, vital signs, uterine tone, and bleeding are monitored quite frequently. The risk of hemorrhage (intra-abdominal or vaginal) is highest during this initial period. Typically, nursing, anesthesia, and surgical staff care for women during this period. Common postpartum complications associated with cesarean birth are reviewed in the *Postpartum Complications* chapter.

As the effects of regional anesthesia resolve, oral analgesics are used for pain control, the Foley catheter is removed, and ambulation is initiated. Food and fluids can be ingested as tolerated, and intravenous hydration is discontinued when the woman can take sufficient amounts of oral liquids. Care activities are directed toward preventing complications such as deep vein thrombosis, ileus, and endometritis.

The increased risk of deep vein thrombosis in the puerperium is magnified by the woman's immobility after surgery. Strategies to mitigate this risk include pneumatic compression devices until the woman is fully ambulating and initiation of early ambulation. While institutional guidelines may vary, there is no evidence for benefit from withholding food or liquids after cesarean birth.[59] Unrestricted oral intake after cesarean birth accelerates the return of gastrointestinal function, and bowel sounds should be audible within 12 to 24 hours after surgery.

Prophylactic antibiotic therapy is unnecessary after cesarean birth if a single antibiotic dose was administered before the incision is made or after umbilical cord-clamping. Women who were receiving group B *Streptococcus* (GBS) prophylaxis will similarly have this treatment discontinued immediately after surgery. However, if the woman develops chorioamnionitis during labor, antibiotics may be either discontinued immediately after birth or continued until the woman is afebrile for 24 hours, depending on the individual circumstances.

Postpartum anemia is more likely after a cesarean birth, because the typical average blood loss with cesarean birth is 1000 mL. Strategies for pain control after surgery may vary, but the goal is to transition to oral pain medications by the time of discharge. When staples were used to close the abdominal incision, the staples may be removed before discharge from the hospital or at a return visit to an ambulatory site within the first week. Timing of the removal depends on the surgeon's preference and individual risks for wound dehiscence.

A woman usually is discharged from a hospital, assuming no major complications, approximately 48 to 72 hours after the cesarean birth. Recently, some studies have found that the amount of opioid prescribed after cesarean birth at the time of hospital discharge, may exceed the amount needed and consumed.[60] Because approximately 1 in every 300 opioid naïve women prescribed opioids after cesarean become persistent users, it is important to individualize the type of drug and amount ordered in discharge prescriptions.

Postpartum health education should include warning signs for surgical-site infections and appropriate instructions regarding physical activity after major abdominal surgery. These recommendations often include advising the woman to lift nothing heavier than the infant and to avoid other activities that may put excessive strain on the surgical site.

Postoperative Care Following Tubal Ligation

Postpartum tubal sterilization is usually performed the day after a vaginal birth or during a cesarean birth. When a tubal ligation is planned, the woman will be asked to discontinue all oral intake for 6 to 8 hours prior to the sterilization procedure. If an epidural was used during labor or birth, the epidural catheter will be retained in place and used during the sterilization procedure. Typically the surgical incisions for tubal sterilization are small, dressed with simple adhesive bandages, and require no special postoperative care. No additional restrictions or instructions are needed for the postpartum tubal sterilization. Additional information regarding types of sterilization and health education can be found in the *Nonhormonal Contraception* chapter.

Early Postpartum Education

Fatigue, excitement, and feeling overwhelmed may limit the ability of new parents to learn too much new information. Therefore, health education in the early puerperium is prioritized and targeted to the needs of each woman and her family. Assessing the parents' learning needs is the first step. Assumptions may be misleading, such as expecting that a multiparous woman already has a wealth of knowledge about recommended infant care or that a primiparous woman is unfamiliar with personal or neonatal care. It may be helpful to focus on essential topics in discussions, then supplement that verbal health education with written materials or electronic sources and provide contact information for further help in the community. Including available family members in postpartum teaching may maximize the benefits. Recommended patient education topics are listed in **Table 33-7**. Important signs of complications are listed in **Table 33-8**. Discomforts specific to breastfeeding (nipple tenderness and cracked nipples) are discussed in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

Table 33-7	Early Postpartum Education Topics
Physical Care	
Breast care during breastfeeding and engorgement	
Perineal care and pelvic floor exercises (Kegel)	
Activity, exercise, nutrition, and rest	
Hygiene, including tub bath or shower as preferred	
Signs and symptoms of postpartum complications and postpartum mood disorders	
Relational Care	
Emotional self-care (postpartum blues)	
Family planning	
Transitional family relationships	
Partner's needs and fears	
Need for time together with partner and separate from the baby	
Postpartum sexuality	
Sibling rivalry	

Table 33-8	Postpartum Signs of Complications
Woman	
Sudden-onset headache, visual changes, or epigastric pain	
Fever or chills	
Redness, heat, firmness, or pain in one breast	
Increased abdominal pain or vulvar pain	
Excessive vaginal bleeding	
Calf pain, heat, or swelling	
Marked changes in mood	
Newborn	
Feeding difficulties	
Yellowing skin (jaundice)	
Fever	
Inability to console	
Insufficient wet/dirty diapers	
> 6 stools per 24 hours/bloody or very watery stools	
Listless behavior, not alert when awake	
Respiratory difficulties, turning blue	

Pelvic Floor Exercises

Teaching women to perform pelvic floor exercises (also known as Kegel exercises) is another strategy for promoting long-term perineal comfort and strength. Performing pelvic floor exercises has been shown to reduce the prevalence of stress urinary incontinence in women during pregnancy and postpartum[61] and to improve sexual function postpartum.[62] Kegel exercises may also increase circulation to the area, promote healing, restore tone to the pelvic musculature, and facilitate ease of movement. The woman may tighten her perineum, drawing it up and in and maintaining this contracted state while moving in bed or lowering herself onto a chair. For strengthening muscle tone, the exercises can be done in sets of 10, repeatedly tightening and relaxing the muscles for 5 to 10 seconds. It is helpful to repeat these sets two to three times daily—a regimen that will avoid direct pressure to the sensitive areas. If the woman has undergone a mediolateral episiotomy, tightening the muscles when performing Kegel exercises pulls on the posterior end of the suture line because the incision interrupts muscles diagonally and may be too uncomfortable to perform until the perineum is healed.

Sexuality Postpartum

There is wide variation in the time after childbirth that women resume sexual activity with their partners, as well as cultural variations in prescribed waiting times. Factors that are correlated with less sexual activity and dyspareunia include operative vaginal delivery, episiotomy, third- and fourth-degree lacerations, and urinary incontinence.[63] Other circumstances that may interfere with postpartum sexuality are reduced privacy, increased

interruptions, and fatigue. There is no scientific basis for the traditional recommendation to delay sexual activity until the 6-week postpartum examination. Instead, a more nuanced approach is that the woman should wait until she feels comfortable and ready to resume sexual activity. Additional recommendations include using vaginal lubrication, particularly if the woman is lactating; trying alternative positions for comfort during penetrative vaginal intercourse; and engaging in other sexual and nonsexual forms of intimacy.

Contraception

The early postpartum period is an opportune time to explore contraceptive plans with the woman. While sexual activity may be far from the mind of the woman who has recently given birth, as many as 50% of women will have initiated sexual activity by the time they return to an office for the traditional postpartum examination at 6 weeks.[64] In women who are not breastfeeding, ovulation returns at a mean time of 39 days after birth. In breastfeeding women, the return of ovulatory cycles varies a great deal, depending on the duration and frequency of breastfeeding. Although it is not common, approximately 1% to 5% of breastfeeding women who are amenorrheic are ovulatory.[65]

It is best to open the discussion with an inquiry regarding the woman's desire for future pregnancy and perceived need for contraception. Insertion of an intrauterine device (IUD) may be performed safely after delivery of the placenta, although expulsion rates are higher than if insertion is performed earlier than 4 weeks postpartum.[66] Depot medroxyprogesterone acetate (DMPA) may be offered at the time of postpartum discharge. However, because a woman in the postpartum period is still in a state of hypercoagulation, estrogen-containing methods should not be used until at least 3 to 4 weeks postpartum according to the U.S. Medical Eligibility Criteria for Contraceptive Use, although other sources suggest a longer waiting period.[67]

The impact of the woman's lactation status on contraceptive options and timing is controversial. According to the U.S. Medical Eligibility Criteria,[67] lactation is not a contraindication to the use of any contraceptive options. Other authorities warn against the use of combined hormonal contraceptives such as oral contraceptive pills, patches, and vaginal rings because of concerns that these contraceptive methods reduce milk supply. For similar reasons, they may recommend delaying use of high-dose progestins such as DMPA for several weeks.

Another concern is the postpartum use of the progestin-only pill (POP) by Hispanic women who had gestational diabetes. In a study of 904 Latina women, POP use was associated with an almost three times greater risk of developing type 2 diabetes within 2 years relative to use of nonhormonal or combined hormonal contraceptives (adjusted RR, 2.87; 95% CI, 1.57–5.27). This risk increased with duration of uninterrupted use.[67] Additional studies of DMPA among women in this population found an increased risk of developing diabetes, especially for breastfeeding women, but one that was not statistically significant.[67] **Table 33-9** lists the recommendations for contraception for women who are postpartum, including differences based on whether they are breastfeeding.[67]

Table 33-9 U.S. Medical Eligibility Criteria for Postpartum Contraception, 2016

Condition and Subcondition	Copper-IUD	LNG-IUD	Implant	Injection	POP	Combined Pill, Patch, Ring
Postpartum (Including Cesarean Delivery)						
< 10 minutes after delivery of the placenta, breastfeeding	1	2				
< 10 minutes after delivery of the placenta, not breastfeeding	1	1				
10 minutes after delivery of the placenta to < 4 weeks	2	2				
≥ 4 weeks	1	1				
Postpartum sepsis	4	4				
Postpartum, (Breastfeeding)						
< 21 days postpartum			2	2	2	4
21 to < 30 days postpartum with other risk factors for VTE			2	2	2	3
21 to < 30 days postpartum without other risk factors for VTE			2	2	2	3
30–42 days postpartum with other risk factors for VTE			1	1	1	3
30–42 days postpartum without other risk factors for VTE			1	1	1	2
> 42 days postpartum			1	1	1	2
Postpartum (Not Breastfeeding)						
< 21 days			1	1	1	4
21–42 days with other risk factors for VTE			1	1	1	3
21–42 days without other risk factors for VTE			1	1	1	2
> 42 days			1	1	1	1

Abbreviations: IUD, intrauterine device; LNG, levonorgestrel; POP, progestin-only pill; VTE, venous thromboembolism.

Category 1: A condition for which there is no restriction for the use of the contraceptive method.

Category 2: A condition for which the advantages of using the method generally outweigh the theoretical or proven risks.

Category 3: A condition for which the theoretical or proven risks usually outweigh the advantages of using the method.

Category 4: A condition that represents an unacceptable health risk if the contraceptive method is used.

Modified with permission from Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR Recomm Rep.* 2016;65(RR-3):1-104.[67]

Postpartum Care Plan

The time period between discharge from direct care to a late postpartum office visit can present considerable challenges to women both physically and psychologically. Before the woman is discharged from care given during the early postpartum period, several items should

be reviewed. The woman should have the contact information for a postpartum visit and plan for when that visit will occur. Contraception planning will include education about when to initiate the method, effectiveness, and risks associated with the chosen method. Women who are breastfeeding may need resources such as lactation counseling. In addition, women should be educated about the signs of postpartum depression, and given resources for accessing care if needed. Any pregnancy-related or postpartum complications should be reviewed and plans made as needed for appropriate follow-up.

Mid-Postpartum Period

Historically, the defined endpoint of the puerperium is 6 weeks—a benchmark likely chosen because by that time the woman's uterus has completed its involution to the nonpregnant state. Traditionally, women have not been seen for a healthcare visit during the time period between discharge and 6 weeks postpartum. Women are at risk for a number of complications during that time span,[68-74] yet their interactions with the healthcare system are often fragmented and insufficient. Although both evidence and consensus are lacking regarding the best approach to maternal and newborn follow-up postpartum, the first 6 weeks postpartum should be viewed as a time during which women continue to benefit from intensive contact with healthcare providers. Moreover, the postpartum period overall should be reconceptualized as a process that occurs over the course of a year following childbirth.[75,76]

Care During the First Six Weeks Postpartum

Contact with a midwife or other healthcare provider during the first 6 weeks postpartum may be beneficial for all women. Interval care during this period can be performed in several ways. Some midwives make telephone calls, some do home visits, and others see the woman and/or infant for a 2-week office visit. In addition, some midwives may work with nurses or train birth assistants to initiate the telephone calls or perform the home visits. Many midwives combine these activities depending on the needs of the woman, infant, and family. Office face-to-face visits are recommended for women who had hypertension, a cesarean birth, breastfeeding difficulties, and chronic medical conditions, and for any woman who has an identified risk for developing postpartum depression.[69]

Phone Calls

Using a telephone call to assess the mother–infant dyad's well-being, particularly if the birth occurred in a setting where the family will be alone with their infant shortly after birth, allows the midwife to triage for emergent problems that might require a trip to the office, the pediatric provider's office, or even the emergency department. Telephone calls are usually made the day after the birth or the day after the woman returns home from the birth center or hospital, and again a few days later. During the telephone call, the midwife screens for problems by asking about both maternal and infant well-being. A comprehensive assessment of the woman's condition by telephone may include obtaining her self-report of physical and emotional well-being, breastfeeding success, and coping with the significant family changes associated with childbirth. The midwife should ask about the presence of signs or symptoms for postpartum complications. Content for the phone call is listed in **Table 33-10**.

Table 33-10	Postpartum Telephone Call: Maternal Assessment
System	**Assessment**
General	Food and fluid intake

	Sleep quality and quantity
	Fever or chills
	Headaches
Breasts	Breast engorgement, nipple condition, pain, masses
	Progression from colostrum to milk
	Feeding frequency and success
Abdomen	Abdominal pain, cramping, epigastric pain
	Bowel function, constipation, incontinence
	Urinary pain or incontinence, urgency
Pelvis	Lochia amount, color, and odor
	Perineal pain, swelling, or redness
	Hemorrhoids
Lower extremities	Edema, pain, redness, varicosities
Adjustment	Emotional changes (postpartum blues or depression)
	Coping with life changes and response to newborn
	Social support and help with household chores
	Physical activity level
	Abdominal and perineal exercises
	Family adjustment to newborn
	Confidence in infant care

The telephone call also provides an opportunity to check on the newborn's condition, regardless of whether the midwife is the primary healthcare provider for the newborn. This conversation can be an important opportunity to triage for adequacy of infant feeding and signs of hyperbilirubinemia, infection, or heart disease. Assessment of the newborn by telephone is outlined in **Table 33-11**.

Table 33-11 **Postpartum Telephone Call: Newborn Assessment**

System	Assessment
General	Temperature
	Sleep patterns
	Consolability
	Alert and interacting with parents
Feeding	Frequency and amounts of formula feeding
	Breastfeeding latch, milk supply, frequency
	Infant satisfaction with feeding
Diaper patterns	Frequency of voids and stools
	Appearance of stools
Skin color	Jaundice
	Cyanosis
Umbilicus	Presence of erythema or exudate at umbilical site
	Cord stump fallen off
Circumcision	Appearance of glans if circumcised male

Limitations of postpartum phone calls include the inability to directly screen for neonatal hyperbilirubinemia and excessive infant weight loss and the requirement of reliable telephone service for the woman.

Home Visits

A home visit involves the same evaluations as does a telephone call but also allows the midwife to observe the woman's responses to the infant's needs and cues, the parent–newborn interaction, the place of the newborn in the home's social environment, and resources in the home (e.g., plumbing, water supply, refrigeration, air conditioning/heat, window screens, supplies for baby care). A home visit may also include a brief physical examination of the woman and newborn.

Two-Week Office Visit

Assessments performed at a 2-week visit focus on expected physical, physiologic, and psychological changes in the postpartum period. Particular attention should be paid to how well the woman is coping and an assessment for postpartum depression. A targeted physical examination may be performed as indicated.

If the woman brings her infant to the 2-week visit, the midwife can observe the woman's interaction with her infant and her responsiveness to the infant's needs and cues. Every contact with a new mother is an opportunity to share with her the developmental capabilities of her infant and to discuss safety, infant stimulation, and parenting skills. This is also a good time to verify that the infant either has already seen or has an appointment to see a pediatric healthcare provider and to discuss the need for these visits and for immunizations.

For breastfeeding women, the 2-week visit also can be used as a time to support effective breastfeeding and target any problems. If the woman needs to select a contraceptive method, the 2-week visit is an excellent opportunity to review available options. Many couples will choose to begin sexual intercourse shortly after the woman's lochia resolves, if not before, and women who are formula feeding may ovulate before a routine postpartum physical is performed at 4 to 6 weeks.

Six-Week Postpartum Visit

The exact timing of the postpartum examination is flexible and may be done as early as 4 weeks postpartum. The significance of this office-based visit goes beyond the confirmation of the woman's return to the nonpregnant state. As the final visit related to maternity care, it marks the end of the heightened attention the woman received from the healthcare system during the childbearing year. The idea that the woman's health needs are now met takes a very narrow view of women's health given all the physical, emotional, and social changes that women experience in the first year after giving birth.[69-76] Ideally the postpartum visit will be the beginning of a new phase of supportive health care, rather than merely the end of a maternity package of services. Viewed in this way, the postpartum examination will include each of the

elements described here in addition to the recommended screening and counseling provided during a periodic well woman visit as described in the *Health Promotion Across the Lifespan* chapter.

The 4- to 6-week postpartum consists of a comprehensive history, physical examination, and pelvic examination, as outlined in the Introduction to the Care of Women chapter. Available records of prenatal, intrapartum, and postpartum care during this pregnancy should be reviewed. A description of the procedure for this postpartum visit is presented in Appendix 33B. Topics that should be addressed during this visit are listed in **Table 33-12**.[74-80]

Table 33-12 Topics for 6-Week Postpartum Visit

Topic	Discussion
Review of pregnancy and birth	Provide an opportunity for the woman to ask any questions she has about her experience. If the woman had complications such as gestational diabetes, hypertensive disorders, or preterm birth, she should be counseled that these conditions increase her lifetime risk for cardiometabolic disease.
Complications experienced during pregnancy or birth	Gestational diabetes: Order fasting plasma glucose or 75-g, 2-hour glucose tolerance test. Hypertension, obesity, diabetes, renal disease: Plan for follow-up as indicated. Stillbirth or miscarriage: Visit for review of autopsy findings, emotional support, bereavement counseling as needed.
Postpartum symptoms	Address urinary incontinence, breastfeeding difficulties, dyspareunia, weight loss, smoking cessation, and substance abuse, as indicated.
Reproductive life plan	An interconception interval of 18 months or more is recommended. For women who chose to use a contraceptive method, establish a plan and review the method's effectiveness, instructions, and side effects.
Anticipatory guidance	Nutrition, physical activity, sexuality, returning to work.
Assess for postpartum mood disorders	Use a validated screening tool for postpartum mood disorders that includes screening for anxiety and depression.
Plan for primary care provider and timing of visit	At the end of the postpartum visit, the woman should have a primary care provider identified and recommendations for timing of the next visit.

Management Plans

A review of the prenatal record will identify conditions from the pregnancy that have not resolved. For example, a woman may need follow-up care for an ovarian or renal cyst identified by ultrasound during the pregnancy. Based on the scope of the midwife's practice, follow-up care should be arranged within the service or by referral to another primary care provider. Screening for postpartum depression is recommended for all women who attend a postpartum visit.[81,82]

Based on the history and physical examination, the midwife may be asked to complete an authorization for maternity leave or return to employment. Proper evaluation should include an understanding of the type of work that the woman performs. Midwives are in a position to advocate for women who desire to continue breastfeeding after returning to work and to offer

guidance on strategies to continue lactation.

Women who need contraception should receive necessary prescriptions, appointments, and education. It is not necessary to wait for menses to resume before starting oral contraceptives or inserting an IUD. If the woman has engaged in unprotected penile–vaginal intercourse, use of emergency contraception and/or urine pregnancy testing after 2 weeks of abstinence or condoms can reasonably ensure that the woman is not pregnant. The postpartum visit is also an opportune time to discuss future plans for pregnancy and provide preconception counseling.

Nutrition and exercise are topics of great importance to many women. By 6 weeks postpartum, most women should be physically ready to resume regular exercise routines, in addition to abdominal and pelvic floor exercises to restore tone to those muscle groups most impacted by pregnancy and childbirth. According to the Physical Activity Guidelines for Americans, women should get at least 150 minutes of moderate exercise per week.[83] It may be advisable to gradually increase activity to this level of exercise when reinitiating activity postpartum. Based on limited evidence, there is no concern that exercise or calorie restriction will adversely impact lactation or infant growth.[84,85] Additional recommendations for physical activity can be found in the *Health Promotion Across the Lifespan* chapter.

Many women desire weight loss to return to their prepregnancy weight or to achieve another weight goal. The best way to achieve these goals is through a combination of healthy nutrition and exercise and by identifying what constitutes a healthful weight goal based on body mass index and other factors. Women should be cautioned against rapid weight loss postpartum; a useful reminder is that it took 9 months to gain the weight, so it may take at least the same time to lose it. A detailed discussion regarding dietary recommendations can be found in the *Nutrition* chapter.

The Journey to Parenthood: Psychological Adaptation to Childbearing

Becoming a parent is a complex process through which a woman achieves competence in the role and integrates maternal behaviors into her identify.[86] This process is influenced by many factors. External factors include society's view of motherhood, cultural expectations, and supports or obstacles within the person's personal environment. Many internal factors are involved as well—for example, knowledge, previous exposure to children, the woman's experience in being nurtured, preexisting mental health conditions, relationship status, and personal expectations, to name just a few.[87] Intense emotional feelings, rapid hormonal changes, physical recovery, and fatigue are also part of the postpartum milieu. In this setting, the woman must reconcile her "imagined baby" with the real newborn, get to know and care for the newborn, meet the demands of this 24-hour responsibility, and adapt to her new role as a parent. The postpartum period can be difficult for anyone, but is even more difficult for women who experience a traumatic birth or other trauma. The process is interactive and ongoing.

Midwifery assessment of the process of becoming a parent requires three discrete evaluations: (1) observation of maternal–infant interactions to determine how well maternal–infant attachment is progressing, (2) a review of the woman's environment to identify supports and factors that may impede progress, and (3) a formal assessment for signs of postpartum mood disorders.

This chapter briefly reviews the psychologic process of becoming a mother. Studies of women's experiences postpartum have generally included small samples and different methodologies, which make it difficult to synthesize their results as a body of work. Nevertheless, feelings of fatigue, being overwhelmed, loss of control, and loss of identity are frequently reported.[88] Women's reports of their needs during postpartum consistently refer to four items: (1) more information about their infant, (2) psychological support, (3) a need to share the experience, and (4) practical or material help.[73,89] These four items provide a good framework for midwifery assessment.

Postpartum mood disorders such as anxiety, post-traumatic stress disorder, and depression are inexorably intertwined with preexisting and prenatal mental health conditions. A detailed review of postpartum mood disorders and how to screen for them is presented in the *Mental Health Conditions* chapter, where the reader will also find information about preexisting mental health conditions and prenatal mental health conditions that can have an impact on the postpartum transition.

Traumatic Birth

The events of childbirth are difficult to predict and often do not match the couple's birth plan. Differences between the planned labor and birth and the reality may include use of analgesia or anesthesia, mode of delivery, gender of the newborn, and the quality of support provided in labor. For some families, the experience goes beyond disappointment, as the parents must grieve after a loss. Examples of losses include stillbirth or early neonatal death, loss of

fertility after an emergency hysterectomy, the discovery of congenital birth defects, voluntary or involuntary relinquishment of the newborn to adoptive parents or the legal system, and admission to the neonatal intensive care unit. The couple's grief reactions to these outcomes will be unique to their specific circumstances, coping strategies, and social support.

A traumatic birth places a woman at risk for post-traumatic stress disorder (PTSD). In the Listening to Mothers II national survey of childbirth, 9% of women met diagnostic criteria for PTSD, and 18% of women experienced significant levels of post-traumatic stress symptoms 7 to 18 months after childbirth.[90]

In most situations involving fearful events, the act of listening is by itself therapeutic. After listening to women tell their experiences of birth during the early postpartum period, midwives may identify those who could benefit from formal counseling after traumatic birth. There are mixed results from randomized trials of debriefing after traumatic birth in reducing symptoms of PTSD or postpartum depression. Women most likely to benefit are those who request counseling or already meet some of the criteria for PTSD.[91,92] Conversely, debriefing can cause a reexperience of the trauma and be more harmful than helpful.

Special Considerations

Smoking

While many women are successful in quitting smoking during pregnancy, relapse is common during the postpartum period. In a multistate study using the population-based Pregnancy Risk Assessment Monitoring System (PRAMS), 48% of women resumed smoking within 6 months postpartum, and those women who received smoking cessation interventions in pregnancy were no more successful than the majority of women who had not received prenatal intervention.[93]

Some interventions to help women remain abstinent from smoking have shown promise, although there is no single intervention that is effective for all women. One telephone support program based on motivational interviewing and relapse prevention principles provided phone calls at 3, 6, 9, and 12 weeks and was tested in an RCT. Of the women receiving the intervention, 74% remained abstinent at 12 weeks, compared to 37% in the control group.[94] In a different study, mailing self-help booklets proved helpful in maintaining higher abstinence rates, particularly for low-income women.[95] Midwives should offer women information about available community programs for maintaining abstinence and educate them on the important health benefits of not smoking for themselves and their children. Additional information about strategies used for smoking cessation can be found in the *Health Promotion for Women Across the Lifespan* chapter.

Adolescents

Becoming a parent at a young age may have negative consequences for both the adolescent and infant. Social and economic factors, rather than biology, are likely responsible for these consequences. Adolescents in the postpartum period are more likely than peers who delay pregnancy to experience postpartum depression, lower self-esteem, substance abuse, and poverty. Further, adolescent parents are less likely to attain higher levels of education and are more likely to experience intimate-partner violence than their peers.[96]

A variety of programs exist for providing care to adolescent mothers postpartum. The goals of these programs usually include improvement of the individual's parenting skills, provision of social support, support for educational attainment, and prevention of repeat pregnancy through use of contraception. Programs may be provided in the home, in the community or school, or in clinics.

Many researchers have investigated the ideal method of contraception for adolescents during the postpartum period and have focused on long-term reversible contraceptive (LARC) methods such as intrauterine devices, implants, and injectable progestins. Adolescents who had 3-year etonogestrel implants inserted prior to postpartum discharge if in a hospital or birth center had longer continuation rates and longer delays to next pregnancy compared to those who received IUDs, DMPA, or oral contraceptives.[97]

Transgender Men

Transgender men may have a very different postpartum experience than cisgender women. Ideally, the midwife and the pregnant transgender man will have planned during pregnancy for postpartum care and decision making. Care considerations include increasing the sensitivity and support from hospital or birth center staff, decisions on whether to feed the infant with human milk or formula, and the timing of testosterone therapy.

Transgender men may be more comfortable with referring to lactation as chestfeeding rather than breastfeeding.[14] Transgender men may have had chest surgery such as bilateral mastectomy that reduced or eliminated their ability to lactate. Others may have delayed such surgery until after childbearing or decided not to have surgery. Transgender men may have varying comfort with lactation because of its association with female body function.[98]

Although limited evidence suggests that testosterone is not excreted into human milk,[14,99,100] some transgender men may elect to postpone resumption of testosterone therapy until after weaning. Whether or not the transgender man chooses to lactate, there is no research to suggest the optimal timing for resumption of testosterone therapy.

Transgender men whose sexual partners are biologically male may need contraception.[14,100] Anticipating these clinical decisions and communicating using an open and respectful approach will optimize the individualization of postpartum care for transgender men and their families.

Infant in the Neonatal Intensive Care Unit

Prematurity is the most likely reason for a newborn to need intensive care, with approximately 12% of all newborns being born prematurely in the United States. Other reasons for neonatal intensive care unit (NICU) admission include hyperbilirubinemia, hypoglycemia, infection, meconium aspiration, birth asphyxia, congenital anomalies, and respiratory distress. Having an infant in the NICU can interfere with the developing maternal–newborn attachment through restricted access to the newborn, difficulty in reading the infant's cues, and inability to commit one's time exclusively to the newborn owing to competing demands both at home and at the hospital. The hospitalization of a newborn is associated with high rates of post-traumatic stress and symptoms of depression.[101] Breastfeeding the hospitalized infant is particularly a challenge when the infant is premature and lacks the neuromuscular development to successfully suckle.

Midwives can do much to support these families when the focus of the healthcare team turns toward the fragile infant.[102] Support for the establishment and continuation of breastfeeding includes initiating pumping with a hospital grade mechanical pump within 6 hours of the birth, and continuing to pump milk at least 6 times per day. Milk supplies may be increased by using techniques such as "hands-on pumping." Midwives may advocate for parents to be able to hold their ill infants in skin-to-skin contact even with monitors and ventilation equipment in place.[103] Parents should be monitored for psychological consequences of the infant hospitalization, with attention being paid to their emotional health.

Relinquishment/Adoption of an Infant

Women may relinquish the care of their infant voluntarily through adoption or surrogacy, or

involuntarily through the legal system. Under these circumstances, midwives might be involved in the care of the birth parents, the adoptive or receiving parents, and/or the newborn. The circumstances around the adoption or removal of the infant and emotional reactions are complex and unique to each family.

Support for the woman who gives birth includes providing psychosocial support, therapeutic communication, follow-up services, and advocacy. The midwife can talk with the woman about her emotions in a nonjudgmental way, validating her feelings and acknowledging that feelings may be conflicted. As the woman's advocate, the midwife can ensure that the decision to relinquish an infant for adoption was not coercive except under the condition of legal removal by child protective services. Referrals to social workers and counselors may be offered to address ongoing issues of grief and visitation.

As primary care providers for women, midwives may also be in the position of caring for women who adopt an infant. The decision to adopt often follows failed attempts at getting pregnant and possibly multiple miscarriages. Thus the emotional reaction to adoption can be complex, as these parents struggle with a sense of legitimacy as parents and fear that the infant will be returned to the biological parents. Postpartum depression is possible for women who become mothers by adoption, as they experience stressors similar to those faced by women who have given birth.[104] Women who adopt will likely have educational needs about infant care and may attempt to initiate lactation. Referral to resources and support groups for adoptive families may be helpful.[105] Laws regarding adoption vary according to jurisdiction, so midwives should be aware of credible agencies and attorneys in this field.

Women in Prison

Incarceration presents another circumstance in which the infant will be separated from the woman during the postpartum period. This group of women presents special challenges for the provision of postpartum care due to the likelihood of separation of the infant from the woman when she returns to the correctional facility. Prison nurseries, in which infants are housed with their incarcerated mothers, represent an alternative to the forced separation of infants from their mothers, but relatively few of these special units are available. When prison nurseries are unavailable, community-based residential parenting programs and placement of the infant with relatives are options for continuing the care of the infant within the family.[106,107]

Midwives can advocate for these families and coordinate with prison health providers to provide comprehensive perinatal and postpartum care. While providing care to women who are prisoners, it is important to maintain privacy while in the presence of guards and to avoid the use of restraints for reasons of safety and respect.

Conclusion

The puerperium is a time of great physical and psychological change. Midwives can provide effective care by attending to the woman in the context of her environment, family, and culture. It is important to remember that the postpartum period is not just the end of the childbearing year, but also the beginning of the nonpregnant phase of the woman's life. There is a need to address the high rates of problems such as incontinence, dyspareunia, and depression that can be unintended consequences of otherwise normal pregnancy and childbirth. By moving beyond the care of individual women, midwives can address the health of women in general through advocacy at the system level. In the context of rising healthcare costs and the desire to increase healthcare access to the underserved, there will be new challenges and opportunities for promoting the health of women.

Resources

Organization	Description	Webpage
Postpartum Support International (PSI)	PSI has a mission of support, education, advocacy, and research for people living with mental illness.	http://www.postpartum.net
Postpartum Progress	Focuses on raising awareness, reducing stigma, providing social support, and connecting mothers to help for perinatal mood and anxiety disorders such as postpartum depression.	http://postpartumprogress.org
Centers for Disease Control and Prevention (CDC)	The U.S. Medical Eligibility Criteria (U.S. MEC) for Contraceptive Use is a resource for appropriate selection of contraception for women with various conditions. Includes print version, mobile apps, and charts.	https://www.cdc.gov/reproductivehealth/contraception/contraception_guidance.htm
Ethnomed	Information on health care and cultures organized by clinical topic, culture, and language. Includes information for practitioners and patients.	https://ethnomed.org

References

1. Andres E, Baird S, Bingenheimer JB, Markus AR. Maternity leave access and health: a systematic narrative review and conceptual framework development. *Matern Child Health J*. 2016;20(6):1178-1192.
2. Earle A, Mokomane Z, Heymann J. International perspectives on work–family policies: lessons from the world's most competitive economies. *Future Child*. 2011;21(2):191-210.
3. Waugh LJ. Beliefs associated with Mexican immigrant families' practice of *la cuarentena* during postpartum recovery. *J Obstet Gynecol Neonatal Nurs*. 2011;40(6):732-741.
4. Raman S, Srinivasan K, Kurpad A, Razee H, Ritchie J. "Nothing special, everything is maamuli": Socio-cultural and family practices influencing the perinatal period in urban India. *PLoS One*. 2014;9(11):e111900.
5. Raven JH, Chen Q, Tolhurst RJ, Garner P. Traditional beliefs and practices in postpartum period in Fujian Province, China: qualitative study. *BMC Pregn Childbirth*, 2007;7(8):1-11.
6. Callister LC, Eads MN, Diehl J. Perceptions of giving birth and adherence to cultural practices in Chinese women. *Am J Matern Child Nurs*. 2011;36(6):387-394.
7. Lundberg PC, Trieu TNT. Vietnamese women's cultural beliefs and practices related to the postpartum period. *Midwifery* 2011;27(5):731-736.
8. Geçkil E, Sahin T, Ege E. Traditional postpartum practices of women and infants and the factors influencing such practices in south eastern Turkey. *Midwifery*. 2009;25(1):62-71.
9. Schim SM, Doorenbos A, Benkert R, Miller J. Culturally congruent care: putting the puzzle together. *J Transcult Nurs*. 2007;18(2):103-110.
10. Coast E, Jones E, Lattof SR, Portela A. Effectiveness of interventions to provide culturally appropriate maternity care in increasing uptake of skilled maternity care: a systematic review. *Health Policy Plan*. 2016;31(10):1479-1491.
11. Callister LC. Cultural meanings of childbirth. *J Obstet Gynecol Neonatal Nurs*. 1995;24(4):327-331.
12. Renaud MT. We are mothers too: childbearing experiences of lesbian families. *J Obstet Gynecol Neonatal Nurs*. 2007;36(2):190-199.
13. Ellis SA, Wojnar DM, Pettinato M. Conception, pregnancy, and birth experiences of male and gender variant gestational parents: it's how we could have a family. *J Midwifery Womens Health*. 2015;60(1):62-69.
14. Obedin-Maliver J, Makadon HJ. Transgender men and pregnancy. *Obstet Med*. 2016;9(1):4-8.
15. Klaus MH, Kennell JH. *Maternal–Infant Bonding: The Impact of Early Separation or Loss on Family Development*. Saint Louis, MO: Mosby; 1976.
16. Widström A-M, Lilja G, Aaltomaa-Michalias P, Dahllof A, Lintula M, Nissen E. Newborn behavior to locate the breast when skin-to-skin: a possible method for enabling early self-regulation. *Acta Paediatr*. 2011;100:79-85.
17. Sullivan R, Perry R, Sloan A, Kleinhous K, Burtchen N. Infant bonding and attachment to the caregiver: insights from basic and clinical science. *Clin Perinatol*. 2011;38:643-655.
18. Goulet C, Bell L, Tribble DS, Paul D, Lang A. A concept analysis of parent–infant attachment. *J Adv Nurs*. 1998;28(5):1071-1081.
19. Kim P. Human maternal brain plasticity: adaptation to parenting. *New Dir Child Adolesc Dev*. 2016(153):47-58.
20. Bystrova K, Widström AM, Matthiesen AS, et al. Skin-to-skin contact may reduce negative consequences of "the stress of being born": a study on temperature in newborn infants, subjected to different ward routines in St. Petersburg. *Acta Paediatr*. 2003;92(3):320-326.
21. Moore ER, Bergman N, Anderson GC, Medley N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;11:CD003519. doi:10.1002/14651858.CD003519.pub4.
22. Erlandsson K, Dsilna A, Fagerberg I, Christensson K. Skin-to-skin care with the father after cesarean birth and its effect on newborn crying and prefeeding behavior. *Birth*. 2007;34(2):105-114.
23. Cong X, Ludington-Hoe SM, Hussain N, et al. Parental oxytocin responses during skin-to-skin contact in pre-term infants. *Early Hum Dev*. 2015;91(7):401-406.
24. Yuksel B, Ital I, Balaban O, et al. Immediate breastfeeding and skin-to-skin contact during cesarean section decreases maternal oxidative stress: a prospective randomized case-controlled study. *J Matern Fetal Neonatal Med*. 2016;29(16):2691-2696.
25. Gordon I, Zagoory-Sharon O, Leckman JF, Feldman R. Oxytocin and the development of parenting in humans. *Biol Psychiatry*. 2010;68(4):377-382.
26. Takahashi Y, Tamakoshi K, Matsushima M, Kawabe T. Comparison of salivary cortisol, heart rate and oxygen saturation between early skin-to-skin contact with different initiation and duration times in healthy full-term infants. *Early Hum Dev*. 2001;87(3):151-157.
27. Schorn MN, Moore E, Spetalnick BM, Morad A. Implementing family–centered cesarean birth. *J Midwifery Womens Health*. 2015;60:682-690.
28. Kainz G, Eliasson M, von Post E. The child's father, an important person for the mother's well-being during the childbirth: hermeneutic study. *Health Care Women Int*. 2010;31:621-635.

29. Brandão S, Figueiredo B. Fathers' emotional involvement with the neonate: impact of the umbilical cord cutting experience *J Adv Nurs*. 2012;68(12):2730-2739.
30. Persson EK, Fridlund B, Kvist LJ, Kykes A. Fathers' sense of security during the first postnatal week: a qualitative interview study in Sweden. *Midwifery*. 2011;28(5):e697-e704.
31. Genesoni L, Tallandini MA. Men's psychological transition to fatherhood: an analysis of the literature, 1989–2008. *Birth*. 2009;36:305-317.
32. Spidsberg BD, Sørlie V. An expression of love: midwives' experiences in the encounter with lesbian women and their partners. *J Adv Nurs*. 2012;68:796-805.
33. Hildingsson I, Cederlöf L, Widén S. Fathers' birth experience in relation to midwifery care. *Women Birth*. 2011;24:129-136
34. Chung M, Raman G, Trikalinos T, Lau J, Ip S. Interventions in primary care to promote breastfeeding: an evidence review for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2008;149;565-582.
35. Renfrew MJ, Dyson L, McCormick F, et al. Breastfeeding promotion for infants in neonatal units: a systematic review. *Child Care Health Dev*. 2009;36:165-178.
36. Fowles ER, Horowitz JA. Clinical assessment of mothering during infancy. *J Obstet Gynecol Neonatal Nurs*. 2006;35(5):662-670.
37. McCollum JA, Ree Y, Chen YJ. Interpreting parent–infant interactions: cross-cultural lessons. *Infants Young Child*. 2000;12(4):22-33.
38. Bowman JM. Controversies in RH prophylaxis: who needs Rh immune globulin and when should it be given? *Am J Obstet Gynecol*. 1985;151:289-294.
39. Aitken SL, Tichy EM. Rh(O)D immune globulin products for prevention of alloimmunization during pregnancy. *Am J Health Syst Pharm*. 2015;72(4):267-276.
40. Crowther CA, Middleton P. Anti-D administration after childbirth for preventing Rhesus alloimmunization. *Cochrane Database Syst Rev*. 1997;2:CD000021. doi:10.1002/14651858.CD000021.
41. Centers for Disease Control and Prevention. Updated recommendations for use of tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis vaccine (Tdap) in pregnant women—Advisory Committee on Immunization Practices (ACIP), 2012. *MMWR*. 2013;62(7):131-135.
42. Eisenach JC, Pan PH, Smiley R, et al. Severity of acute pain after childbirth, but not type of delivery, predicts persistent pain and postpartum depression. *Pain* 2008;140(1):87–94.
43. American College of Obstetricians and Gynecologists. Practice advisory on codeine and tramadol in breastfeeding women 2017. Available at: https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-on-Codeine-and-Tramadol-for-Breastfeeding-Women. Accessed December 28, 2017.
44. Fahey JO. Best practices in management of postpartum pain. *J Perinat Neonat Nurs*. 2017;31(2):126-136.
45. Wuytack F, Smith V, Cleary BJ. Oral non-steroidal anti-inflammatory drugs (single dose) for perineal pain in the early postpartum period. *Cochrane Database of Systematic Reviews* 2016, Issue 7. Art. No.: CD011352. doi:10.1002/14651858.CD011352.pub2.
46. Koren G, Cairns J, Chitayat D, Gaedigk A, Leeder SJ. Pharmacogenetics of morphine poisoning in a breastfed neonate of a codeine-prescribed mother. *Lancet*. 2006;368:704.
47. U.S. Food and Drug Administration. FDA Drug Safety Communication: FDA restricts use of prescription codeine pain and cough medicines and tramadol pain medicines in children; recommends against use in breastfeeding women. April 20, 2017. Available at: https://www.fda.gov/Drugs/DrugSafety/ucm549679.htm. Accessed July 5, 2017.
48. Cascorbi I. Pharmacogenetics of cytochrome p4502D6: genetic background and clinical implications. *Eur J Clin Invest*. 2003;33(suppl 2):17-22.
49. Madadi P, Moretti M, Djokanovic N, et al. Guidelines for maternal codeine use during breastfeeding. *Can Fam Physician* 2009;55(11):1077-1078.
50. Kelly LE, Chaudhry SA, Rieder MJ, et al. A clinical tool for reducing central nervous system depression among neonates exposed to codeine through breast milk. *PLoS One*. 2013;8(7):e70073.
51. Sutton CD, Carvalho B. Optimal pain management after cesarean delivery. *Anesthesiol Clin*. 2017;35(1):107-124.
52. Neville MC, Morton J, Umemura S. Lactogenesis: the transition from pregnancy to lactation. *Pediatr Clin North Am*. 2001;48:35.
53. Mangesi L, Zakarija-Grkovic I. Treatments for breast engorgement during lactation. *Cochrane Database Syst Rev*. 2016;6:CD006946. doi:10.1002/14651858.CD006946.pub3.
54. Roberts KL, Reiter M, Schuster D. A comparison of chilled and room temperature cabbage leaves in treating breast engorgement. *J Hum Lact*. 1995;11(3):191-194.
55. Vieira F, Mota DDCF, Castral TC, Guimarães JV, Salge AKM, Bachion MM. Effects of anhydrous lanolin versus breast milk combined with a breast shell for the treatment of nipple trauma and pain during breastfeeding: a randomized clinical trial. *J Midwifery Womens Health*. 2017. [Epub ahead of print]. doi:10.1111/jmwh.12644.
56. Dennis CL, Jackson K, Watson J. Interventions for treating painful nipples among breastfeeding women. *Cochrane Database Syst Rev*. 2014;12:CD007366. doi:10.1002/14651858.CD007366.pub2.

57. Hedayati H, Parsons J, Crowther CA. Topically applied anaesthetics for treating perineal pain after childbirth. *Cochrane Database Syst Rev.* 2005;2:CD004223. doi:10.1002/14651858.CD004223.pub2.
58. East CE, Begg L, Henshall NE, Marchant PR, Wallace K. Local cooling for relieving pain from perineal trauma sustained during childbirth. *Cochrane Database Syst Rev.* 2012;5:CD006304. doi:10.1002/14651858.CD006304.pub3.
59. Mangesi L, Hofmeyr GJ. Early compared with delayed oral fluids and food after caesarean section. *Cochrane Database Syst Rev.* 2002;3:CD003516. doi:10.1002/14651858.CD003516.
60. Bateman BT, Cole NM, Maeda A, et al. Patterns of opioid prescription and use after cesarean delivery. Obstet Gynecol. 2017;130(1):29-35.
61. Kocaöz S, Erog˘lu K, Sivaslıog˘lu AA. Role of pelvic floor muscle exercises in the prevention of stress urinary incontinenc during pregnancy and the postpartum period. *Gynecol Obstet Invest*. 2012;75:34-40.
62. Citak N, Cam C, Arslan H, et al. Postpartum sexual function of women and the effects of early pelvic floor muscle exercises. *Acta Obstet Gynecol Scand*. 2010;89(6):817-822.
63. Leeman LM, Rogers RG. Sex after childbirth: postpartum sexual function. *Obstet Gynecol*. 2012;119(3):647-655.
64. Rowland M, Foxcroft L, Hopman WM, Patel R. Breastfeeding and sexuality immediately post partum. *Can Fam Physician*. 2005;51:1366-1367.
65. Gray RH, Campbell OM, Apelo R, et al. Risk of ovulation during lactation. *Lancet*. 1990;335:25.
66. Chen BA, Reeves MF, Hayes JL, Hohmann HL, Perriera LK, Creinin MD. Postplacental or delayed insertion of the levonorgestrel intrauterine device after vaginal delivery: a randomized controlled trial. *Obstet Gynecol*. 2010;116(5):1079-1087.
67. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR Recomm Rep*. 2016;65(RR-3):1-104.
68. Gould CV, Umscheid CA, Agarwal RK, Kuntz G, Pegues DA. Guideline for prevention of catheter-associated urinary trac infections 2009. *Infect Control Hosp Epidemiol*. 2010;31(4):319-326.
69. American College of Obstetricians and Gynecologists. Committee Opinion No.666: optimizing postpartum care. *Obstet Gynecol*. 2016;127:e187-e192.
70. Thung FS, Norwitz ER. Postpartum care: we can and should do better. *Am J Obstet Gynecol*. 2010;202(1):1-4.
71. Walker LO, Murphey CL, Nichols F. The broken thread of health promotion and disease prevention for women during the postpartum period. *J Perinat Educ*. 2015;24(2):81-92.
72. Burgio KL, Zyczynski H, Locher JL, Richter HE, Redden DT, Wright KC. Urinary incontinence in the 12-month postpartum period. *Obstet Gynecol*. 2003;102:1291-1298.
73. Declercq ER, Sakala C, Corry MP, Applebaum S, Herrlich A. Major survey findings of Listening to Mothers III: New Mothers Speak Out: report of national surveys of women's childbearing experiences, conducted October–December 2012 and January–April 2013. *J Perinat Educ* 2014;23:17-24.
74. Haran C, van Driel M, Mitchell BL, Brodribb WE. Clinical guidelines for postpartum women and infants in primary care: a systematic review. *BMC Pregn Childbirth* 2014;14:51.
75. Fahey JO, Shenassa E. Understanding and meeting the needs of women in the postpartum period: the Perinatal Maternal Health Promotion Model. *J Midwifery Womens Health*. 2013;58(6):613-621.
76. Borders N. After the afterbirth: a critical review of postpartum health relative to method of delivery. *J Midwifery Women Health*. 2006;51(4):242-248.
77. Carter EB, Stuart JJ, Farland LV, et al. Pregnancy complications as markers for subsequent maternal cardiovascular disease: validation of a maternal recall questionnaire. *J Womens Health*. 2015;24(9):702-712.
78. Enkhmaa D, Wall D, Mehta PK, et al. Preeclampsia and vascular function: a window to future cardiovascular disease risk *J Womens Health*. 2016;25(3):284-291.
79. Rich-Edwards JW, Klungsoyr K, Wilcox AJ, Skjaerven R. Duration of pregnancy, even at term, predicts long-term risk of coronary heart disease and stroke mortality in women: a population-based study. *Am J Obstet Gynecol*. 2015;213(4):518.e1-8.
80. Martinez NG, Niznik CM, Yee LM. Optimizing postpartum care for the patient with gestational diabetes mellitus. *Am J Obstet Gynecol*. April 26, 2017. [Epub ahead of print]. doi:10.1016/j.ajog.2017.04.033.
81. Guille C, Newman R, Fryml LD, Lifton CK, Epperson CN. Management of postpartum depression. *J Midwifery Womens Health*. 2013;58(6):643-653.
82. American College of Nurse-Midwives. Depression in women: position statement. 2003; updated 2013. Available at: http://midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000061/Depression%20in%20Women Accessed July 14, 2017.
83. U.S. Department of Health and Human Services. 2008 physical activity guidelines for Americans. Be active, happy and healthy. 2008. Available at: http://www.health.gov/paguidelines/pdf/paguide.pdf. Accessed June 25, 2013.
84. Daley AJ, Thomas A, Cooper H, et al. Maternal exercise and growth in breastfed infants: a meta-analysis of randomized controlled trials. *Pediatrics*. 2012;130(1):108-114.
85. Weaver K. Review: dietary restriction, with or without aerobic exercise, promotes weight loss in postpartum women. *Evid*

Based Nurs. 2008;11(1):14.
86. Mercer RT. Becoming a mother versus maternal role attainment. *J Nurs Scholarsh*. 2004;36(3):226-232.
87. Koniak-Griffin D, Logsdon MC, Hines-Martin V, Turner CC. Contemporary mothering in a diverse society. *J Obstet Gynecol Neonatal Nurs*. 2006;35:671-678.
88. Javadifar N, Majlesi F, Nikbakht A, Nedjat S, Montazeri A. Journey to motherhood in the first year after child birth. *J Fan Reprod Health*. 2016;10(3):146-153.
89. Slomian J, Emonts P, Vigneron L, et al. Identifying maternal needs following childbirth: a qualitative study among mothers, fathers and professionals. *BMC Pregn Childbirth*. 2017;17:213.
90. Beck CT, Gable RK, Sakala C, Declercq ER. Posttraumatic stress disorder in new mothers: results from a two-stage U.S national survey. *Birth*. 2011;38(3):216-227.
91. Bastos MH, Furuta M, Small R, McKenzie-McHarg K, Bick D. Debriefing interventions for the prevention of psychological trauma in women following childbirth. *Cochrane Database Syst Rev*. 2015;4:CD007194. doi:10.1002/14651858.CD007194.pub2.
92. Meades R, Pond C, Ayers S, Warren F. Postnatal debriefing: have we thrown the baby out with the bath water? *Behav Res Ther*. 2011;49(5):367-372.
93. Tran T, Reeder A, Funke L, Richmond N. Association between smoking cessation interventions during prenatal care and postpartum relapse: results from 2004 to 2008 multi-state PRAMS data. *Matern Child Health J*. 2013;17(7):1269-1276.
94. Jiménez-Muro A, Nerín I, Samper P, et al. A proactive smoking cessation intervention in postpartum women. *Midwifery*. 2012;29(3):240-245.
95. Brandon TH, Simmons VN, Meade CD, et al. Self-help booklets for preventing postpartum smoking relapse: a randomized trial. *Am J Public Health*. 2012;102(11):2109-2115.
96. Ruedinger E, Cox JE. Adolescent childbearing: consequences and interventions. *Curr Opin Pediatr*. 2012;24(4):446-452.
97. Lewis LN, Doherty DA, Hickey M, Skinner SR. Implanon as a contraceptive choice for teenage mothers: a comparison o contraceptive choices, acceptability and repeat pregnancy. *Contraception*. 2010;81(5):421-426.
98. MacDonald T, Noel-Weiss J, West D, et al. Transmasculine individuals' experiences with lactation, chestfeeding, and gender identity: a qualitative study. *BMC Pregn Childbirth*. 2016;16:106.
99. Glaser RL, Newman M, Parsons M, Zava D, Glaser-Garbrick D. Safety of maternal testosterone therapy during breast feeding. *Int J Pharm Compd*. 2009;13(4):314-317.
100. Light AD, Zimbrunes SE, Gomez-Lobo V. Reproductive and Obstetrical care for transgender patients. *Curr Obstet Gynecol Rep*. 2017;6:149-155.
101. Lefkowitz DS, Baxt C, Evans JR. Prevalence and correlates of posttraumatic stress and postpartum depression in parents of infants in the neonatal intensive care unit (NICU). *J Clin Psychol Med Settings*. 2010;17(3):230-237.
102. Kantrowitz-Gordon I. Expanded care for women and families after preterm birth. *J Midwifery Womens Health*. 2013;58(2):158-166.
103. Nyqvist K, Anderson G, Bergman N, et al. Towards universal kangaroo mother care: recommendations and report from the First European conference and Seventh International Workshop on Kangaroo Mother Care. *Acta Paediatr*. 2010;99(6):820-826.
104. Mott SL, Schiller CE, Richards JG, O'Hara MW, Stuart S. Depression and anxiety among postpartum and adoptive mothers. *Arch Womens Mental Health*. 2011;14(4):335-343.
105. Foli KJ. Nursing care of the adoption triad. *Perspect Psychiatr Care*. 2012;48(4):208-217
106. Clarke JG, Adashi EY. Perinatal care for incarcerated patients: a 25-year-old woman pregnant in jail. *JAMA*. 2011;305(9):923-929.
107. Committee on Health Care for Underserved Women. ACOG Committee Opinion No. 511: health care for pregnant and postpartum incarcerated women and adolescent females. *Obstet Gynecol*. 2011;118(5):1198-1202.

APPENDIX

33A

Management of the Fourth Stage of Labor Following Spontaneous Vaginal Birth

IRA KANTROWITZ-GORDON

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Physical Examination and Management

Vital Signs

Check maternal vital signs every 5 to 15 minutes. If the woman's condition or bleeding pattern changes, assess vital signs more frequently as indicated.

Bleeding

The majority of postpartum hemorrhages occur during the fourth stage of labor. Evaluate vaginal bleeding every 5 to 15 minutes immediately after birth of the infant or more frequently as indicated by changes in the woman's condition and bleeding pattern.

Uterus

Evaluate the uterus for position and tone every 5 to 15 minutes if there is no excessive bleeding that suggests more frequent evaluation.

- *Position*. Assess the uterus by abdominal palpation; it is usually midline in the middle of the abdomen, between the symphysis pubis and the umbilicus. If the uterus is above the umbilicus or significantly deviated to one side of the umbilicus, blood clots may be filling in the uterus or the uterus may be displaced by a full bladder. Evacuation of the uterus or emptying of the bladder should be considered.
- *Tone*. The uterus should be firm to the touch and immediately identifiable. A soft, boggy uterus that is difficult to palpate is not well contracted. Gentle massage of the uterus through the abdomen can promote uterine contraction and should be performed if the

uterus is not firm. Assess for postpartum hemorrhage and presence of blood or clots in the uterus if the uterus does not become firm following abdominal massage.

Bladder

Evaluate the bladder immediately after the third stage and at least 30 minutes after birth for distention and urinary retention. A distended bladder can cause increased uterine bleeding, since the uterus is unable to contract when the bladder is full and displacing it. A woman without regional anesthesia usually can void spontaneously. If she has regional anesthesia, she may need urinary catheterization to empty the bladder.

Perineum

Inspect the vagina and perineum for lacerations, bruising, or early hematoma formation immediately after the birth and after the conclusion of the third stage of labor. Inspect the cervix if indicated. Edema or bruising at the introitus or throughout the perineal area should be noted and documented.

Additional Assessments and Care Practices

- Count needles and gauzes used in the birth to ensure that no gauze is inadvertently left in the vagina and all sharps are disposed of safely. Remove any sharps from the sterile field.
- Clean the woman and bed clothes as needed.
- A perineal pad or ice pack may be placed against the perineum to reduce edema and provide pain relief. An ice pack may be placed next to the perineum intermittently for the first 24 hours after birth.
- Encourage skin-to-skin placement of the neonate following birth, which allows women to notice when their neonates are beginning to root.
- Help the woman position herself to promote infant latch if she plans to breastfeed.
- Assess for comfort. A short period of tremor not associated with infection can be common in the fourth stage of labor and can be relieved with warm blankets, reassurance, and relaxation techniques.
- Assess intake and encourage intake of oral fluids.
- Assess family dynamics.

APPENDIX

33B

History and Physical Examination During the Early Postpartum Period

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Physical examinations during the immediate postpartum period, like any examination, begin with performing a personal greeting, ascertaining if the moment is an appropriate time for the examination, and adapting the environment to provide comfort and privacy. Throughout the examination, the midwife can clarify questions (**Table 33B-1**), integrate teaching, and provide anticipatory guidance.

Table 33B-1	Interval Postpartum History for 6-Week Postpartum Visit
Healthcare encounters	Calls to the midwife or the physician Visits to the hospital emergency department Hospital admissions
Sexual activity	Resumption of sexual activity and number of times Woman's/partner's enjoyment and satisfaction Dyspareunia
Contraception	Contraceptive used or desired Past contraceptive experience
Exercise	Exercise and activities since childbirth Pelvic floor exercises
Vaginal bleeding	Resolution of lochia Episodes of excessive bleeding Resumption of menses
Infant	Attendance at well-child visits Growth and well-being of the infant
Mental health	Signs or symptoms of postpartum mood disorder

Postpartum Physical Examination

Table 33B-2 summarizes the procedure for a postpartum physical examination. The pelvic examination should begin with a visual inspection of the perineum for healing. The bimanual examination will include assessment for vaginal tone by having the woman do a Kegel exercise with one of the examiner's fingers inserted in the vagina. Vaginal walls will be inspected for decreased rugae and atrophy.

Table 33B-2 **Postpartum Physical Examination: Procedure and Expected Findings**

	Procedure	Expected Findings	Deviations from Normal
General	Observe general affect, mobility, and signs of pain or splinting.	Tired, ambulatory without assistance, repeating birth events, usually happy	S: Manner suggesting pain, discomfort, dissatisfaction, unhappiness O: Inability to ambulate without assistance; sad or teary
Vital signs	Assess temperature, blood pressure, pulse, and respiratory rate.	Temperature: normal or low-grade elevation Blood pressure: returns to normal by 24 hours Pulse: 65–80 bpm at rest Respirations: 12–16/min at rest	Temperature: > 38°C (100.4°F) Blood pressure: higher than 140/90 mm Hg or lower than 85/60 mm Hg Pulse: higher than 100 bpm
Neurologic		Oriented to person, place, and time Not in apparent pain Normal reflexes	Disoriented, headache—sudden onset or severe, and/or hyperreflexia Excessively sedated or somnolent, unable to easily awaken Excessive or unexplained pain or restlessness
Thyroid		Normal size and consistency or not palpable	The thyroid gland may be enlarged, indicating postpartum thyroiditis.
Cardiovascular	The examination of the heart and lungs during the early puerperium is performed in the same manner as when the woman is not pregnant or postpartum. Auscultate the heart (rate, rhythm, cardiac sounds). • Systolic ejection murmur persists past the first week of puerperium in 20% of women. • Heart rate decreases to the nonpregnant level within 10 days postpartum.	S: No chest pain. May feel heartbeat increasing with minimal activity O: Regular rate and rhythm	S: Chest pain, palpitations O: Tachycardia
Lungs/ pulmonary	Auscultate the lungs, and percuss as appropriate (the midwife may assist the woman to a sitting position for evaluation of the inferior lobes). • Respiratory volume increases to the nonpregnant rate within the first postpartum week. • Diminished breath sounds or crackles in lung base suggest pulmonary edema or pneumonia.	S: No shortness of breath or difficulty breathing O: Clear to auscultation	S: Shortness of breath O: Adventitious sounds

Breasts[a]	Observe breasts for symmetry. • Palpate to assess engorgement if present. • Palpate axilla to assess for accessory mammary tissue. Inspect the nipple epithelium for erythema, bruising, petechial, and cracks or bleeding. • Observe the integrity of the areola and inspect for blisters or cracks. • Roll or gently squeeze the nipple to assess erectility; express colostrum. • If the woman is not breastfeeding, verify she has a bra that provides comfortable support. • If the woman is breastfeeding, verify adequate bra support. Note: The immediate postpartum period is not a time for a reliable clinical breast examination for cancer screening.	S: Neonate is nursing frequently with a good latch Nipples sore, but not painful Colostrum for 3–5 days with breast fullness before milk is seen O: Soft for 1–2 days; some fullness beginning days 3–5 Nipples intact and erect when stimulated Soft swelling in the axilla may be evidence of accessory mammary tissue that can cause discomfort, but is otherwise benign	S: Neonate is not latching well or frequently Painful nipples No breast filling by days 4–5 O: Nipples: flat, inverted, cracked, bruised, blistered, or bleeding Breasts: erythema, engorged
Abdomen/ gastrointestinal	Examination of the abdomen during the puerperium begins with the uterus, which will be the largest palpable organ. • Position the woman in supine position. • Palpate the fundus abdominally and verify its location. ◆ Immediately after birth, the fundus is several centimeters below the umbilicus. ◆ On the first day postpartum, the fundus slightly relaxes and rises to the level of the umbilicus. ◆ The height of the fundus gradually decreases until it is not palpable abdominally, which usually occurs by approximately 2 weeks postpartum. ◆ Deviations may exist if the woman is at extremes of height or weight. • Palpate the suprapubic area to rule out distended bladder. ◆ There is a high index of suspicion that the bladder is full if the uterus is displaced upward and to one side (generally the right side).	S: Eating and drinking without difficulty. Bowel movements usually return after 2–3 days with normal bowel patterns by 2 weeks. Abdomen: uterine contractions/cramping particularly with nursing O: Abdomen: nontender, nondistended; bowel sounds present; muscle tone is decreased; distension in the first 2–3 days is common, diastasis recti is an expected finding Uterus: fundus firm, nontender, midline, height normal for postpartum day Surgical scar if present is approximated and dry without evidence of infection	S: Nausea or vomiting, abdominal pain, constipation or diarrhea O: Abdomen: distended, tender, or no bowel sounds Uterus: increasing fundal height, deviation from midline, or tender Surgical scar not approximated, surrounded by an area of redness, or discharge present

	• If post cesarean birth, inspect the incision using REEDA for later documentation. • Assess the CVAT. • Request the woman to lift her head onto her chest while the midwife simultaneously palpates the center of the abdomen to assess the degree of separation of the rectus abdominis (rectus diastasis). Determine the size of the separation in centimeters or fingerbreadths for later documentation.		
Urinary	Assess for urinary retention, incontinence, and symptoms of infection.	S: Voiding spontaneously, diuresis, and natriuresis normal, especially between days 2 and 5 Mild external burning, some retention and/or incontinence, and lack of sensation or urge to void are common in the first 2 days postpartum if the woman had epidural analgesia, an operative vaginal birth, or extensive perineal trauma, but should resolve rapidly Tender or sore at area of repair O: Bladder: nondistended—not palpable	S: Pelvic pressure or pain increasing Persistent retention or incontinence O: Bladder: distended Costovertebral angle tenderness
Perineum	Because it is likely that the midwife will come in contact with bodily fluids, most clinicians defer examination of the perineum until after assessment of the woman's extremities. • Obtain adequate visualization (a flashlight, penlight, or flashlight app works well) with good lighting.	Perineum: mild erythema, edema, or bruising may be present Repair: approximated without drainage Lochia: amount decreasing, color, odor	Perineum: increasing tenderness, erythema, edema, bruising Laceration repair: separation of repair, suppuration, or excessive edema, signs of hematoma Lochia: malodor, clots, soaked or overflowing pads

	• Ask the woman to lie on her side: ◆ Choose the side based on the woman's comfort and the layout of the room. ◆ Lateral view and gentle elevation of the upper buttocks allow visualization of the posterior aspect of the repair and the anus. • Observe the woman for discomfort throughout the examination. • Consider use of a large mirror to show the woman her perineum if she desires. • Change to a new pair of nonsterile gloves. • If laceration or episiotomy is present, inspect the full length of the suture line using the REEDA scale for later documentation.[b] ◆ Mild edema is normal in the first 2–3 days postpartum. ◆ If signs of a hematoma are present, use a gloved hand to palpate gently with thumb and forefinger to distinguish between normal edema and hematoma. Signs of hematoma include unilateral labial swelling, black/blueish area of erythema, report of vaginal or rectal pressure, or sudden onset of severe pain. • Evaluate lochia on a sanitary pad, observing the color and extent of saturation and noting the length of time since the pad was changed. ◆ If needed, discard the soiled pad and provide a new one to the woman. • Discard used gloves, and perform hand hygiene.		
Anus	Assess for presence or absence of hemorrhoids.	Normal in appearance Hemorrhoids: pink	Hemorrhoids: deep blue/purple

Lower extremities	Observe for unilateral calf tenderness, erythema, or swelling that may suggest deep vein thrombosis.	S: No pain Generalized muscle soreness O: Edema is common Reflexes normal	S: Pain, aching, or swelling, particularly in one leg O: Deep calf tenderness, one calf larger in the transverse diameter than the other, heat, varicosities that are erythematous and/or painful on palpation
Integument		Normal color No immediate changes. Hyperpigmentation may be retained, but will fade with time. Striae, spider nevi, capillary hemangiomas, varicosities, and skin tags regress but may not completely disappear.	
Laboratory values		Hemoglobin: decreases slightly in the first 24 hours after birth, then plateaus for 4 days, followed by a slow rise to day 14 WBC count: falls to 6000–10,000/mm^3, then returns to normal values by 6 days	A 500-mL blood loss will usually result in approximately a 1-g reduction in hemoglobin or 3% reduction in hematocrit. These values return to normal by 4–6 weeks postpartum.

Abbreviations: CVAT, costovertebral angle for tenderness; O, objective; REEDA, Redness, Ecchymosis, Edema, Discharge/Drainage, and Approximation; S, subjective; WBC, white blood cell.

[a] During the phases of lactogenesis, the breast consistency may be soft, filling (i.e., tense, increasing firmness, slightly enlarged), or engorged (i.e., enlarged, hard, reddened, shiny; skin temperature elevated; painful, dilated veins).

[b] The REEDA scale provides a mnemonic for documentation of healing lacerations or incisions. This midwife-developed instrument was created to provide a standard description of perineal healing, but today it is used widely for documentation of any healing incision. REEDA refers to Redness, Ecchymosis, Edema, Discharge/Drainage, and Approximation. Although a scoring system has been used with this scale in the past, today most midwives use descriptors for each component.[1]

[c] Evaluation of the extremities has traditionally included Homan's sign to assess for deep vein thrombosis (DVT). This test is not diagnostic and could theoretically loosen a thrombus that might be present. Therefore, Homan's sign is not recommended today. DVT is increased 60-fold in the first 3 months postpartum compared with women who are not pregnant or postpartum.[2] Other signs that are more predictive of deep vein thrombosis than a positive Homan's sign include unilateral calf tenderness, erythema, and swelling.

A speculum examination is not necessary unless a Pap test is indicated. Due to the discomfort of the speculum examination, the midwife should wait for complete healing of birth lacerations or episiotomies before inserting a speculum. While minor lacerations may be completely healed, more extensive repairs can take months to fully heal and for pain to resolve. If an intrauterine device was inserted immediately postpartum, then the midwife should inspect for the presence of strings during the speculum or bimanual examination because spontaneous expulsion is more likely.

Before starting the examination:

- Perform hand hygiene, preferably in the woman's sight, and don nonsterile/clean gloves.
- Assist the woman in assuming a comfortable supine or dorsal position.

Laboratory Tests

Laboratory assessment may be required based on history, as indicated in **Table 33B-3**. Under current cervical cytology screening guidelines, women who are at least 21 years old and who do not have a history of abnormal Pap tests need a Pap test every 3 years. Pelvic ultrasound may be indicated for women with complex cysts or other abnormalities identified by ultrasound during pregnancy. Since the postpartum visit marks the end of the childbearing year as well as the continuation of well woman health promotion across the lifespan, recommended screening and counseling for health maintenance, as described in the *Health Promotion Across the Lifespan* chapter, should also be provided as indicated.

Table 33B-3 Postpartum Laboratory Tests

Laboratory Tests	Indications	Timing	Normal Range
Hemoglobin (Hgb) or hematocrit (Hct)	Prenatal anemia, postpartum hemorrhage, cesarean birth	At least 4 weeks postpartum unless symptomatic of acute anemia	Hgb ≥ 12 g/dL Hct ≥ 36%
Thyroid-stimulating hormone (TSH)	Current or recent thyroid disease, history of postpartum thyroiditis, enlarged thyroid, symptoms of postpartum depression	6-week postpartum visit, 3 months and 6 months postpartum	TSH: 0.4–5.0 mU/L
Cervical cytology	Age 21 or older and history of unresolved abnormal cytology or human papillomavirus tests, or 3 years since last test	After perineal and vulvar healing is complete	Negative for intraepithelial lesion or malignancy
Sexually transmitted infection screening (gonorrhea, chlamydia, syphilis, human immunodeficiency virus)	All sexually active women younger than 25 years and women age 25 years and older with a history of sexually transmitted infections, unprotected sexual intercourse, multiple sexual partners in past 12 months	Annually and within 3 months of past infection	Negative
2-hour 75-g glucose challenge	Gestational diabetes Also screen women age 40 years and older with a BMI of 30 or higher	6–12 weeks postpartum and annually thereafter	Fasting glucose < 100 mg/dL 2-hour glucose < 140 mg/dL
Additional Screening Recommended for Health Maintenance[3]			
Alcohol misuse	Screen all women using a validated screening tool and provide counseling and education as described in the *Health Promotion Across the Lifespan* chapter.		
Depression	Screen all women using a validated screening tool and provide management as described in the *Health Promotion Across the Lifespan* and *Postpartum Complications* chapters.		
Intimate partner violence	Screen all women using a validated screening tool as described in the *Health Promotion Across the Lifespan* chapter.		
Lipid disorders	Screen women age 40 years and older using nonfasting or fasting total cholesterol, LDL-C, and HDL-C.		
Tobacco Use	Screen all women using a validated screening tool and provide counseling and education as described in the *Health Promotion Across the Lifespan* chapter.		

References

1. Davidson N. REEDA: evaluating postpartum healing. *J Nurse Midwifery.* 1974;19(2):6-8.
2. Pomp ER, Lenselink AM, Rosendaal FR, Doggen CJ. Pregnancy, the postpartum period and prothrombotic defects: risk of venous thrombosis in the MEGA study. *J Thromb Haemost.* 2008;6(4):632-637.
3. U.S. Preventive Services Task Force. Published recommendations. Available at: https://www.uspreventiveservicestaskforce.org/BrowseRec/Index/browse-recommendations. Accessed July 4, 2017

CHAPTER

34

Postpartum Complications

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Introduction

Most women experience an uneventful postpartum recovery (puerperium) while discovering the subtle but extraordinary capabilities of the newborn. Occasionally, however, complications arise that must be recognized and managed promptly.

Approximately 70% of pregnancy-related deaths occur on the day of birth or in early postpartum period.[1] Historically, infection and hemorrhage were the leading causes of postpartum mortality. With the advent of antibiotics, the most frequent causes of death have changed, such that throughout the twentieth century, hemorrhage, hypertension, and thromboembolism were the three leading causes of maternal mortality and morbidity.[1] Today, however, pregnant women are more likely to be older and have chronic health conditions compared to their counterparts in previous generations. The incidence of obesity and cesarean sections has also increased. In the United States, cardiovascular conditions, cardiomyopathy, infection, and hemorrhage are now the four leading causes of pregnancy-related mortality.[1] Approximately 50% of deaths related to pregnancy are preventable.[2] Therefore, it is critical that midwives recognize signs and symptoms of severe maternal morbidity in the postpartum period.[3]

This chapter reviews complications that may occur postpartum after the fourth stage of labor and that may affect the reproductive tract organs. Urinary retention—an early postpartum complication that rarely causes significant morbidity—is presented first. Management of uncomplicated urinary retention is within the midwifery scope of practice. A review of complications associated with severe maternal morbidity or mortality, such as bleeding (e.g., secondary hemorrhage and hematoma), wound complications (seroma, hematoma, dehiscence, and infection), cardiomyopathy, preeclampsia/eclampsia, and thromboembolism, follows. Conditions less often seen in clinical practice, such as postpartum thyroid disorders, are presented last. Immediate postpartum hemorrhage, which occurs during the third stage of labor or in the first hours after birth, is reviewed in the *Third Stage of Labor* chapter. Postpartum mood and anxiety disorders are discussed in the *Mental Health Conditions* chapter, and breastfeeding complications such as mastitis are reviewed in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

Urinary Retention

Postpartum urinary retention can present as the inability to void at all or as covert postpartum urinary retention wherein the woman can void but has a residual bladder volume of more than 150 mL. Urinary retention occurs in approximately 10% of women following vaginal birth, and covert postpartum urinary retention may occur in up to 47% of women.[4,5] Risk factors include epidural analgesia, instrumental delivery, episiotomy, high birth weight, and primiparity.[5,6] Potential causes of urinary retention include slow recovery of sensation after epidural anesthesia, excessive vulvar edema that compresses the urethra, trauma and edema of the bladder neck, and injured bladder innervation. Although long-term adverse effects are rare, urinary retention increases the risk of urinary tract infection and bladder dysfunction in the short term.

Postpartum urinary retention should be suspected when a woman has not urinated in 6 to 8 hours, there is palpable bladder above the symphysis pubis on abdominal examination, the uterine fundus is displaced upward and to the side of the midline, or a full bladder is identified on ultrasound.

Initially, noninvasive care should be implemented to help the woman successfully urinate. Such measures include maintaining privacy, adequate analgesia, warm-water immersion, placement of a few drops of peppermint oil in the toilet water or bedpan, and the sound of running water. Although little research evidence supports the effectiveness of these strategies, they are not harmful. However, if these measures are not immediately successful, urinary catheterization should promptly be used to effectively empty the bladder. Urinary stasis is a risk factor for urinary tract infection and needs to be resolved soon once it is diagnosed.

Urinary retention is usually diagnosed clinically on the basis of history of inability to void and/or abdominal examination to palpate a full bladder. Women who have a palpable full bladder but who are able to void spontaneously should have the amount of voided urine measured; the woman should then be reassessed immediately after voiding to rule out covert postpartum urinary retention. Ultrasound or post-void catheterization can be used to diagnose covert postpartum urinary retention. The treatment of covert postpartum urinary retention is catheterization.

In general, intermittent catheterization is associated with less risk of a catheter-associated urinary tract infection than is placement of an indwelling catheter for individuals with bladder-emptying dysfunction. If an indwelling catheter is used, it should be maintained for the shortest period possible.[7] In this situation, a common approach is to leave the indwelling catheter in place overnight, to be removed in the morning. If the woman is unable to urinate the next day, it may be necessary to leave the catheter in place for a longer period.

Although 150 mL is commonly used as the criterion for diagnosing covert urinary retention, there is no evidence that this cut-off value reflects an increased risk for adverse outcomes. Mulder et al. found that following vaginal birth (n = 439), the mean post-residual void was 140 mL, and that the majority of women with a post-residual volume greater than 150 mL were able to empty the bladder spontaneously by the fourth postpartum day.[5,8] Therefore, reliance on the 150 mL criterion may result in a higher rate of indwelling catheter use than is necessary. Given the lack of clear evidence for a post-residual amount that is associated with adverse

effects, the choice of intermittent versus indwelling catheterization should be individualized.

Figure 34-1 provides a sample algorithm for management of a woman who has postpartum urinary retention. Institutional guidelines will dictate the cut-off values for each step in this algorithm. If a woman is able to successfully void after catheterization, she should be assessed for adequate urinary output for the next two or three occasions to evaluate whether she is still experiencing retention to some degree.

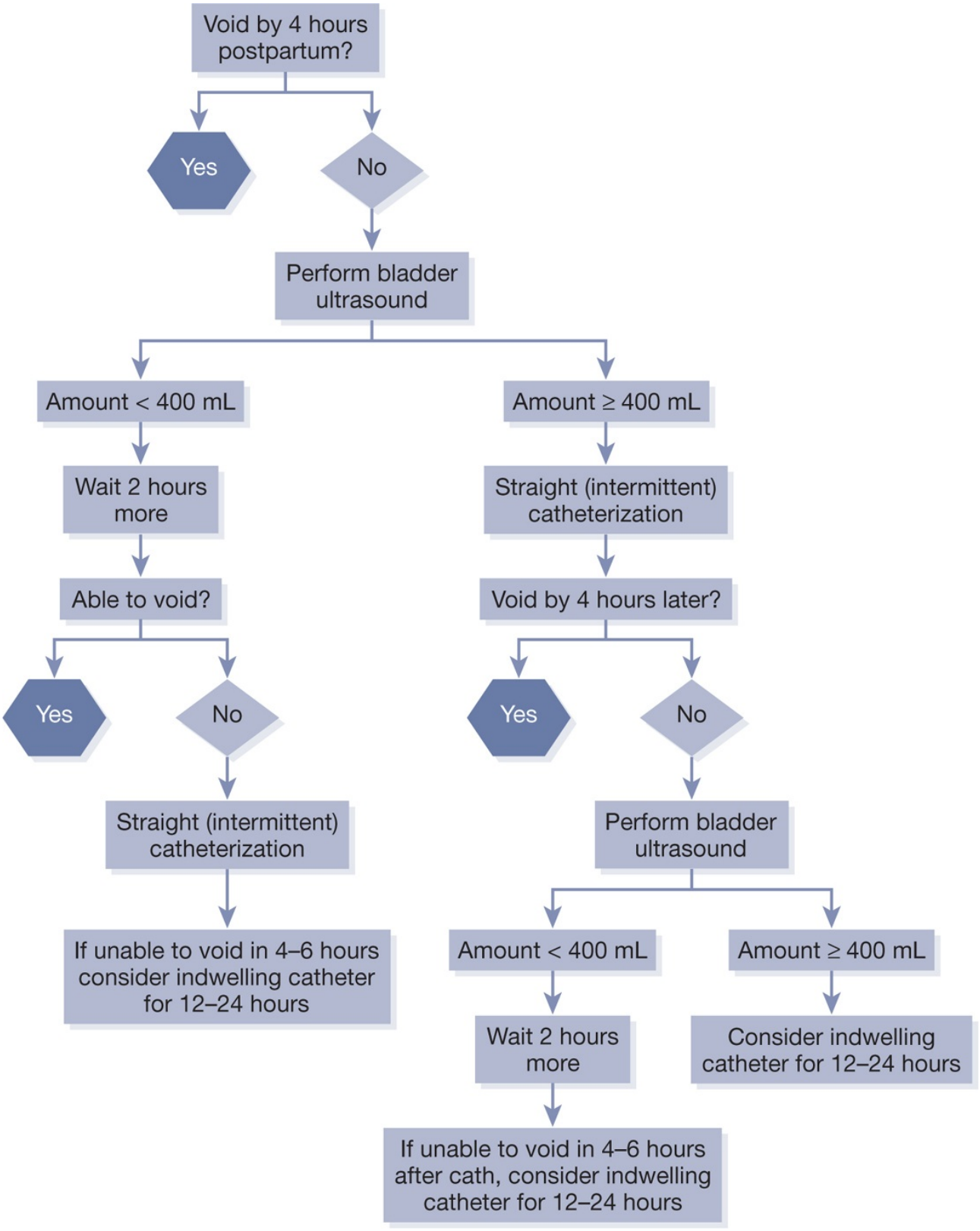


Figure 34-1 Sample algorithm for assessment of voiding and management of urinary retention following vaginal birth.

Puerperal Fever and Puerperal Infection

Puerperal pyrexia or fever is defined as a temperature of 38.0°C (100.4°F) or higher, occurring on any 2 of the first 10 days postpartum excluding the first 24 hours. The first postpartum 24-hour period is excluded because a mild transient temperature elevation can develop as a physiologic reaction to childbirth during this time period. Similarly, a brief temperature elevation of less than 38.0°C occasionally occurs with the onset of milk production 3 to 5 days after birth.

The three most common differential diagnoses associated with postpartum temperature elevations of 38.0°C or more are endometritis, wound infections (e.g., surgical site infection), and urinary tract infections.[9] These three infections account for approximately 31% of hospital readmissions following childbirth in the United States.[10] Less commonly, postpartum fever may be caused by septic pelvic thrombophlebitis, transfusion reactions, or drug reactions. Despite the availability of effective antibiotic therapies, puerperal infections are among the most frequent causes of maternal mortality globally.

Puerperal infection is defined as any infection in the genital tract following childbirth, miscarriage, or pregnancy termination. Organisms that cause puerperal infections include those that normally exist in the lower genital tract or in the bowel, bacteria that are found in the nasopharynx, bacteria that are present on the hands of attending personnel, and those that are present in the air and dust of the environment. Organisms commonly associated with puerperal infection include gram-positive aerobes, such as *Streptococcus* species (A, B, and D), *Enterococcus*, and *Staphylococcus aureus* and *epidermis*; gram-negative aerobes, such as *Escherichia coli*, *Klebsiella*, *Enterobacter*, and *Proteus* species; and gram-variable organisms, such as *Gardnerella vaginalis*. A fever of 39.0°C or higher that develops within 24 hours after a cesarean section may indicate a group A *Streptococcus* infection. Anaerobes associated with puerperal infection include *Peptostreptococcus*, *Peptococcus*, *Bacteroides* species, *Clostridium*, and *Fusobacterium*. Other organisms implicated in puerperal infection include *Mycoplasma*, *Chlamydia*, and *Neisseria gonorrhoeae*.

In addition to the elevated temperature, signs and symptoms of puerperal infection include general malaise, pain, chills, increased pulse, abdominal pain, and possibly malodorous lochia. Because symptoms of puerperal infection are often generalized, the differential diagnosis includes appendicitis, pyelonephritis, and pneumonia. It is important to consider the possibility of extended infections. These infections originate from a localized infection and extend via the path of venous circulation or the lymphatics to produce bacterial infection in more distant sites. Extended sites of puerperal infection include pelvic cellulitis, salpingitis, oophoritis, peritonitis, pelvic and femoral thrombophlebitis, and sepsis.

Physician consultation and/or referral is indicated for women who require evaluation for any postpartum infection.

Endometritis

Uterine infection, which is variously referred to as endometritis or endomyometritis, may occur anywhere within the uterus (i.e., myometrium, endometrium, or both). Endometritis

typically develops within 2 to 4 days postpartum or as late as 2 to 6 weeks postpartum, and can be a mild or a severe infection. This type of infection occurs in 1% to 2% of women following a vaginal birth, approximately 27% in women who have a cesarean birth without administration of prophylactic antibiotics, and 11% in women who are given prophylactic antibiotics before the cesarean section is performed.[11,12] **Table 34-1** lists risk factors for endometritis, with cesarean section being the most important factor putting the woman at risk for this infection.[13] Adverse outcomes associated with endometritis (if not treated) include salpingitis, peritonitis, necrotizing fasciitis, sepsis, septic thrombophlebitis, and death.

Table 34-1 Risk Factors for Postpartum Endometritis

Bacterial Colonization
Bacterial vaginosis
Colonization with group A or B *Streptococcus*
Maternal Characteristics
Obesity
Diabetes
Severe anemia
HIV infection
Pregnancy Characteristics
Preterm birth
Post-term pregnancy
Labor Characteristics
Cesarean section
Chorioamnionitis
Internal monitoring
Manual removal of the placenta
Meconium staining (thick)
Operative vaginal delivery
Prolonged labor
Prolonged rupture of membranes

The classic triad of symptoms associated with endometritis is fever, tachycardia, and uterine tenderness. Women may also experience chills, malaise, foul-smelling lochia, or anorexia. The onset of symptoms varies depending on the infectious organism.

Evaluation includes physical examination to rule out other sources of infection, such as pneumonia, mastitis, pyelonephritis, or surgical site infection. A urine culture will rule out pyelonephritis and chest X ray may be indicated. Endometrial cultures are not obtained because uncontaminated cultures are difficult to obtain. Blood cultures are not routinely recommended if the woman is not acutely ill as most women respond well to empiric therapy. Thus, the diagnosis is generally clinical and based on ruling out other sources of infection.

Consultation should be obtained rapidly for any woman who has signs or symptoms that

indicate she is at risk for severe morbidity, such as hypotension, tachypnea, oxygen saturation of less than 95%, or shortness of breath.

Most endometrial infections are polymicrobial in nature, and frequently include a combination of aerobes and anaerobes from the genital tract. Broad-spectrum antibiotics are the treatment of choice, with several regimens being available. The gold standard is intravenous clindamycin (Cleocin) and gentamicin (Garamycin), administered until the woman is afebrile for 24 to 48 hours.[11] Most women respond to intravenous treatment within 48 to 72 hours and do not require follow-up with oral antibiotics, although oral antibiotics may be recommended for women with acute bacteremia. Women who present with mild endometritis several days after a vaginal birth may be offered oral antibiotics. Intramuscular regimens are available for women in low-resource settings where intravenous treatment is not available.

If the woman is not afebrile after 48 to 72 hours, the most likely problem is resistant organisms such as enterococci, and a change of antibiotic regimen will resolve the infection. In this situation, physician consultation and referral are indicated, as the choice of antibiotic depends on multiple factors including the possibility of other differential diagnoses such as pelvic abscess.

Prevention of Endometritis

Reducing cesarean section rates would significantly lower the rate of postpartum endometritis. Likewise, judicious obstetric practice—that is, prenatal treatment of infections, limited vaginal examinations during labor, avoidance of unnecessary induction of labor, delayed rupture of membranes, and minimal uterine manipulation during birth—can decrease the risk of a woman developing infection postpartum.

One dose of antimicrobial therapy, administered within 60 minutes before a cesarean section is performed, significantly reduces the incidence of endometritis; thus, this has become standard practice in the United States.[14] Vaginal cleansing with chlorhexidine prior to surgery also reduces the incidence of endometritis.[15] Use of traction on the umbilical cord to remove the placenta instead of manual removal is recommended as well.

Wound Complications

Wound complications include infections or dehiscence in a repaired perineal laceration or episiotomy following a vaginal birth, as well as surgical site complications such as seroma, hematoma, dehiscence, and infections in an abdominal incision following a cesarean birth.

Common pathogens associated with wound infections include *S. aureus*, *Streptococcus*, and both aerobic and anaerobic bacilli. The signs and symptoms of infection are similar for both abdominal and perineal wounds. Women usually experience a low-grade temperature, seldom greater than 38.3°C. Localized pain and edema are accompanied by red and inflamed repair edges. Exudate and wound separation or dehiscence may occur as well. Perineal wound infections are often accompanied by dysuria.[13]

Perineal Wound Infection and Laceration Dehiscence

Infection or dehiscence in a perineal laceration or episiotomy repair is generally confined to the skin and subcutaneous tissue. The treatment includes removal of sutures, plus opening, debriding, and cleansing the wound to allow the area to heal by granulation. Physician consultation is indicated. Antibiotics may be administered if cellulitis is present. Most perineal wound infections are not repaired again unless a third- or fourth-degree perineal extension is present. A reduction in the incidence of wound infections has been reported with the administration of prophylactic antimicrobial agents when a third- or fourth-degree laceration occurs.[16]

Surgical Site Complications

Midwives frequently collaborate in the care of women who have a cesarean birth, so they need knowledge of expected findings during recovery from this surgery and signs of surgical complications. Abdominal surgical site infections following cesarean birth usually appear 4 to 7 days after the procedure.

A seroma is a collection of fluid (serum) that appears under the surgical incision. Clear fluid may leak from the incision, and it may appear swollen around the edges. Seromas generally do not need to be treated but are observed carefully to make sure they do not become too large. Occasionally the skin incision is opened in a small area to allow the fluid to drain and/or a drainage tube can be placed temporarily.[17] Similarly, small hematomas usually reabsorb without intervention but larger ones may require evacuation of the clot. Both seromas and hematomas increase the woman's risk of infection. Once such a complication is identified, physician consultation is indicated and the surgical incision is reassessed frequently until it is determined that the seroma or hematoma is not increasing in size.

Surgical site infections can require antibiotics and occasionally drainage. Culture of exudates is not usually required. Reclosure of an abdominal wound may be indicated if dehiscence occurs. Depending on the degree of infection and dehiscence, management may include daily debridement, wound packing, antibiotic treatment, and drainage if necessary. Physician consultation or referral is indicated. If the woman has a persistent fever lasting for 3 or more days, she will be evaluated for complications such as sepsis, septic pelvic thrombophlebitis, abscess, and, rarely, septic shock.

Many techniques can help prevent surgical site infections. In addition to those used to prevent endometritis, such as use of prophylactic antibiotics, other techniques include shaving with clippers, skin cleansing with chlorhexidine (rather than iodine), and skin closure with sutures rather than staples.[17]

Urinary Tract Infection: Pyelonephritis

During postpartum, women are at increased risk for urinary tract infections (UTIs) for several reasons, including trauma to the urethra during birth, urinary retention, and pregnancy-induced dilation of the urethra and ureters. The organisms responsible for UTIs during the puerperium are similar to those observed in nonpregnant women—namely, *Escherichia coli*, which accounts for 80% to 90% of such UTIs, *Proteus mirabilis*, and *Klebsiella pneumoniae*.

Urinary tract infections may cause urinary frequency, urgency, dysuria, and, occasionally, lower abdominal pain. Pyelonephritis is characterized by low-grade fever, flank pain, costovertebral angle tenderness (CVAT), nausea, and vomiting. In contrast, postpartum women with upper or lower tract UTIs often present with fever and generalized symptoms without dysuria, frequency, or urgency. Therefore, any woman with a fever postpartum should be evaluated for a UTI.

Definitive diagnosis of UTI is made by culture of a clean-catch urine specimen that reveals a significant number of a single type of bacteria. Management of UTI and pyelonephritis during the postpartum period is the same as that for primary care outside of pregnancy. Nevertheless, one retrospective cohort review of 256 cases of antepartum pyelonephritis and 23 cases of postpartum pyelonephritis found antepartum and postpartum pyelonephritis to be equally morbid and more debilitating than pyelonephritis outside of the perinatal period.[18] The authors therefore urged prompt diagnosis and treatment of postpartum urinary tract infections.

Postpartum Bleeding Complications

After immediate postpartum hemorrhage, the two bleeding complications most frequently observed in the postpartum period are vulvar hematomas and secondary (late) postpartum hemorrhage, although fortunately, neither are common. Postpartum hemorrhage (PPH) is classified as primary or secondary. Primary hemorrhage occurs within the first 24 hours and is reviewed in the *Third Stage of Labor* chapter. Secondary (late) PPH occurs between 24 hours and 6 to 12 weeks postpartum.[19]

Genital Tract Hematomas

A hematoma is a localized collection of extravasated blood that is usually clotted. The dangers associated with its development include hemorrhage, anemia, and infection. The incidence of postpartum hematoma varies from 1 per 300 to 1 per 1500 births; this condition is most often the result of trauma from instrumental birth or episiotomy.[20] A hematoma arises following spontaneous or traumatic rupture of a blood vessel. Development occurs most often during birth or within a few hours after the birth. The most common sites for hematoma formation include the vulva, the vagina, and the broad ligament (**Figure 34-2**). Because postpartum hematomas vary in size from small to large, some may be overlooked, which could account for the variations in incidence estimates.

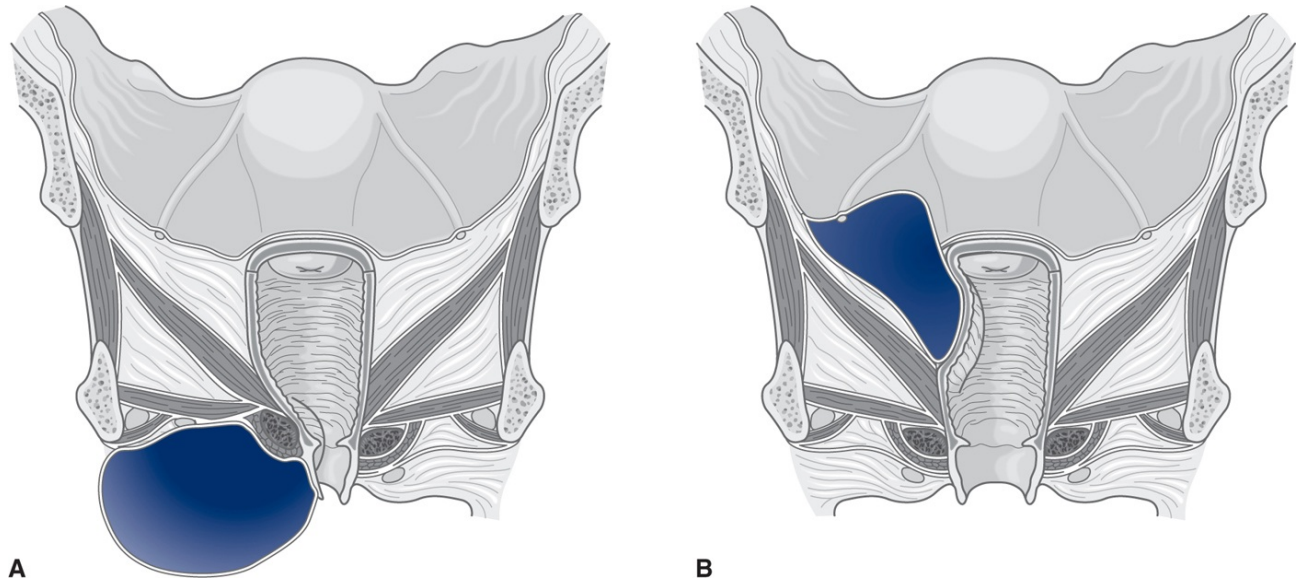


Figure 34-2 **A**. Vulvar hematoma. **B**. Broad ligament hematoma.

Risk factors for postpartum hematoma formation include operative vaginal birth and unrepaired laceration of a torn blood vessel, which can occur during injection of local anesthesia or during repair of a perineal or vaginal laceration. Failure to achieve hemostasis or ligate torn vessels above the vaginal apex prior to repair of an episiotomy or laceration also increases the risk of hematoma formation. Other risk factors include primiparity, multiple

gestation, large fetal weight, preeclampsia, coagulopathies, vulvovaginal varicosities, and prolonged second-stage labor.[20]

Signs and symptoms of vulvar or vaginal hematomas include (1) perineal, vaginal, urethral, bladder, or rectal pressure and severe pain; (2) a tense, fluctuant swelling; and (3) bluish or blue-black discoloration of tissue. The characteristic presentation of a hematoma is extreme pain out of proportion to the expected amount of discomfort and pain for that time period, and pain that develops quite rapidly in the first 2 to 6 hours after birth.[20] Women who have epidural analgesia may report a sudden onset of rectal pressure as the only symptom.

Presenting signs and symptoms of a broad ligament hematoma include lateral uterine pain sensitive to palpation, extension of pain into the flank, a painful swelling identified on high rectal examination, a ridge of tissue just above the pelvic brim extending laterally (i.e., the edge of the swollen broad ligament), and possibly abdominal distension. Subperitoneal hematoma formation—a rare occurrence following vaginal birth—leads to unrestricted bleeding and is typically not recognized until the woman experiences hypovolemic shock.

Vulvar hematomas are the most easily recognizable genital tract hematomas, while vaginal hematomas are generally identifiable when careful inspection and palpation of the vagina and cervix are performed. Small and moderate-sized hematomas may be spontaneously absorbed and managed expectantly. If a vulvar hematoma is small and visible, the edges can be demarcated with a pen, and it can be observed frequently to determine whether it is still growing.

If the hematoma is large or possibly continuing to grow (which indicates active bleeding within the hematoma space), a complete blood count can be ordered as soon as the hematoma is identified and an intravenous line started. Vital signs will need to be monitored frequently to observe for signs of shock, and pain medication prescribed as needed. A physician should be consulted for evaluation and care. Management might include monitoring hematocrit levels, vaginal packing to produce a counterpressure, or an incision to evacuate blood and blood clots and ensure closure of the cavity. The woman with a diagnosis of a hematoma may also need other surgical interventions, such as interventional radiology, blood replacement, or antibiotics.

Secondary Postpartum Hemorrhage

Approximately 1% of women experience a secondary postpartum hemorrhage, with most of these cases occurring within the first 2 weeks postpartum.[19] Common causes of delayed postpartum hemorrhage include uterine subinvolution at the placental site, retained placental fragments or membranes, and endometritis.[21,22] Less common causes include von Willebrand disease (vWD) or other coagulopathies, carcinoma, aneurysms, and arteriovenous malformations. Primary postpartum hemorrhage is a known risk factor for secondary postpartum hemorrhage.[23]

Uterine Subinvolution

The process of uterine involution involves anatomic changes in the size and shape of the uterus

as well as physiologic changes in the endometrial cavity that include involution of the altered spiral arteries that supported the placenta. Thrombus and myometrial smooth muscle contraction initially limit blood loss. The endometrial arteries regenerate their elasticity, shrink, and coil, and the cytotrophoblasts that invaded the endometrial decidua disappear.[21] By approximately 2 weeks postpartum, the uterus can no longer be palpated abdominally, though involution is not typically complete until 6 weeks after birth. When the uterus does not return to its prepregnant size and position within the expected time frame, the term uterine *subinvolution* is used.

The process of involution may be hampered by various factors—most commonly, retained placental fragments, leiomyomas, or infection. Subinvolution of the placental site occurs when the uteroplacental arteries (modified spiral arteries during pregnancy) do not remodel normally. The arteries may become filled with thrombi, have inadequate sloughing, or not regenerate an endothelial lining.[21] New-onset bleeding or hemorrhage as a result of placental site subinvolution most often occurs during the second postpartum week; it can present as an increase or return of lochia rubra or as a frank hemorrhage.[22] Interruption of involution may also occur when maternal activity is excessive; the direct etiology is not clear, but it is postulated that excessive activity might slow the healing process, although there is no direct evidence supporting this theory.

Diagnosis and Management of Postpartum Bleeding

The differential diagnoses for postpartum bleeding initially include placental subinvolution, retained placental fragments, and endometritis. Management depends on the degree of bleeding and whether the woman is hemodynamically stable.

Elements of the woman's history that can help establish a diagnosis are listed in **Table 34-2**. Bimanual pelvic examination findings may include a soft, tender uterus that is larger than expected for the postpartum week. Because retained placental fragments can cause endometritis, however, it is not always easy to differentiate between these two etiologies based on physical examination. Signs of endometritis also include malodorous lochia, tenderness or pain in the adnexa on movement of the uterus, and cervical motion tenderness elicited during a vaginal examination. If retained placental fragments are suspected, ultrasound is indicated. Note that ultrasound does not always provide definitive results, and it may not differentiate between placental fragments and blood clots.[24]

Table 34-2 Evaluation of Postpartum Bleeding

Evaluation	Clinical Considerations
Mode of birth	Retained placental fragments are more common after a vaginal birth; endometritis is more common after cesarean birth
History of menorrhagia or primary postpartum hemorrhage	History of menorrhagia may indicate the presence of a coagulopathy
Current medications	Medications that may predispose the woman to uterine bleeding such as anticoagulants; uterine relaxants

History of vaginal penetration or trauma	Possible new vaginal laceration
Symptoms of infection	Endometritis may present with fever, pain, and malodorous lochia

If the bleeding is heavier than usual but not markedly so, the woman is stable, the uterus has the expected size and shape, and there is no indication of infection or retained placental fragments, then outpatient treatment may begin with ergonovine (Ergotrate) or methylergonovine (Methergine), 0.2 mg by mouth, every 3 to 4 hours for 24 to 48 hours. Women taking either of these uterotonic agents should be informed that bleeding and cramping may increase during the course of therapy.

Physician consultation should be obtained if bleeding is heavy, the woman has signs of infection, or other abnormal findings are noted in the history and physical examination. For example, a history of menorrhagia or previous postpartum hemorrhage may indicate von Willebrand disease, as does the timing of a hemorrhage that occurs between 2 to 5 days postpartum. Preliminary laboratory evaluation includes a complete blood count, platelet count, activated partial thromboplastin time (aPTT), prothrombin time, and fibrinogen levels.[25,26]

Although different subtypes of von Willebrand disease are distinguished, 80% of cases identified during the postpartum period are due to decreased levels of factor VIII. Management depends on the specific subtype. The factor VIII level increases during pregnancy, which usually provides protection from bleeding and consequently is not a concern until approximately 2 days postpartum, when factor VIII levels decrease.[25,26] Postpartum hemorrhage risk secondary to von Willebrand disease persists for as long as 4 months postpartum. Approximately 50% of women with a diagnosis of von Willebrand disease are likely to experience a postpartum hemorrhage.[25,26] The initial evaluation is not directed at diagnosing this disease, but rather at ruling out other causes of bleeding. Other tests that can aid diagnosis include complete blood count and beta human chorionic gonadotropin (beta-hCG).

If infection is present, administration of broad-spectrum antibiotics such as ampicillin–sulbactam or cefoxitin will be recommended. If chlamydia is suspected, azithromycin 1 g orally (one dose) will be added to the regimen. Antibiotics can be administered intravenously or orally depending on the severity of the woman’s symptoms. If retained placental fragments are suspected, curettage may be performed.

Women who experience a sudden, severe delayed postpartum hemorrhage will often present to the emergency department for care. They may need acute care to support their hemodynamic stability.

Preeclampsia/Eclampsia in the Postpartum Period

Hypertension in the postpartum period may represent persistence of hypertension that developed antepartum or intrapartum, or it may be a new-onset condition. Complications of hypertension, which are among the leading causes of severe maternal morbidity and mortality, include cerebral hemorrhage, pulmonary edema, renal failure, hepatic rupture, and death. Approximately 18% of maternal mortality worldwide is secondary to hypertensive disorders of pregnancy, and 75% of maternal deaths that are secondary to hypertension occur after birth.[27,28] Early identification and prompt treatment can significantly reduce the incidence of these complications.[29]

Blood pressure may be transiently elevated postpartum secondary to administration of intravenous fluids or medications, but the presence of neurologic, cardiac, or gastrointestinal symptoms suggests a more serious underlying pathology.[30,31] The differential diagnosis in such cases includes persistence of gestational hypertension or preeclampsia, HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome, chronic hypertension, new-onset preeclampsia, and, more rarely, cerebral vasoconstriction syndrome, cerebral vascular accident, or hemolytic uremic syndrome.[30,31]

Preeclampsia is often first recognized antepartum or intrapartum, but it can develop de novo in the postpartum period. Approximately 30% of women who develop eclampsia do so in the postpartum period.[31] Postpartum preeclampsia often presents more subtly than onset during the prenatal period. Neurologic indicators (e.g., visual disturbance, headache), nausea, and vomiting appear more frequently during postpartum-onset preeclampsia compared to the prenatal-onset or intrapartum-onset condition.[31] Hypertension and proteinuria are not always present in postpartum preeclampsia and should not be considered the defining symptoms. New-onset late postpartum preeclampsia/eclampsia generally occurs after 48 hours postpartum and prior to 6 weeks postpartum.[31] This type of preeclampsia has been associated with maternal age of 40 years and older, black race, Latina ethnicity, body mass index of 30 or more, and gestational diabetes mellitus.[32]

Maternal blood pressure normally decreases for the first 48 hours postpartum, but then women with preeclampsia will experience an increase to a peak at 3 to 6 days postpartum.[29,31] Thus, women who develop postpartum preeclampsia are often at home when this condition manifests. Early recognition is key to prevention of eclampsia or worsening of the woman's condition—which is why all women should be educated about the signs and symptoms of postpartum preeclampsia prior to being discharged from care.[29] In addition, women who had preeclampsia prior to giving birth should have their blood pressure checked within the first 7 to 10 days after birth.

Physician consultation is indicated for women who have any degree of hypertension postpartum. Those who have mild hypertension (140–150 mm Hg systolic and 90–100 mm Hg diastolic) without any other signs or symptoms of pathology are likely to be treated with oral antihypertensive agents that are compatible with breastfeeding.

Blood pressure measurements exceeding 140/90 mm Hg require immediate assessment for further symptoms. The consensus bundle for severe hypertension during pregnancy and

postpartum developed by the National Partnership for Maternal Safety calls for every perinatal unit to have a protocol that includes notification of a physician or the primary care provider if the systolic blood pressure is 160 mm Hg or higher or the diastolic blood pressure is 110 mm Hg or higher for two measurements within 15 minutes.[29] If a woman being evaluated by a midwife presents with hypertension in these ranges, physician consultation and referral should be obtained quickly, as treatment should be initiated within 60 minutes following the second elevated reading.

Peripartum Cardiomyopathy

Cardiac disorders have not traditionally been a common complication among pregnant or postpartum women. Today, however, cardiovascular disorders account for 15.5% of pregnancy-related deaths in the United States, and cardiomyopathy accounts for 11% of such deaths.[33]

Peripartum cardiomyopathy (PPCM) is a relatively uncommon condition in which a woman develops new heart failure during the last few weeks of pregnancy or the first 5 months postpartum and there is no other identifiable cause.[34],[35] Regionally within the United States and worldwide, large variations are noted in the incidence of this condition. The overall incidence in the United States is 1 in 4000 births, but the incidence of PPCM in southern California is about 1 in 3000 births, and that in Haiti is approximately 1 in 300 births.[36]

For the midwife, care for a woman with PPCM is limited to prompt referral to an appropriate specialist or interdisciplinary care team, preferably including a cardiologist experienced in heart failure.[37] Although care and management of a woman with PPCM are not in the scope of midwifery practice, being able to recognize and refer early can be beneficial for women, as it is sometimes possible for the heart to recover some of its lost function.[34]

The etiology of PPCM remains unknown. Risk factors for this cardiovascular disorder include chronic hypertension, preeclampsia, twin gestation, teenage pregnancy, advanced maternal age, family history of heart failure, and use of tocolytics during pregnancy. One challenge in diagnosing PPCM is that the symptoms are very similar to the normal symptoms seen during pregnancy and in preeclampsia.[37] Fatigue, shortness of breath, edema, and weight gain are symptoms of both PPCM and normal pregnancy, for example. The symptoms most commonly associated with PPCM are marked shortness of breath, orthopnea, tachycardia, palpitations, chest pain, cough, and edema.[38]

The definitive diagnosis of PPCM is made by echocardiogram, but a 12-lead electrocardiogram showing either signs of left ventricular hypertrophy or ST-T wave abnormalities, or a markedly elevated beta natriuretic peptide (BNP) blood test, indicates a strong likelihood of PPCM.[34] Physical examination may reveal crackles in the bases of the lungs, pitting edema, jugular vein distension, a heart sound, or laterally shifted point of maximum impulse when auscultating the heart.[34]

Although PPCM can present during the last few weeks of pregnancy, approximately 75% of women with this condition are diagnosed in the postpartum period.[34] This pattern might indicate that features of PPCM are present during pregnancy, but that women seek help only when the symptoms of shortness of breath, fatigue, and so on do not resolve postpartum.

Women who survive PPCM face many challenges: A strict diet, fluid restriction, and a medication regimen become a daily part of life for many. It is unclear why some women regain their ejection fraction and others do not, but evidence suggests that an ejection fraction remaining greater than 30% for the course of the illness is a key component.[39] In extreme cases, heart transplantation or a surgically implanted mechanical ventricular assist device may be needed for survival.[40]

Vigilance is needed on the part of the midwife to recognize and refer women suspected of

having PPCM. At this time, it is not recommended that women with a history of PPCM become pregnant again, as there is an increased risk of recurrence of PPCM. Nevertheless, this condition is not an absolute contraindication to pregnancy, provided that the ejection fraction has recovered to baseline.

Postpartum Thrombophlebitis and Thromboembolism

Postpartum thrombophlebitis is defined as inflammation of a vein accompanied by formation of a thrombosis (blood clot). Thrombophlebitis can occur in both superficial and deep veins. Thrombosis is more common in women with varicosities and in women who are perhaps genetically susceptible to vein-wall relaxation and venous stasis. Venous thromboembolism (VTE) remains a leading cause of morbidity and mortality in the United States.[1] During the postpartum period, women have a 21.5-fold to 84-fold increased risk for venous thrombosis and embolism compared to nonpregnant women.[41] Results from the Nationwide Inpatient Sample (NIS) revealed a 47% increase in postpartum hospitalizations for VTE between 1994 and 2009.[41] Many thromboembolic events can be anticipated or prevented.

Risk assessment for VTE should begin prior to pregnancy or prenatally. Subsequently, follow-up risk assessment should occur during the intrapartum period and up to 6 weeks postpartum.[42] Nearly half of all pregnancy-related thromboses occur during the puerperium.[43]

Virchow's triad describes the three factors that influence thrombosis formation during pregnancy and postpartum: (1) stasis, (2) hypercoagulable state, and (3) vascular trauma. Venous-wall relaxation resulting from the effects of progesterone and pressure on the veins by the gravid uterus fosters venous stasis. Pregnancy is also a hypercoagulable state, and this state persists throughout the early postpartum period. Venous-wall inflammation and endothelial damage secondary to distension, in the setting of this hypercoagulable state, predisposes the woman to thrombus formation.[44] Prolonged venous compression during positioning for labor or birth may contribute to the problem. Cesarean section also is considered a risk factor for thrombosis; thus, mechanical thromboprophylaxis, such as alternating compression devices, is recommended before and after cesarean birth.

Deep vein thrombosis (DVT) is more common in the left lower extremity (accounting for 70% to 80% of cases), presumably due to compression of the left iliac vein from the gravid uterus and the crossing of the right iliac artery, as well as the nearly 50% decreased blood flow to the lower extremities.[44-47] Women with BMIs of 30 or greater are at higher risk for postpartum and postsurgical thrombus formation than women of lower BMIs; this risk also increases as BMI increases.[48] Daily thromboprophylaxis with low-molecular-weight heparin starting 12 hours post birth or surgery is advised for women with class III obesity (BMI ≥ 40). Weight-based dosage may be more effective as BMI increases than a standard dose of enoxaparin 40 mg daily.[48]

The greatest risk associated with thrombophlebitis is pulmonary embolism.[46] This is true for women who develop DVT, whereas pulmonary embolism is unlikely in women who have a superficial thrombophlebitis. Tachypnea, dyspnea, and sharp chest pain of sudden onset are the symptoms most commonly associated with pulmonary embolism. Sudden onset of these symptoms requires urgent evaluation. Many other, less specific symptoms may be present, including altered lung or heart sounds and apprehension as the woman's blood oxygen level decreases. Under no circumstances should the woman's leg be massaged when thrombophlebitis is suspected. Risk factors for postpartum thrombophlebitis are summarized in **Table 34-3**.

Table 34-3	Risk Factors for Venous Thrombosis
Maternal History–Related Risks	
Obesity	
Smoking	
Age > 35 years	
History of thrombosis	
Inherited thrombophilias	
Antiphospholipid antibody syndrome	
Sickle cell disease	
Heart disease	
Diabetes	
Immobility (paraplegia)	
Pregnancy-Related Risks	
Hypercoagulable state	
Venous stasis	
Multiple pregnancy	
Preeclampsia	
Labor- and Birth-Related Risks	
Operative vaginal birth	
Cesarean birth	
Infection	
Vascular trauma	
Immobilization	
Postpartum hemorrhage	
Preterm birth	
Stillbirth	

Based on Waldman M, Sheiner E, Sergienko R, Shoham-Vardi I. Can we identify risk factors during pregnancy for thrombo-embolic events during the puerperium and later life? *J Matern Fetal Neonatal Med.* 2015;28(9):1005-1009[45]; American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 84: prevention of deep vein thrombosis and pulmonary embolism. *Obstet Gynecol*. 2007;110:429-440. [Reaffirmed 2016][47]; American College of Obstetricians and Gynecologists. Practice Bulletin No. 156: obesity in pregnancy. *Obstet Gynecol.* 2015;126:e112-e126.[48]

The signs and symptoms of superficial thrombophlebitis and deep vein thrombosis are summarized in **Table 34-4**. DVT presents as unilateral edema and an increase in leg circumference. Tenderness and a feeling of warmth may be noted over the affected veins. Superficial thrombophlebitis is more localized in appearance, but it may be difficult to distinguish between the two conditions based on physical examination.

Table 34-4	Signs and Symptoms of Superficial Thrombophlebitis Versus Deep Vein Thrombosis
Condition	**Signs and Symptoms**
Superficial	Inflammation at the site

thrombophlebitis	Leg pain Localized heat Palpation of a knot or cord, tenderness
Deep vein thrombosis	Abrupt onset with leg pain worsening with motion or when standing Generalized edema of ankle, leg or calf Mild tachycardia and possible slight elevation in temperature Pain with calf pressure Tenderness along course of involved vessel with possible palpable cord

Based on Bennett A, Chunilal S. Diagnosis and management of deep vein thrombosis and pulmonary embolism in pregnancy. *Semin Thromb Hemost.* 2016;42:760-773[46]; American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 84: prevention of deep vein thrombosis and pulmonary embolism. *Obstet Gynecol*. 2007;110:429-440. [Reaffirmed 2016].[47]

The differential diagnosis includes superficial thrombophlebitis versus deep vein thrombosis, muscle strain or trauma, ruptured cyst, vasculitis, and lymphedema. Compression ultrasound is the primary diagnostic test, with or without color Doppler imaging.[49] Diagnostic ultrasound is indicated for DVT as well as for superficial thrombophlebitis to rule out underlying DVT. Laboratory analysis includes high-sensitivity d-dimer studies, which measure fibrin degradation.

Eliciting of Homan's sign (pain on dorsiflexion of the foot) was once thought to be a diagnostic sign of deep vein thrombosis. A positive Homan's sign may, in fact, indicate DVT—but it could also indicate a strained calf muscle. This sign is present in fewer than one-third of women who have a confirmed DVT. Conversely, a negative Homan's sign does not rule out deep vein thrombosis. Furthermore, eliciting Homan's sign could disturb a thrombosis; thus, this test is no longer recommended for evaluation of leg pain in the postpartum period.

Management differs significantly for superficial thrombophlebitis and deep vein thrombosis. Treatment of superficial thrombophlebitis includes leg rest, elevation of the affected extremity, supportive stockings, and nonsteroidal anti-inflammatory drug (NSAID) analgesia as needed. Deep vein thrombosis treatment includes these elements, but adds anticoagulants are added. Ambulation should occur gradually as tolerated. Supportive stockings are recommended. Anticoagulation therapy is usually continued for at least 6 months or longer if needed. Warfarin (Coumadin) is considered safe during lactation. Physician referral is indicated for management of DVT.

Thyroiditis and Thyroid Storm

Some postpartum thyroid disorders may occur slowly and present with subtle symptoms; alternatively, they may develop suddenly as a marked illness. These disorders can occur anytime during the first postpartum year, so symptoms may be remote from childbirth and not identified as a postpartum complication. Postpartum thyroid disease may be difficult to recognize, as symptoms are similar to those commonly encountered in the postpartum period: lack of energy, sleep difficulties, appetite changes, weight changes, memory difficulty, and depressed affect. These symptoms are also found in women with postpartum mood disorders, as discussed in the *Mental Health Conditions* chapter. Postpartum thyroid dysfunction has been associated with postpartum depression.[50] Thyroid illness and mood disorder symptoms are so similar that thyroid-stimulating hormone (TSH) screening is an initial step in the evaluation of postpartum mood disorders.

Postpartum Thyroiditis

Postpartum thyroiditis is an autoimmune thyroiditis that can present as (1) transient hypothyroidism, (2) transient hyperthyroidism, or (3) hyperthyroidism followed by hypothyroidism and then recovery.[51,52] The prevalence varies from 1.1% to 16.7%.[53] Women at increased risk for postpartum thyroiditis include those with gestational diabetes, autoimmune disorders such as type 1 diabetes, previous thyroid dysfunction, and history of thyroid disorders.[54] Although most women recover and are euthyroid within one year, a small proportion of women remain hypothyroid and need long-term replacement therapy.

The clinical course of this disorder starts with thyroid inflammation that damages thyroid follicles. These changes result in excessive thyroid hormone release and hyperthyroidism, which is then followed by transient hypothyroidism once the stores of thyroid hormone are exhausted. Ultimately, the inflammation subsides and thyroid hormone synthesis resumes. The hyperthyroid phase typically occurs around 3 months postpartum and resolves spontaneously within 2 to 3 months.

Signs and symptoms of postpartum thyroiditis resemble common postpartum recovery symptoms. In addition, there is usually no recognizable pain or swelling of the thyroid. Common symptoms of postpartum thyroiditis depend on the phase (hyper or hypo) of the illness. **Table 34-5** compares and contrasts common signs and symptoms of both the hyperthyroidism and hypothyroidism phases of the illness.

Table 34-5 Signs and Symptoms of Postpartum Thyroiditis

Condition	When Condition Manifests	Signs and Symptoms
Hyperthryoiditis	1–4 months postpartum	Anxiety, fatigue, goiter, heat intolerance/sweats, insomnia, irritability, weight loss
Hypothyroiditis	4–8 months postpartum	Constipation, depression, dry skin, fatigue, goiter, impaired concentration

Based on Yalamanchi S, Cooper DS. Thyroid disorders in pregnancy. *Curr Opin Obstet Gynecol.* 2015;27(6):406-415[53]; Carney LA, Quinlan JD, West JM. Thyroid disease in pregnancy. *Am Fam Physician.* 2014;89(4):273-278[54]; Argatska A, Nonchev B, Orbetzova M, Pehlivanov B. Postpartum thyroid dysfunction in women with autoimmune thyroiditis. *Gynecol Endocrinol.* 2016;32(5):379-382.[55]

Because thyroiditis does not become manifest until later in the postpartum period, its symptoms may go unnoticed in either phase of the disorder and misdiagnosis may occur. In the hyperthyroid phase, low levels of TSH, the presence of thyroid peroxidase antibodies, and a lack of TSH receptor antibodies suggest the diagnosis of postpartum thyroiditis. In the hypothyroid phase, an elevated TSH level and a positive test for antithyroid peroxidase antibodies are pathognomonic for this disorder. Postpartum depression and anxiety have similar symptoms, however, and should be included in the differential diagnosis. Women experiencing thyroiditis can have an episode of mild dysphoria that could be mistaken for psychosis.

Treatment depends on the phase and degree of symptoms. If symptoms are mild, no treatment will be instituted. If symptoms of hyperthyroidism are bothersome, beta blockers are the usual treatment of choice, whereas thyroid supplementation with levothyroxine (Synthroid) is prescribed for hypothyroid symptoms.[51,55] Physician consultation or management is indicated. There is no consensus on criteria for treatment, dose of medication, or duration of treatment.

Thyroid Storm

Thyroid storm, also known as clinical thyrotoxicosis, is a rare, life-threatening, acute exacerbation of hyperthyroidism. It occurs earlier than thyroiditis (usually during the first month postpartum) and is distinguished by an abrupt onset of symptoms. This disorder, which results from overproduction of thyroid hormone, is characterized by the sudden onset of a hypermetabolic state. Signs and symptoms include nausea and vomiting, diarrhea, fever (> 41°C), tachycardia, and tremor. Potential outcomes include dehydration, seizures, coma, and death if thyroid storm goes untreated. Increased T_4 levels can lead to cardiomyopathy and heart failure.[56]

The diagnosis of thyroid storm is made clinically, and treatment is initiated prior to obtaining the results of confirmatory laboratory studies. Women in thyroid storm usually present to an emergency department acutely ill and may initially be assumed to have preeclampsia. The presence of neuropsychiatric symptoms and hyperpyrexia distinguish thyroid storm from postpartum preeclampsia or eclampsia.[55] Women in thyroid storm will be hospitalized for intensive care management.

Conclusion

Midwives who are astute in assessing the normal physiology of the puerperium will readily recognize pathologic processes and should be prepared to initiate early treatment and prevent worsening illness. Despite the fact that most postpartum complications require physician management, midwives will typically initiate the diagnostic work-up and recognize occasions when it is appropriate to consult, collaborate, or refer, thereby reducing first-line morbidity and mortality. While physicians medically manage some complications, the midwife will continue to manage other aspects of the woman’s postpartum course and adjustments.

Resources

Organization	Description	Webpage
California Maternal Quality Care Collaborative (CMQCC)	Preeclampsia Toolkit	https://www.cmqcc.org/resources-tool-kits/toolkits/preeclampsia-toolkit
Centers for Disease Control and Prevention (CDC)	Guideline for the Prevention of Surgical Site Infection, 2017	http://jamanetwork.com/journals/jamasurgery/fullarticle/2623725
Global Library of Women's Medicine (GLOWM)	Management of Secondary Postpartum Hemorrhage	https://www.glowm.com/pdf/PPH_2nd_edn_Chap-56.pdf

References

1. Creanga AA, Sylverson C, Seed K, Callaghan WM. Pregnancy-related mortality in the United States, 2011–2013. *Obstet Gynecol*. 2017;130:366-373.
2. Zuckerwise LC, Lipkind HS. Maternal early warning systems: towards reducing preventable maternal mortality and severe maternal morbidity through improved clinical surveillance and responsiveness. *Semin Perinatol*. 2017;41(3):161-165.
3. American College of Obstetricians and Gynecologists, Society for Maternal–Fetal Medicine; Kilpatrick SK, Ecker JL. Severe maternal morbidity: screening and review. *Am J Obstet Gynecol*. 2016;215(3):B17-B22.
4. Kekre AN, Vijayanand S, Dasgupta R, Kekre N. Postpartum urinary retention after vaginal delivery. *Int J Gynaecol Obstet*. 2011;112(2):112-115.
5. Mulder FEM, Rengerink KO, van der Post JAM, Hakvoort RA, Roovers JP. Delivery-related risk factors for covert postpartum urinary retention after vaginal delivery. *Int Urogynecol J*. 2016;27:55-60.
6. Mulder FE, Schoffelmeer MA, Hakvoort RA, et al. Risk factors for postpartum urinary retention: a systematic review and meta-analysis. *BJOG*. 2012;119(12):1440-1446.
7. Gould CV, Umscheid CA, Agarwal RK, Kuntz G, Pegues DA; Healthcare Infection Control Practices Advisory Committe Guideline for prevention of catheter-associated urinary tract infections 2009. Updated 2017. Available at: https://www.cdc.gov/infectioncontrol/pdf/guidelines/cauti-guidelines.pdf. Accessed September 9, 2017.
8. Mulder FE, Hakvoort RA, de Bruin JP, Janszen EW, van der Post JAM, Roovers JP. Long-term micturition problems of asymptomatic postpartum urinary retention: a prospective case-control study. *Int Urogynecol J*. 2017. [Epub ahead of print]. doi:10.1007/s00192-017-3457-6.
9. Buddeburg BS, Aveling W. Puerperal sepsis in the 21st century: progress, new challenges and the situation worldwide. *Postgrad Med J*. 2015;91(1080):572-578.
10. Clapp MA, Little SE, Zheng J, Robinson JN. A multi-state analysis of postpartum readmissions in the United States. *Am J Obstet Gynecol*. 2016;215:113.e1-113.e9.
11. Smaill FM, Gyte GM. Antibiotic prophylaxis versus no prophylaxis for preventing infection after cesarean section. *Cochrane Database Syst Rev*. 2010;1:CD007482. doi:10.1002/14651858.CD007482.pub2.
12. Mackeen AD, Packard RE, Ota E, Speer L. Antibiotic regimens for postpartum endometritis. *Cochrane Database Syste Rev*. 2015;2:CD001067. doi:10.1002/14651858.CD001067.pub3.
13. Karsnitz DB. Puerperal infections of the genital tract: review and management. *J Midwifery Womens Health*. 2013;58(6):632-642.
14. American College of Obstetricians and Gynecologists. Practice Bulletin No. 120: use of prophylactic antibiotics in labor an delivery. *Obstet Gynecol*. 2011;117(6):1472-1483. [Reaffirmed 2013].
15. Haas DM, Morgan S, Contreras K. Vaginal preparation with antiseptic solution before cesarean section for preventing postoperative infections. *Cochrane Database Syst Rev*. 2014;12:CD007892. doi:10.1002/14651858.CD007892.pub5.
16. Buppasiri P, Lumbiganon P, Thinkhamrop J, Thinkhamrop B. Antibiotic prophylaxis for third- and fourth-degree perineal tear during vaginal birth. *Cochrane Database Syst Rev*. 2014;10:CD005125. doi:10.1002/14651858.CD005125.pub4.
17. Kawakita T, Landy HJ. Surgical site infections after cesarean delivery: epidemiology, prevention and treatment. *Matern Health Neonat Perinatol*. 2017;3:12. doi:10.1186/s40748-017-0051-3.
18. McDonnold M, Friedman A, Raker C, Anderson B. Is postpartum pyelonephritis associated with the same maternal morbidity as antepartum pyelonephritis? *J Matern Fetal Neonat Med*. 2012;25(9):1709-1711.
19. American College of Obstetricians and Gynecologists. Practice Bulletin No. 76: postpartum hemorrhage. *Obstet Gynecol*. 2006;108:1039-1047. [Reaffirmed 2013].
20. Ridgway LE. Puerperal emergency: vaginal and vulvar hematomas. *Obstet Gynecol Clin North Am*. 1995;22(2):275-282.
21. Weydert JA, Brenda JA. Subinvolution of the placental site as an anatomic cause of postpartum uterine bleeding. *Arch Pathol Lab Med*. 2006;130:1538-1542.
22. Kavalar R, Arko D, Fokter DN, Takacˇ I. Subinvolution of placental bed vessels: case report and review of the literature. *Wien Klin Wochenschr*. 2012;124(19-20):725-730.
23. Debost-Legrand A, Riviere O, Dossou M, Vendittelli F. Risk factors for severe secondary postpartum hemorrhages: a historical cohort study. *Birth*. 2015;42(3):235-241.
24. Sellmyer MA, Desser TS, Maturen KE, Jeffrey RB Jr, Kamaya A. Physiologic, histologic, and imaging features of retaine products of conception. *Radiographics*. 2013;33(3):781-796.
25. American College of Obstetricians and Gynecologists. Committee Opinion No. 580: von Willebrand disease in women. *Obstet Gynecol*. 2013;122:1368-1373. [Reaffirmed 2015].
26. Pacheco LD, Costantine MM, Saade GR, Mucowski S, Hankins GD, Sciscione AC. von Willebrand disease and pregnancy: a practical approach for the diagnosis and treatment. *Am J Obstet Gynecol*. 2010;203(3):194-200.
27. Khan KS, Wojdyla D, Say L, Gulmezoglu AM, Van Look PF. WHO analysis of causes of maternal death: a systematic review. *Lancet*. 2006;367:1066-1074.
28. Zuleta-Tobón JJ, Pandales-Pérez H, Sánchez S, Vélez-Álverez GA, Velásquez-Penagos JA. Errors in the treatment of

hypertensive disorders of pregnancy and their impact on maternal mortality. *Int J Gynaecol Obstet* 2013;121:78-81.
29. Bernstein PS, Martin JN, Barton JR, et al. Consensus bundle on severe hypertension during pregnancy and the postpartum period. *J Midwifery Womens Health*. 2017;62(4):493-501.
30. American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. 2013. Available at: https://www.cdc.gov/infectioncontrol/pdf/guidelines/cauti-guidelines.pdf. Accessed September 15, 2017.
31. Sibai BM. Etiology and management of postpartum hypertension-preeclampsia. *Am J Obstet Gynecol*, 2012;206(6):470-475.
32. Bigelow CA, Pereira GA, Warmsley A, et al. Risk factors for new-onset late postpartum preeclampsia in women without history of preeclampsia. *Am J Obstet Gynecol*. 2014;210:338.e1-338.e8.
33. Centers for Disease Control and Prevention. Pregnancy mortality surveillance system: trends in pregnancy-related deaths. Available at: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html. Accessed January 6, 2017.
34. Sliwa K, Hilfiker-Kleiner D, Petrie MC, et al. Current state of knowledge on aetiology, diagnosis, management, and therap of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy. *Eur J Heart Failure*. 2010;12(8):767-778.
35. Jagodzinska A, Gondek A, Pietrzak B, Cudnoch-Jedrzejewska A, Mamcarz A, Wielgos M. Peripartum cardiomyopathy: from pathogenesis to treatment. *J Perinat Med*. 2017. [Epub ahead of print]. doi:10.1515/jpm-2016-0247.
36. Okeke T, Ezenyeaku C, Ikeako L. Peripartum cardiomyopathy. *Ann Med Health Sci Res*. 2013;3(3):313-319.
37. Fowler K, Schafer D, Sica M, et al. Peripartum cardiomyopathy (PPCM): interdisciplinary coordination for a complex patient population. *Heart Lung*. 2017;46(3):212-213.
38. Dekker RL, Morton CH, Singleton P, Lyndon A. Women's experiences being diagnosed with peripartum cardiomyopathy: qualitative study. *J Midwifery Womens Health*. 2016;61(4):467-473.
39. Elkayam U, Habakuk O. The search for a crystal ball to predict early recovery from peripartum cardiomyopathy? *JACC: Heart Failure*. 2016;4(5):389-391.
40. Hamdan R, Nassar P, Zein A, Issa M, Mansour H, Saab M. Peripartum cardiomyopathy, place of drug therapy, assist devices, and outcome after left ventricular assistance. *J Crit Care*. 2017;37:185-188.
41. Ghaji N, Boulet SL, Tepper N, Hooper WC. Trends in venous thromboembolism among pregnancy-related hospitalizations, United States, 1994–2009. *Am J Obstet Gynecol*. 2013;209:433.e1-433.e8.
42. D'Alton ME, Friedman AM, Smiley RM, et al. National Partnership for Maternal Safety: consensus bundle on venous thromboembolism. *J Midwifery Womens Health*. 2016;61(5):649-657.
43. Saghazadeh A, Rezaei N. Inflammation as a cause of venous thromboembolism. *Crit Rev Oncol Hematol*. 2016;99:272-285.
44. Chan WS, Spencer FA, Ginsberg JS. Anatomic distribution of deep vein thrombosis in pregnancy. *Can Med Assoc J*. 2010;182(7):657-660.
45. Waldman M, Sheiner E, Sergienko R, Shoham-Vardi I. Can we identify risk factors during pregnancy for thrombo-embolic events during the puerperium and later life? *J Matern Fetal Neonatal Med*. 2015;28(9):1005-1009.
46. Bennett A, Chunilal S. Diagnosis and management of deep vein thrombosis and pulmonary embolism in pregnancy. *Semin Thromb Hemost*. 2016;42:760-773.
47. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 84: prevention of deep vein thrombosi and pulmonary embolism. *Obstet Gynecol*. 2007;110:429-440. [Reaffirmed 2016].
48. American College of Obstetricians and Gynecologists. Practice Bulletin No. 156: obesity in pregnancy. *Obstet Gynecol*. 2015;126:e112-e126.
49. Guyatt GH, Akl EA, Crowther M, Gutterman DD, Schuünemann HJ; American College of Chest Physicians Antithrombotic Therapy and Prevention of Thrombosis Panel. Executive summary: antithrombotic therapy and prevention of thrombosis, 9th ed.: American College of Chest Physicians evidence-based clinical practice guidelines. *Chest*. 2012;141:7S-47S.
50. LeDonne M, Mento C, Settineri S, Antonelli A, Benvenga S. Postpartum mood disorders and thyroid autoimmunity. *Front Endocrinol (Lausanne)*. 2017;8:91. doi:10.3389/fendo.2017.00091.
51. Goldstein AL. New-onset Graves' disease in the postpartum period. *J Midwifery Womens Health*. 2013;58:211-214.
52. Alexander EK, Pearce EN, Brent GA, et al. 2017 guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2017;27(3):315-389. Available at: http://online.liebertpub.com/doi/pdf/10.1089/thy.2016.0457. Accessed September 15, 2017.
53. Yalamanchi S, Cooper DS. Thyroid disorders in pregnancy. *Curr Opin Obstet Gynecol*. 2015;27(6):406-415.
54. Carney LA, Quinlan JD, West JM. Thyroid disease in pregnancy. *Am Fam Physician*. 2014;89(4):273-278.
55. Argatska A, Nonchev B, Orbetzova M, Pehlivanov B. Postpartum thyroid dysfunction in women with autoimmune thyroiditis. *Gynecol Endocrinol*. 2016;32(5):379-382.
56. American College of Obstetricians and Gynecologists. Practice Bulletin No. 148: thyroid disease in pregnancy. *Obstet Gynecol*. 2015;125:996-1006.

CHAPTER

35

Breastfeeding and the Mother–Newborn Dyad

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Introduction

Human milk is universally recognized as the unequaled, optimal form of nourishment for infants during the first 6 months to 1 year of life.[1-4] Breastfeeding has numerous health benefits for both a woman and her infant.[5-7] Although breast milk is by far the best nutritional source for infants, a few women cannot breastfeed or choose not to breastfeed. This chapter reviews breastfeeding, contraindications, common problems, management of medications for breastfeeding women, and other infant feeding methods. The term "breast milk" as used throughout this chapter refers to human milk. Commercially or home-prepared breast milk substitutes are colloquially known as formulas and are termed as such throughout this chapter.

Definitions

Research has shown that the key breastfeeding indicators that have clinically significant impacts on health outcomes are exclusivity, duration, and timing of breastfeeding initiation after birth. Although breastfeeding research is hampered by use of a variety of definitions for breastfeeding behaviors, most breastfeeding research uses the 2008 World Health Organization (WHO) definitions, which are presented in **Table 35-1**.[8] In this chapter the term "bottle feeding" denotes use of a bottle containing either breast milk or formula and should not be assumed to be a synonym for breast milk substitutes. The term "chestfeeding" is usually considered more appropriate than "breastfeeding" within the transgender and gender nonconforming (TGNC) community.

Table 35-1 Breastfeeding Terms

Term	Definition
Breastfeeding	Feeding of breast milk (including milk expressed or from a wet nurse). A breastfeeding infant may also ingest any other food or liquids.
Breastfeeding initiation	Breastfeeding of a newborn at least once.
Early initiation of breastfeeding	Provision of mother's milk within the first hour after birth. The World Health Organization (WHO) recommends early initiation of breastfeeding.
Exclusive breastfeeding	Feeding of only breast milk (including milk expressed or from a wet nurse); allows drops, syrups, vitamins, minerals, and medicines but no other substances ingested orally. WHO recommends that infants exclusively breastfeed up to 6 months of age; the American Academy of Pediatrics also recommends exclusive breastfeeding for approximately 6 months.
Predominant breastfeeding	Feeding of breast milk (including milk expressed or from a wet nurse) as the predominant source of nourishment. May include water, water-based drinks, fruit juice, drops, syrups, vitamins, minerals, and medicines, but does not include nonhuman milk or food-based fluids.
Partial or complementary breastfeeding	Feeding of some breast milk in addition to other liquids (e.g., breast milk substitute/formula or milk) and foods.
Breastfeeding duration	The number of days, months, or years that a child receives any breast milk. WHO recommends that breastfeeding continue, with the addition of appropriate complementary (family) foods at around 6 months, for up to 2 years or age or longer, as long as the mother and child desire.

Based on World Health Organization. Indicators for assessing infant and young child feeding practices: Part 1 definitions. 2008. Available at: http://whqlibdoc.who.int/publications/2008/9789241596664_eng.pdf. Accessed August 31, 2017.[8]

Prevalence of Breastfeeding

Among women who gave birth in 2013, 81.1% initiated breastfeeding, more than half (51.8%) were breastfeeding at 6 months, and almost one-third (30.7%) were breastfeeding at 12 months.[9] Despite many national, state, and local initiatives to support breastfeeding, most states are not meeting the *Healthy People 2020* goals for breastfeeding initiation and exclusive breastfeeding at 3 to 6 months.[10] Furthermore, significant racial and ethnic differences in breastfeeding rates are evident. Non-Hispanic black newborns have lower rates of breastfeeding initiation compared to Hispanic and non-Hispanic white newborns (64.3% versus 81.5% and 81.9%, respectively) and lower rates of exclusive breastfeeding through 6 months (14% versus 22.5% and 18.2%, respectively).[11] Because breastfeeding rates decline the most during the first few months after birth, efforts to support women during the early postpartum period are largely focused on this time period.

Lactogenesis and the Composition of Human Milk

Lactogenesis II (also referred to as secretory activation) heralds the onset of copious milk production following childbirth. Lactogenesis II generally occurs 2 to 4 days after delivery of the placenta (60 hours) but may not occur for up to 5 to 7 days postpartum.[12] The physiology of lactogenesis II, which is triggered by the withdrawal of progesterone, is reviewed in detail in the *Anatomy and Physiology of Postpartum* chapter.

Human milk is an individual-specific biofluid that contains multiple bioactive, cellular, and nutritional ingredients (**Figure 35-1**). Approximately 80% of the content of human milk is water. The other 20% consists of a wide range of substances:[13]-[15]

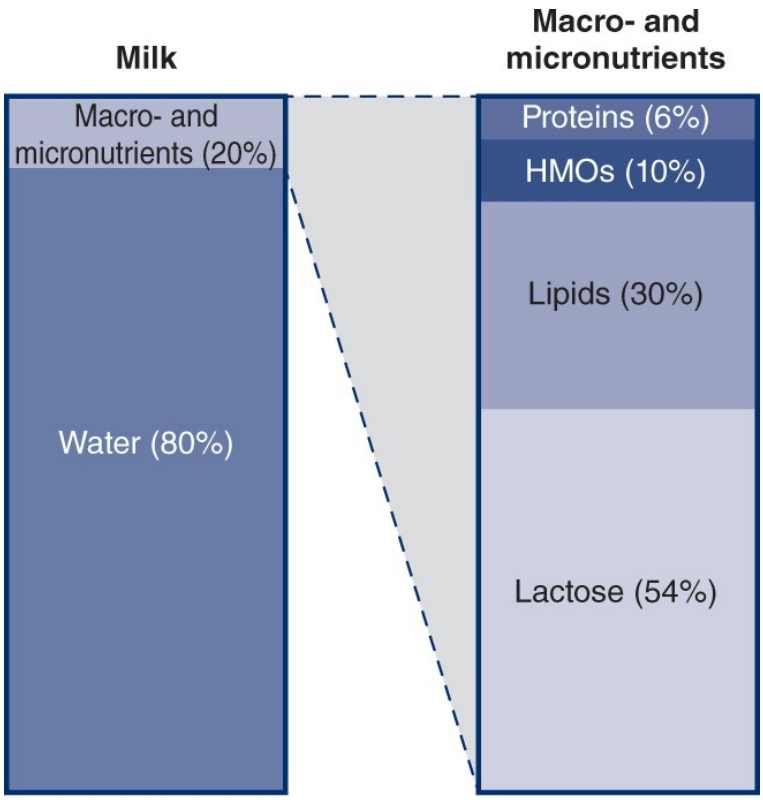


Figure 35-1 Composition of human milk.

Abbreviation: HMOs, human milk oligosaccharides.

Note: Although the percentage of various constituents vary over time and even during the course of a single breastfeeding session, there are > 200 individual bioactive substances that have been identified in human milk.

- Macronutrients: The primary macronutrients in human milk are carbohydrates (predominately lactose), milk fat (lipids), proteins such as whey and casein, and minerals including calcium and phosphorus.
- Micronutrients: Vitamin D and vitamin K are present in constitutionally low levels. Other vitamins vary on the basis of maternal diet.
- Bioactive agents: Substances that can inhibit inflammation and induce antibody responses include growth factors, cells involved in immune response (macrophages, T cells, lymphocytes, leukocytes), cytokines, immunoglobulins (predominately IgA, but also IgG

and IgM), and oligosaccharides.

Both inter-individual and intra-individual variability exist in the composition of human milk. The composition of human milk is affected by a number of factors, including gestational age at the time of birth, time of day, stage of lactogenesis, and maternal environmental factors. The lipid component of human milk is the most variable in terms of concentration.

Human milk is commonly classified as colostrum (prenatal onset of lactogenesis II), transitional milk (2–5 days after birth), and mature milk (lactogenesis II and III). Colostrum contains relatively large quantities of protein and less lactose compared to mature milk. This liquid also contains large quantities of secretory immunoglobulins (IgA), oligosaccharides, and growth hormones. Based on these components, some authors suggest that the primary role of colostrum is immunologic rather than nutritional because the immunoglobulins in colostrum provide a great deal of passive immunity that protects the newborn in several ways. Compared to mature milk, both colostrum and transitional milk have more calories in the form of fat and carbohydrates and appears creamy. Transitional milk is not a discrete entity per se, but rather is the milk produced as colostrum changes to mature milk.

Approximately 24 hours before lactogenesis II is clinically evident, the content of lactate and citrate in breast milk increases, while lesser amounts of sodium and chloride are present. Signs of lactogenesis II include breast fullness, tingling and hardness, leaking, and change of milk color from yellow/creamy to blue/watery.

Mature milk is thinner than transitional milk and has a higher water content. Mature milk is subdivided into foremilk and hindmilk. The foremilk that appears at the beginning of a feed has more carbohydrates, whereas the hindmilk that appears near the end of the feeding session has more fat and lipids.[14] Once lactation is established, women produce approximately 700–800 mL of breast milk per day, although this amount can vary considerably.

The numerous bioactive agents in human milk have remarkable functions that protect and facilitate many aspects of infant health and development. Although a detailed presentation of this interactive physiology between a lactating woman and her breastfed infant is beyond the scope of this chapter, the interested reader can access this material in recent reviews.[16,17]

The Importance of Breastfeeding

Breastfeeding is associated with multiple positive short-term and long-term health outcomes for both the lactating woman and her infant.

The Importance of Breast Milk for the Infant

The body of research that has linked breastfeeding to infant health outcomes is difficult to summarize given the wide diversity of study designs, varied numbers of participants, and presence of confounding social and family factors that can independently affect outcomes. In addition, benefits of breastfeeding are somewhat different in resource-rich versus resource-poor nations.[7] Health benefits that have demonstrated a strong positive dose–response relationship are listed in **Table 35-2** and **Table 35-3**.

Table 35-2	Importance of Breastfeeding for Infants
Benefit	**Percent Lowered Risk (%)**
Any Breastfeeding	
Gastroenteritis	64
Inflammatory bowel disease	31
Obesity	24
Otitis media	23
Type 2 diabetes	40
Sudden infant death syndrome (SIDS)	36 (any breastfeeding > 1 month)
Breastfeeding ≥ 3 Months	
Asthma	26 (if no family history) 40 (if positive family history)
Atopic dermatitis	27 (if no family history) 42 (if positive family history)
Otitis media	50
Type 1 diabetes	30
Breastfeeding > 6 Months	
Acute lymphocytic leukemia (ALL)	20
Recurrent otitis media	77
Upper respiratory tract infection	63

Based on American Academy of Pediatrics. Breastfeeding and the use of human milk. *Pediatrics.* 2012;129(3):e827-e841[1]; Chowdhury R, Sinha B, Sankar MJ, et al. Breastfeeding and maternal health outcomes: a systematic review and meta-analysis. *Acta Paediatr.* 2015;104:96-113[5]; Ip S, Chung M, Raman G, et al. Breastfeeding and maternal and infant health outcomes in developed countries. *Evid Rep Technol Assess.* 2007;153:1-186[6]; Victoria CG, Bahl R, Barros A, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet.* 2016;387:475-490.[7]

Table 35-3	Health Outcomes Associated with Breastfeeding for Women Who Are Lactating
Lower Risk of Disorders	
Breast cancer	
Cardiovascular disease	
Endometrial cancer	
Hypertension	
Ovarian cancer	
Postpartum depression	
Type 2 diabetes mellitus in women who had gestational diabetes	
Rheumatoid arthritis	
Improved Health Benefits	
Lower postpartum blood loss	
Increased childhood spacing	
Social Benefits	
Improved maternal–child relationship (lower risk of neglect or abuse)	

Based on American Academy of Pediatrics. Breastfeeding and the use of human milk. *Pediatrics.* 2012;129(3):e827-e841[1]; Chowdhury R, Sinha B, Sankar MJ, et al. Breastfeeding and maternal health outcomes: a systematic review and meta-analysis. *Acta Paediatr.* 2015;104:96-113[5]; Ip S, Chung M, Raman G, et al. Breastfeeding and maternal and infant health outcomes in developed countries. *Evid Rep Technol Assess.* 2007;153:1-186[6]; Victoria CG, Bahl R, Barros A, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet.* 2016;387:475-490.[7]

In resource-poor nations, breastfeeding substantially lowers rates of infant morbidity, infant mortality, gastroenteritis, respiratory disorders, and diarrheal disease.[7] All-cause mortality is increased when infants who are predominately breastfed are compared to those who are exclusively breastfed (relative risk [RR], 1.48; 95% confidence interval [CI], 1.13–1.92), and is much more dramatically increased when infants who are not breastfed are compared to infants who are exclusively breastfed (RR, 14.4; 95% CI, 6.13–33.9).[18] Although the effects on morbidity and mortality are not as striking in resource-rich nations, breastfeeding is clearly protective against gastroenteritis, respiratory disease, asthma, eczema, and allergic rhinitis.[1,7,18,19] A series of meta-analyses summarizing the oral health effects of breastfeeding found that breastfed infants had half the risk of dental caries and half the risk of malocclusions (imperfect positioning of the teeth when the jaws are closed) compared to nonbreastfed peers.[20,21]

Long-term effects of breastfeeding for children are also apparent, although the strength of the etiologic link between breastfeeding and lowered incidence of disease is not fully known for all the associations that have been identified. Children who are formula-fed have higher rates of morbidity and mortality compared to breastfed infants. Children who are breastfed have a lower risk of developing otitis media (recurrent and nonrecurrent), asthma, type 1 and type 2 diabetes, obesity, upper and lower respiratory tract infections, atopic dermatitis, inflammatory bowel disease, celiac disease, gastroenteritis, childhood leukemias, lymphomas, and sudden infant death syndrome (SIDS).[1,7,22-24]

Human milk is even more important for preterm infants. Preterm newborns who do not receive human milk have significantly higher risks of developing necrotizing enterocolitis, neonatal sepsis, metabolic syndrome in adolescence, and hospital readmission. These neonates also have a higher risk for poorer neurodevelopmental outcomes compared to preterm infants fed human milk.[1,6,7,25,26]

The effects of breastfeeding on cognitive development of the child have not been fully determined. Children who were breastfed perform better on intelligence tests as they mature, but these findings may be confounded by caregiver characteristics such as educational attainment, home environmental factors, and socioeconomic factors that are associated with promoting neurodevelopment. To date, studies of neurodevelopmental outcomes of breastfeeding have not generated conclusive results.[27,28]

The Importance of Breastfeeding for the Woman

Breastfeeding also has important health benefits for women. Breastfeeding induces lactational amenorrhea and, therefore, is associated with increased spacing of pregnancies.[5] The duration of lactational amenorrhea and its effectiveness in protecting against conception is related to how often a woman breastfeeds in the first 6 months postpartum. This topic is reviewed in more detail in the *Family Planning* chapter. Immediately after a woman gives birth, breastfeeding accelerates uterine involution via oxytocin secretion and decreases postpartum blood loss. Over the longer term, women who breastfeed have reduced risks for developing several reproductive organ cancers (e.g., breast, endometrial, ovarian), rheumatoid arthritis, type 2 diabetes, hypertension, hyperlipidemia, and cardiovascular disease.[1,5,29-31] In addition, women who do not breastfeed may have a slower return to their prepregnancy weight, although this cannot be generalized to all women given many confounding variables, such as maternal diet choices and maternal body mass index (BMI) prior to and at the birth of the infant.[1,6]

The relationship between breastfeeding and psychological health appears to be bidirectional. Women with prenatal depression are less likely to initiate breastfeeding, and there is a positive relationship between postpartum depression and shorter duration of breastfeeding. Although some studies have found breastfeeding decreases the risk of postpartum depression, others have not confirmed this finding; thus, a clear understanding of the impact of breastfeeding on perinatal mood disorders remains to be elucidated.[32]

It is also important to look at the effects of breastfeeding on the overall maternal–infant relationship. Oxytocin is a potent neuroendocrine peptide that buffers the stress reactivity of the hypothalamic–pituitary axis and enhances maternal–infant bonding.[33,34] The oxytocin secretion stimulated by breastfeeding, therefore, may partly explain the finding of one longitudinal observational study that women who breastfed are less likely to neglect or abuse their child, even after adjusting for confounding variables.[35]

Barriers to Breastfeeding

Formula feeding is costly to both families and society as a whole. The products needed to formula-feed a child are expensive, even when families receive supplemental financial support from the U.S. Department of Agriculture's Women, Infants and Children Supplemental Food Program (WIC). Moreover, the personal and societal costs of treating diseases that are more likely to develop in infants who are not breastfed lends strong support to national and international breastfeeding initiatives. The low rates of breastfeeding in the face of the unambiguous importance of this practice has driven a great deal of research on both barriers to breastfeeding and interventions that can effectively increase breastfeeding rates.[36-38]

Labor and Birth Interventions That Adversely Affect Breastfeeding

Several interventions that may be routinely used or required during labor and birth may have negative effects on subsequent breastfeeding.[39] Many hospital interventions that were standard practices until just a few years ago (e.g., suctioning at birth, routine separation of the maternal–newborn dyad, and provision of pacifiers or supplemental formula) facilitate formula-feeding and lead to lower breastfeeding rates. These hospital interventions are gradually being eliminated as the body of evidence indicating their negative effects on breastfeeding grows.

Epidural Analgesia

All drugs administered to laboring women cross the placenta. Indeed, the presence of epidurally administered anesthetics and opioids can be confirmed (measured) in the umbilical artery and vein after birth.[40,41] Although epidural analgesia is not associated with lower Apgar scores or neurodevelopmental tests in the first 24 hours postpartum,[42] small studies have found that larger exposures to opioids adversely affect breastfeeding initiation.[43] For example, Beilin et al. randomized women who had successfully breastfed their children in the past to differing doses of fentanyl placed in the epidural space for managing labor pain. The women who received the higher dose reported more difficulty establishing breastfeeding and were more likely to discontinue breastfeeding by 6 weeks postpartum when compared to women who did not have fentanyl added to the epidural (17% versus 2%; P =.005). Although this study involved only 107 women, it used a rigorous methodology and its findings support those from a plethora of smaller studies with varied study designs. This association does not mean that opioid exposure *always* adversely affects breastfeeding. Long-term adverse effects on breastfeeding following intrapartum exposure to opioids has not been conclusively demonstrated. Furthermore, these studies did not always control for intrapartum factors other than epidural analgesia that might independently affect breastfeeding success. For example, factors that increase a woman's need for epidural analgesia, such as prolonged labor or malposition may independently have an adverse effect on breastfeeding.

Breastfeeding difficulty in the first days postpartum may have a negligible effect in women with a strong intention to breastfeed, but a much larger adverse effect in women who have other breastfeeding challenges. The most important intervention to ameliorate possible

negative effects of drugs administered during labor is immediate and uninterrupted skin-to-skin contact for at least the first hour after birth, preferably until the newborn moves to the breast and initiates breastfeeding.

Operative Vaginal Birth

Forceps- or vacuum-assisted deliveries can follow long and difficult labors, leaving women exhausted and in pain—conditions that are detrimental to early breastfeeding initiation. Vacuum-assisted birth is associated with cephalohematoma, which may result in newborn pain. Forceps-assisted birth increases the risk of an impaired suckling reflex if the forceps placement bruises the newborn's facial muscles or nerves.[39]

Cesarean Birth

Several retrospective studies have found an association between cesarean birth and lower rates of breastfeeding. It is not known if the surgery itself has an independent effect on breastfeeding or if the indications for the surgery are the most important factors. Women who have a medically indicated cesarean birth are more likely to have experienced a prolonged labor and longer exposure to epidural analgesia, and are more likely to develop chorioamnionitis. Cesarean births are also associated with more blood loss, which may play a role in delayed onset of lactogenesis II. Women who experience this type of an operative birth often have abdominal incisional pain, which can make comfortable positioning for breastfeeding challenging. Moreover, surgically delivered newborns have a higher risk of developing transient tachypnea, which can then interfere with breastfeeding initiation.

Delayed skin-to-skin contact between mother and newborn due to cesarean section interferes with early breastfeeding initiation in the critical first hour after birth. Every effort should be made to initiate early and uninterrupted skin-to-skin contact after cesarean birth in the operating room, or at least in the recovery area.

Practices That Facilitate Breastfeeding

Several clinical interventions for individual women and system-wide policies that facilitate breastfeeding initiation and continuation have been shown to increase breastfeeding rates.

Immediate Skin-to-Skin Contact After Birth

Immediate and uninterrupted skin-to-skin contact after birth significantly increases breastfeeding initiation rates and breastfeeding duration rates.[44] Because the benefits from this practice are so striking, immediate placement of the newborn in a skin-to-skin position on the mother's chest is increasingly standard practice following vaginal birth and is being adopted in many institutions for women undergoing cesarean birth.[45] Skin-to-skin contact for long periods of time is especially beneficial for preterm infants.

The concept of baby-led, mother-guided physiologic breastfeeding refers to maternity care practices that place the mother and infant together with the newborn's hand's free (**Figure 35-2**). The newborn expresses feeding cues, and the mother responds and guides the newborn into feeding positions; the role of the healthcare provider is to support this interaction. This approach contrasts with practices in which the healthcare provider actively places the newborn in a nursing position.[46]



Figure 35-2 Skin-to-skin physiologic breastfeeding initiation.

Photo courtesy of Robin Schafer, CNM.

Policies That Facilitate Breastfeeding

In 2011, the U.S. Department of Health and Human Services issued *The Surgeon General's Call to Action to Support Breastfeeding*, a document urging clinicians, employers,

researchers, government leaders, and communities to help women meet the *Healthy People 2020* goals for breastfeeding and their personal breastfeeding goals.[3,10] This document included 20 specific actions to be taken to facilitate this goal, including actions directed toward system-wide improvements (**Table 35-4**). Support for breastfeeding can be organized into several categories, including maternity care practices, professional education, professional support, breastfeeding education, support in the workplace, peer-support programs, and social marketing.

Table 35-4 The Surgeon General's Call to Action to Support Breastfeeding: Actions for Healthcare Providers

1. Ensure that maternity practices around the United States are fully supportive of breastfeeding.
2. Develop systems to guarantee continuity of skilled support for lactation between hospitals and healthcare settings in the community.
3. Provide education and training in breastfeeding for all health professionals who care for women and children.
4. Include basic support for breastfeeding as a standard of care for midwives, obstetricians, family physicians, nurse practitioners, and pediatricians.
5. Ensure access to services provided by international board-certified lactation consultants.
6. Identify and address obstacles to greater availability of safe banked donor milk for fragile infants.

Reproduced with permission from U.S. Department of Health and Human Services. *The Surgeon General's Call to Action to Support Breastfeeding.* Washington, DC: U.S. Department of Health and Human Services, Office of the Surgeon General; 2011. Available at: https://www.surgeongeneral.gov/library/calls/breastfeeding/index.html. Accessed August 28, 2017.[3]

Federal support for breastfeeding echoes multiple position statements and recommendations from maternal and child health organizations, including the U.S. Preventive Services Task Force (USPSTF)[36]; the Centers for Disease Control and Prevention[37]; the American Academy of Pediatrics (AAP)[1]; the American College of Nurse-Midwives (ACNM)[4]; and the American College of Obstetricians and Gynecologists.[47] Each of these organizations recognizes breast milk as the "optimal infant nutrition."

Telelactation, in which a woman receives support from a lactation consultant via two-way video on personal mobile devices, is drawing increased interest as well. Such services may offer support 24 hours per day for a fee or monthly subscription.[48]

One of the most well-known interventions designed to facilitate breastfeeding is the global Baby-Friendly Hospital Initiative, launched by the World Health Organization and United Nations Children's Fund.[38] Hospitals that successfully implement the 10 steps outlined in the Baby-Friendly Hospital Initiative are awarded a special designation. The 10 steps focus on maternity care practices and hospital policies that are known to promote breastfeeding duration. Research on the breastfeeding outcomes associated with the Baby-Friendly Hospital Initiative has used multiple methodologies, designs, and populations. This heterogeneity makes it difficult to assess the body of literature. In short, research on the Baby-Friendly Hospital Initiative appears to improve breastfeeding duration and breastfeeding exclusivity but the

effect of this package of interventions on breastfeeding initiation has not been clearly established.[49] More information about the Baby-Friendly Hospital Initiative and effective breastfeeding interventions can be found in the Resources section at the end of this chapter.

Contraindications to Breastfeeding

While uncommon, some situations and conditions make breastfeeding inadvisable. Maternal and neonatal medical conditions in which breastfeeding is contraindicated are listed in **Table 35-5.**[1,38,50-53]

Table 35-5	Conditions in Which Breastfeeding Is Contraindicated or Should Be Temporarily Suspended
Disease/Disorder/Medication	**Breastfeeding Recommendation**
Neonatal Disorders	
Galactosemia	Contraindicated
Glucose-6-phosphate dehydrogenase (G6PD) deficiency	If breastfeeding, the woman should avoid fava beans, nitrofurantoin, primaquine, and phenazopyridine to avoid inducing hemolysis in the infant
Phenylketonuria	May partially breastfeed if this practice is alternated with modified formula feeding and monitored
Maternal Infectious Diseases	
Brucellosis, untreated	Contraindicated
Cytomegalovirus	Individualized based on infant circumstances
Herpes, active lesion on breast	Infant may be given expressed breast milk, as the infectious agent is not transmitted through breast milk
Human immunodeficiency virus (HIV) positive	Developed nations: Contraindicated Developing nations: Benefits of breastfeeding may outweigh risk of infection
Human T-cell lymphotropic virus (HTLV) 1 or 2 infection	Contraindicated
Maternal Infectious Diseases	
Influenza, H1N1	Separate infant and mother if possible Infant may be given expressed breast milk, as the infectious agent is not transmitted through breast milk
Tuberculosis, untreated	Infant may be given expressed breast milk, as the infectious agent is not transmitted through breast milk
Varicella, if infection develops 5 days before birth to 2 days after birth	Separate infant and mother Infant may be given expressed breast milk, as the infectious agent is not transmitted through breast milk
Maternal Medications[a,b]	
Alcohol	Occasional alcohol use is acceptable, but avoid nursing for 2 hours after drinking
Amiodarone (Cordarone)	Contraindicated; may cause infant hypothyroidism
Anticonvulsants	Phenobarbital (Luminal), ethosuximide (Zarontin), and primidone (Mysoline) can have high concentrations in breast milk and may cause infant sedation; physician consultation is recommended for women who need any anticonvulsant medication
Antiretroviral medications	Contraindicated
Chemotherapy agents for cancer (e.g., antimetabolites that interfere with DNA	Discontinue breastfeeding for as long as these agents are in breast milk

replication and cell division such as methotrexate)	
Chloramphenicol (Chloromycetin)	Requires close monitoring of blood levels in the woman and infant to prevent bone marrow suppression
Codeine or tramadol-containing analgesics	FDA recommends that breastfeeding women not use codeine-containing medications or tramadol.[51] ACOG states codeine-containing drugs and tramadol may be used if needed in the first postpartum period. If an opioid is required, use the lowest dose possible and monitor the breastfed infant for signs of toxicity.[51] Contraindicated if the woman is known ultra-metabolizer
Cyclosporine (Neoral)	Requires close monitoring of blood levels in the woman and infant
Drugs of abuse	Cocaine, heroin, and marijuana are found in high concentrations in breast milk and associated with delays in infant development
Ergotamines (e.g., dihydroergotamine [Migranal])	Contraindicated
Iodine	Can cause hypothyroidism in infant
Metronidazole (Flagyl)	Controversial. Metronidazole (Flagyl) is excreted into breast milk in relatively high concentrations—approximately 20% of the maternal dose. This drug is a known mutagenic agent in animals. The American Academy of Pediatrics recommends discontinuing breastfeeding for 12–24 hours after a single 2-g maternal dose. Other authors have stated doses in the range of 200–400 mg/dose are compatible with breastfeeding. There are no reports of adverse effects on breastfeeding infants.
Lithium (Lithobid)	Requires close monitoring of blood levels in the woman and infant
Maternal Medications[a,b]	
Radiation therapy (requires temporary cessation)	Discontinue breastfeeding for as long as radioactivity is in breast milk
Retinoids (e.g., isotretinoin [Accutane])	Contraindicated
Statins (e.g., simvastatin [Zocor])	Contraindicated
Sulfa (e.g., sulfamethoxazole [Bactrim])	Should be avoided if the infant is premature, jaundiced, or has G6PD deficiency

Abbreviations: ACOG, American College of Obstetricians and Gynecologists; FDA, U.S. Food and Drug Administration.

[a] This table is not comprehensive. Access current information about breastfeeding effects of drugs not on this list at the National Library of Medicine's LactMed website (https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm).

[b] Additional medications for which the effects on a breastfed infant are unknown include anxiolytics such as lorazepam (Ativan), antipsychotics, and antidepressants.

Based on Centers for Disease Control and Prevention. When should a mother avoid breastfeeding? Available at: https://www.cdc.gov/breastfeeding/disease/index.htm. Accessed March 14, 2017[50]; U.S. Food and Drug Administration. FDA Drug Safety Communication: FDA restricts use of prescription codeine pain and cough medicines and tramadol pain medicines in children; recommends against use in breastfeeding women. Silver Spring (MD): FDA; 2017. Available at: https://www.fda.gov/Drugs/DrugSafety/ucm549679.htm. Retrieved April 24, 2017[51]; American College of Obstetricians and Gynecologists. Practice advisory on codeine and tramadol in breastfeeding women. April 27, 2017. Available at: https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-on-Codeine-and-Tramadol-for-Breastfeeding-Women. Accessed December 28, 2017[52]; American Academy of Pediatrics, Committee on Drugs. Transfer of drugs and other

chemicals into human milk. *Pediatrics.* 2001;108(3):776-789.[53]

Alcohol, Tobacco, and Marijuana

Little recent research has reviewed tobacco use and breastfeeding. The Centers for Disease Control and Prevention (CDC) and other professional organizations recommend that women breastfeed even if they continue smoking during pregnancy or restart tobacco use after birth. Although nicotine is found in breast milk, no studies attribute infant health problems to maternal tobacco use during breastfeeding. If a woman does plan to smoke, she is advised to smoke after breastfeeding and to smoke outside the home to limit the infant's exposure to secondhand smoke, which is known to have detrimental health effects.

The effects of alcohol use during pregnancy are well documented. but few studies have examined alcohol use during breastfeeding. Alcohol diffuses easily into breast milk, such that concentrations approximate maternal blood alcohol level. Alcohol inhibits the milk ejection reflex, temporarily decreasing milk production. Women are advised to wait approximately 2 hours after consuming alcohol before breastfeeding or to wait to partake of alcohol until just after breastfeeding.[54]

Marijuana increasingly is available for medicinal or recreational use. Because marijuana has been legalized for recreational use in some states, women may equate marijuana with alcohol and consider it safe to use during breastfeeding. Marijuana is lipophilic and accumulates in breast milk very easily. Moderate amounts of delta-9-tetrahydrocannabinol (THC) have been found in breast milk.[55] Few studies have followed infants of breastfeeding women who regularly use marijuana; however, extrapolation of data following in utero exposure and data on the effects of marijuana on adult brains suggest that marijuana use is not recommended for women who are breastfeeding.[55]

Pharmaceuticals and Breastfeeding

When considering use of drugs in pregnancy, four key questions need to be considered: (1) How much of the drug transfers into breast milk? (2) If the drug does transfer to breast milk, does it have an adverse effect on the infant such that it is contraindicated or must be used with specific cautions? (3) Does the drug inhibit breast milk production? and (4) Is the drug a galactagogue (an agent that stimulates breast milk production)? The principles of prescribing and basic pharmacokinetic and pharmacodynamic considerations for medications are reviewed in the *Pharmacotherapeutics* chapter. Website resources for assessing the safety of pharmaceutical use during lactation are listed in the Resources section at the end of this chapter.

Transfer of Drugs into Breast Milk

Alveolar lactocyte cells line the alveoli that make up the glandular secretory tissue within the breast. During the first few weeks after birth, large gaps exist between alveolar cells in the maternal breast alveoli, facilitating easy transfer of various agents, including immunoglobulins. As milk production is established and the alveolar cells expand, the intracellular gaps close, so that drug transfer becomes more difficult.

Several pharmacologic relationships play key roles in drug transfer into breast milk:[56]

- Drugs that are highly bound to plasma proteins, such as ibuprofen (Advil), are less likely to transfer to breast milk compared to drugs that are less protein-bound.
- Drugs that move from plasma to tissue rapidly reduce the free drug concentration in plasma, thereby lowering the chance of diffusion into breast milk. Methylprednisolone (Medrol), for example, works within cells and quickly moves into maternal tissue.
- Highly lipid-soluble drugs pass easily into breast milk and may accumulate in milk fat. Opioids, for example, may be found in high concentrations in breast milk.
- High-molecular-weight molecules such as heparin and insulin do not pass easily into breast milk.
- Some drugs cross membranes in their un-ionized form. Breast milk is slightly acidic compared to plasma, allowing highly basic drugs to be ionized and concentrated in breast milk. Thus, weak bases such as codeine and oxycodone may become ionized in breast milk and "trapped" in the milk, further increasing their concentrations in breast.
- Active transport is used by a small number of drugs (e.g., ranitidine [Zantac], nitrofurantoin [Macrobid], acyclovir [Zovirax]).

The method of calculating breast milk concentrations of drugs that is the most clinically useful is the relative infant dose (RID). The RID is the maximum percentage of the maternal dose that the infant consumes assuming a milk intake of approximately 150 mL/kg/day and a standard therapeutic maternal dose. An RID of less than 10% is considered safe for healthy term infants.[56] The National Library of Medicine's LactMed Database uses the RID calculation. Information about LactMed and RID calculations can be found in the Resources

section at the end of this chapter.

Fortunately, most drugs commonly used by lactating women are compatible with breastfeeding. Although more research is needed regarding many agents, drug transfer is rarely a reason to discontinue breastfeeding, and the midwife should be encouraged to seek out the most current and reliable information when counseling a woman about the safety of this practice. **Table 35-6** summarizes guidelines for breastfeeding and the use of medications.

Table 35-6 **General Guidelines for Breastfeeding and Medication Use**

1. Use pharmaceuticals only when necessary.
2. Use the smallest therapeutic dose for the shortest time that is effective.
3. Whenever possible, choose an agent with a short half-life, an agent with a relative infant dose that is less than 10%, and an agent that is not delivered by sustained release.
4. Arrange the timing of drug dosing so that it is given immediately after nursing or before the infant has a long sleep period.
5. If the drug is contraindicated for infant exposure, pumping and discarding the breast milk may be necessary.
6. It is rare that a medication is a reason to discontinue breastfeeding.

Drugs That Are Contraindicated During Breastfeeding

In 2001, the American Academy of Pediatrics Committee on Drugs published an evidence-based listing of drug information for breastfeeding.[53] This document has stood the test of time and is still considered one of the most evidence-based resources for use of drugs by breastfeeding women. The 2013 update to this document expanded on selected topics such as antidepressants, illicit drugs, analgesics, and vaccines. Table 35-5 summarizes the considerations for the drugs that are most commonly encountered in primary care and are contraindicated during breastfeeding, as well as the drugs that may be used with specific cautions.[38,55-59]

Drugs That Inhibit Milk Production

It has long been believed that contraceptive doses of estrogens suppress milk production. However, research has shown that estrogen doses greater than 30 mcg per day are needed to suppress lactation; the doses in current contraceptive formulations are likely to be smaller than this amount, so they are not associated with lactation suppression. The current CDC guidelines for use of estrogen-containing contraceptive products by breastfeeding women are listed in the *Postpartum Care* chapter.[60] These recommendations reflect the results of a detailed expert review of this literature that included consideration of the importance of breastfeeding and the theoretical risks of estrogen and progesterone in relation to the timing of lactogenesis. **Table 35-7** lists drugs that may inhibit breast milk production.

Table 35-7	Drugs That May Inhibit Breast Milk Production

Generic (Brand)
Bromocriptine (Parlodel)
Cabergoline (Dostinex)
Estrogen-containing contraceptives[a]
Ergotamine (Cafergot)
Progestins[a]
Pseudoephedrine (Sudafed)
Testosterone
Tamoxifen (Nolvadex) and other estrogen-receptor blockers

[a] See the CDC medical eligibility criteria for timing of initiation of estrogen or progestin containing contraceptives: Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep.* 2016;65(RR-3):1-104.[60]

In the past, drugs such as bromocriptine (Parlodel) that inhibit milk production were used to suppress lactation for women who cannot or chose not to breastfeed. The evidence for effectiveness for these agents is weak, however, and possible adverse effects have not been well characterized. These agents are not recommended for use in clinical practice.[61] Nonpharmacologic methods to ease the comfort of breast engorgement include breast binding, wearing a tight brassiere, and ice packs.

Galactagogues

When lactogenesis is not well established and conservative management techniques have failed to increase milk supply, *galactagogues*—drugs that can stimulate breast milk production—may be considered. Pharmaceutical galactagogues are dopamine antagonists that increase prolactin levels.[62] The evidence for effectiveness of galactagogues is limited, however, and they should be used cautiously given the possible side effects associated with use of these drugs.

Domperidone has been shown to significantly increase milk production, but is not available in the United States. The Food and Drug Administration (FDA) has recommended that domperidone not be used because it can cause QT-segment elevation on electrocardiogram during intravenous therapy, which in rare cases may be fatal.[63]

Metoclopramide (Reglan) is a dopamine antagonist used for gastrointestinal indications. A side effect of the drug is galactorrhea; thus, it is used on an off-label basis to help women increase their milk supply. Metoclopramide has a number of possible side effects, including fatigue, mental exhaustion, and extrapyramidal effects, so it should be used for only a limited time.[64] More severe side effects of this agent include tardive dyskinesia (involuntary repetitive movements such as grimacing) and dystonia, which warranted the FDA “black box” warning on the drug’s label, advising against prolonged use or high doses of the drug. Metoclopramide has not been shown to be more effective than a placebo.[64]

Herbal galactagogues such as fenugreek (*Trigonella foenum-graecum L.*) have been used

for years and are well accepted in many countries, yet data demonstrating their efficacy remain elusive. Herbal preparations should be considered with caution, as these products are not standardized, may be contaminated, and may interact with prescription drugs.[65]

Analgesics for Postpartum Pain Relief

Pain following episiotomy, laceration repair, operative vaginal birth, or cesarean birth may decrease a woman's comfort and desire to breastfeed. Conversely, pain control with opioid medication may sedate a mother and newborn and decrease feeding. Nonpharmacologic methods of pain reduction, such as cold or warm packs, may be helpful in this regard.

Guidelines for use of opioids during the immediate postpartum period are presented in the *Postpartum Care* chapter.[66] If opioids are required for pain relief in the immediate postpartum period, the lowest dose possible for the shortest duration possible should be used as part of a multimodal, stepwise process to ensure optimal pain relief.[52] Although the FDA has recommended that codeine and tramadol (Ultram) not be used by breastfeeding women,[51] there are occasions when opioid medications are needed. If a breastfeeding woman requires opioids for pain control, physician consultation, a process of shared decision making, and careful documentation in the healthcare record are recommended.

Nonsteroidal anti-inflammatory drugs (NSAIDs) are compatible with breastfeeding. Low or no amounts of ibuprofen appear in breast milk following 400 mg doses every 6 hours, making this agent an ideal postpartum pain reliever and antipyretic.

Antidepressant, Anxiolytic, and Antipsychotic Medications

The effects of antidepressants on a breastfeeding infant are not fully known. Most antidepressants appear in breast milk in low concentrations, and many can be taken safely during lactation.[57,67] The medications in this class that have an RID of 10% or higher include bupropion (Wellbutrin), diazepam (Valium), fluoxetine (Prozac), citalopram (Celexa), lithium (Lithobid), lamotrigine (Lamictal), and venlafaxine (Effexor). When a breastfeeding woman requires a medication in one of these classes, the half-life, duration of action, consequences of discontinuing the medication, and factors involved in changing from one medication to another should be taken into consideration. Consultation with obstetric or psychiatric providers is recommended.

Assessment of the Maternal–Newborn Dyad During Breastfeeding

The goal of breastfeeding assessment is to evaluate breastfeeding effectiveness and to detect problems that may lead to inadequate milk transfer or breastfeeding cessation. Although the initial breastfeeding assessment should be performed prenatally, the assessment is repeated or may be performed for the first time prior to the initial assessment of the breastfeeding session. A breastfeeding assessment is performed during evaluation of any breastfeeding concern and always includes evaluation of both the woman and the newborn (**Table 35-8**).

Table 35-8	Postnatal Breastfeeding Assessment
Breastfeeding Intention	
If the woman is planning to breastfeed, the following history is obtained.	
Assessment of the Woman	
History	
History of conditions that may adversely affect breastfeeding, including polycystic ovary syndrome, diabetes, or thyroid disorder	
History of breast surgery including mastectomy, lumpectomy, augmentation, or reduction	
Prior unsuccessful breastfeeding	
Current use of opioids	
Physical Examination of the Breast	
Breast: engorgement (yes/no); any scars or abnormalities	
Nipple type (retracted, inverted-appearing, common, flat), injury (cracks, abrasions, blisters, signs of infection)	
Nipple lesions: cracks or blisters	
Presence or absence of breast tissue in axilla and along milk lines. If present, this extra-mammary tissue may engorge during lactogenesis II and the woman can be counseled about expected findings if engorgement occurs.	
Assessment of the Newborn	
History	
Gestational age at birth	
Mode of birth: Operative assisted birth with forceps or vacuum may cause trauma such as cephalohematoma or bruising.	
Labor medications and duration of exposure	
Neonatal complications and/or interventions	
Voiding frequency, stool frequency, stool consistency	
Physical Examination	
Weight	
Head (cephalohematoma)	
Neck: range of motion or signs of discomfort with range of motion	
Mouth to rule out ankyloglossia[a] or thrush	

[a] Ankyloglossia (tongue-tie) is a condition in which the lingual frenulum is short or thick, such that the infant cannot

extend the tongue optimally. Although ankyloglossia has been associated with breastfeeding difficulties and nipple pain, studies of frenotomy have not consistently shown it is effective in improving breastfeeding duration. Routine examination of the frenulum is not necessary as part of an initial breastfeeding assessment.

Table 35-9 illustrates several types of nipples.[68] Most breast sizes, shapes, and configurations are compatible with breastfeeding; a few may pose temporary or permanent challenges. Flat or inverted nipples may be a challenge for the newborn to grasp; however, prenatal preparation techniques such as nipple rolling do not improve early breastfeeding. Regardless of shape or configuration, the important consideration is whether milk can be expressed and the baby can extract milk.

Table 35-9 Basic Types of Nipples

Type of Nipple	Before Stimulation	After Stimulation
The majority of women have what is referred to as a **common nipple**. It protrudes slightly when at rest and becomes erect and more graspable when stimulated. An infant has no trouble finding and grasping this nipple to pull in a large amount of breast tissue and stretch it to the roof of the mouth.		
The **flat nipple** has a very short shank that makes it less easy for the infant to find and grasp. In response to stimulation, slight movement inward or outward may be present, but not enough to aid the infant in finding and initially grasping the breast on center.		
An **inverted-appearing nipple** may appear inverted but becomes erect and easily graspable after stimulation.		
The **retracted nipple** is the most common type of inverted nipple. On stimulation it retracts, making attachment difficult.		
The truly **inverted nipple** is retracted both at rest and when stimulated. Such a nipple is very uncommon and more difficult for the baby to grasp.		

Reproduced with permission from International Lactation Consultant Association; Mannel R, Martens PJ, Walker M, eds. *Core Curriculum for Lactation Consultant Practice*. 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2013.[68]

Several breastfeeding assessment tools have been developed and tested.[69] To date, none has proved robust enough to be reliably used in clinical practice among all populations. A breastfeeding assessment of the maternal–infant breastfeeding dyad includes evaluation of five aspects of the breastfeeding session: (1) feeding cues in the infant, (2) latch-on and infant suckle, (3) transfer of milk, (4) maternal comfort, and (5) signs of infant satiety.[70]

Feeding Cues

Neonates are able to cue, suck, and swallow within a few minutes after birth (**Table 35-10**).[70] Newborns use touch, sight, smell, and hearing to locate the breast and move toward it with reflexive and deliberate motions. If these early cues go unheeded, the infant will escalate to more intense cues, which are also stress cues: hands and fists move toward the mouth, increased rooting, and whimpering and other vocalizations of distress.

Table 35-10 **Infant Feeding Cues or Prefeeding Behavior**

Feeding Cue	Timing of Cue	Description
Increasing alertness	Early	Especially rapid eye movement under closed eyelids
Rooting	Early	Turning the head, especially with searching movements of the mouth
Flexing	Early	Legs and arms
Flexed arm or closed fist	Early	May bring hand toward mouth
Sucking	Early, mid	Fist or finger
Mouthing	Early	Motions made with the lips and tongue
Fussing or restlessness	Mid	Crying or squirming
Full cry	Late	Turns red, high-pitched scream

Modified with permission from Cadwell K. Latching-on and suckling of the healthy term neonate: breastfeeding assessment. *J Midwifery Womens Health.* 2007;52(6):638-642.[70] © 2007, with permission from Wiley.

Infants are more likely to breastfeed well when in the light sleep or active alert state of consciousness. Many infants become drowsy and fall asleep while nursing. It can be difficult to rouse an infant from a deep restorative sleep state specifically for the purposes of breastfeeding. Although most parents quickly learn their infant's hunger cues, these cues can be reviewed with new parents as needed.[71]

Latch-on and Infant Suck

Deep latch onto the breast is fundamental to adequate milk transfer and maternal comfort. First, the mother should find a comfortable position for a 30-minute feeding session. Upright, semi-reclining, and fully reclining on her side are all common and comfortable positions.[72] The goal is for the infant to be close to the breast and the mother's body well supported so she can relax while breastfeeding. The infant should face the mother's breasts directly and be supported by her arms or hands.

Most infants can move to the breast and self-attach if the mother is in a semi-reclining position and the infant is lying prone on the woman's body with access to her uncovered breasts. If the angle of the infant's open mouth can be seen at all, it should be greater than 90° (usually 130–150°) with both lips flared outward. If the woman is upright, she can bring the alert infant toward her breast, watching for a wide-open gape, as shown in **Figure 35-3**.[72]

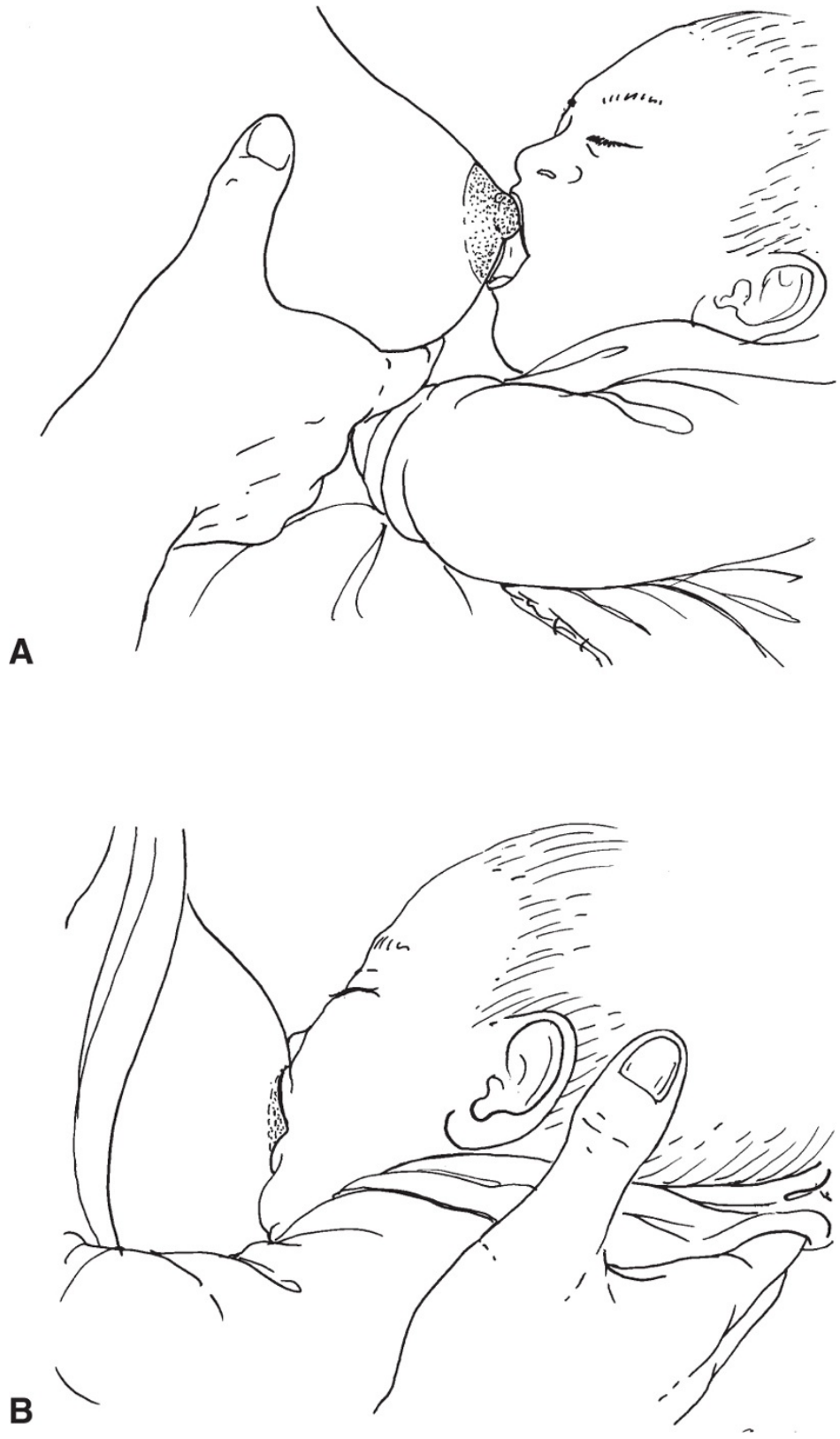


Figure 35-3 Latch-on. **A.** Mouth gaped wide open. **B.** Moving onto the breast.

Reproduced with permission from Wambach KA, Riordan J. *Breastfeeding and Human Lactation.* 5th ed. Burlington, MA: Jones & Bartlett Learning; 2016.[73]

The infant uses lips, tongue, oral cavity, jaw, facial muscles, and buccal fat pads to generate both negative and positive pressure. Positive pressure comes from the tongue moving in a

wave-like peristaltic motion, stripping the milk back toward the pharynx. Negative pressure comes from the jaw first dropping to enlarge the oral cavity and allow the milk to flow in a bolus during the let-down reflex, then rising to allow swallowing and a breath. The mother's nipple may stretch to twice its resting length inside the infant's mouth, with the tip resting near the hard–soft palate juncture.[74] The infant sucks at a rate of approximately 1.2 sucks per second.

Positions that can be used for breastfeeding are described in **Table 35-11**.[68]

Table 35-11 **Common Positions for Breastfeeding**

Position	Description	Advantages	Illustration
Cradle or Madonna position	The woman sits upright. The infant is side-lying, facing the mother; the infant nurses from the same breast as the woman's supporting arm.	Commonly used and feels natural, especially for older infants. Small infants may need more head/shoulder support.	
Cross-cradle posture	The woman sits upright. The infant is side-lying, facing the mother; the infant nurses from the opposite breast as the woman's supporting arm.	Useful for nursing a small or preterm infant; provides more stability for the infant's upper body and head.	
Football or clutch (underarm)	The woman is upright. The infant's feet are pointed toward the woman's back; the infant is supine on the woman's supporting arm or next to her body, and nurses from the same breast as the supporting arm.	Useful for women with large breasts or post-cesarean birth.	
Semi-reclining ("laid back")	The woman leans back and the infant lies prone on her chest.	Gravity helps both woman and infant. The infant can self-attach and get a deep latch, and the woman can relax without concern that the infant will fall away from her body.	
Side-lying, or "recovery" position	The woman lies on her side. The infant is side-lying facing the mother's breast. The woman's bottom arm is over or under the infant's head; the top arm is around the infant's legs or body.	The woman can fall asleep safely while the infant is nursing.	
Australian, or baby on top	The woman is "down-under," lying on her back, with the infant supported on her chest.	The infant can get a deep latch but may need head support. Useful if the woman has forceful milk ejection.	

Modified with permission International Lactation Consultant Association; Mannel R, Martens PJ, Walker M, eds. *Core Curriculum for Lactation Consultant Practice.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2013.[68]

Assessment of Milk Transfer During Breastfeeding

Breastfeeding has a visible and audible rhythm, driven by the infant's ability to coordinate the suck, swallow, and breathe actions.[75] At the start of a feed, there is a faster suck–swallow–breathe rhythm, with the infant taking several sucks before swallowing and breathing. When the milk ejection reflex (milk "let-down") occurs, milk flows more rapidly in a bolus for

approximately 1–2 minutes and the infant responds with a 1:1:1 suck–swallow–breathe rhythm. As the flow slows, the infant reverts to more sucks before a swallow and breath. Most women experience at least two milk ejection reflexes per feeding session, even if they perceive only the first one. During the milk ejection reflex, the infant's suck–swallow–breathe pattern slows and deepens to adapt to the fast-flowing, large bolus of milk. As the milk flow slows, the infant reverts to a lighter, faster pattern with more sucks per swallow until the next milk ejection reflex occurs. The infant may stay on the breast between milk ejection reflexes and rest. The infant will release the breast comfortably when satiated.

Maternal Comfort During Breastfeeding

Physical comfort of the woman is paramount when initiating breastfeeding and implies more than a comfortable physical posture. The breastfeeding woman should have all things she may find she needs within easy reach, such as reading materials, glasses, water, or telephone. She also may find it helpful to have the aid of breastfeeding devices such as a specially designed pillow to rest the infant on during breastfeeding (although other pillows may work equally as well). Post-cesarean or operative vaginal delivery pain relief may be needed for the woman to be comfortable.

Many women will feel nipple pain or pinching during the initial breastfeeding. Pain may be caused by poor latch, nipple trauma, or infection. Engorgement gives a sensation of fullness that becomes achy and throbbing if the breast is not emptied. Emptied breasts feel soft and pain-free between feedings.

Signs of Infant Satiety

Breastfed infants feed frequently, which is normal and consistent with their physiology and the composition of human milk. Healthy infants can be fed on demand, because frequent milk removal is the most important mechanism regulating ongoing breast milk synthesis. Infants fed "on cue" usually nurse 8 to 12 times during each 24-hour period. In the first month, infants need approximately 20–24 ounces of breast milk per day, and their stomach capacity is less than 1 ounce.[76]

Some infants tend to have a pattern of paired or cluster feeds—that is, two or more feeds, with short periods of sleep or a quiet alert state between feeding cues. Gradually, over the first few weeks after birth, most infants fall into more predictable patterns. In one study, approximately 13% of infants always had paired feeds; approximately 30% fed from only one breast per feed, and the remaining 57% mixed paired and single-side feeds.[77] In the first 6 months, exclusively breastfed infants who were fed "on cue" had 11 ± 3 feeds per 24 hours, taking 76 ± 12.6 g per feed (range, 0–240 g).[77] Formula-fed infants feed slightly less frequently than breastfed infants.

Weight Gain and Elimination Patterns as Signs of Effective Breastfeeding

Weight gain, frequent voids, and stool patterns are reliable indicators of thriving infants.[78] If

there is a question or concern about effective feeding, a pre- and post-feed weight check (infant in the same clothing and diaper) on a sensitive scale can verify that milk transfer occurred based on the infant's increased weight after feeding. Breastfed infants gain an average of 4–7 ounces per week, and often alternate linear growth with weight gain.

Newborns may void only a few times in the first days after birth, before lactogenesis is established. Once breastfeeding is established, infants who are receiving sufficient nutrition will void frequently, approximately 6–8 times per day.[78] Breastfed infants may have stools after every feeding. The frequency and consistency change as breastfeeding is established. Normal newborn stool patterns are reviewed in the *Anatomy and Physiology of the Newborn* chapter.

Breastfeeding Problems

Breastfeeding rates decline the most in the first month after birth. The most common reasons women give for stopping breastfeeding are insufficient milk production, pain and difficulty with infant suckling, and lack of maternal confidence.[3,78-82] Most of these concerns can be resolved with some assistance, education, and/or support.[3] Helping women resolve breastfeeding concerns may be one of the midwifery care practices that has the largest beneficial effect on the health of women and their families.

Table 35-12 presents cues, triggers, and teaching points for the common concerns that women voice regarding breastfeeding.[81] **Table 35-13** summarizes signs and symptoms of common breast conditions reviewed in this chapter as an aid for developing a differential diagnosis.[82]

Table 35-12 Touch Points for Overcoming Obstacles to Breastfeeding Exclusivity

Time Point	Parental Concern	Main Obstacle	Provider Advice
Prenatal	"I want to breastfeed, but since I am going to work, I need to be able to formula-feed, too."	Lack of information about combining breastfeeding and working	Strongly encourage attendance at a prenatal breastfeeding class (deserves equal time to birthing class education)
		Lack of information about milk expression and access to breast pumps	Consider a longer maternity leave, if possible Prepare to simplify life during the transition to parenting
	"My husband/partner and other family members will want to help feed the baby. Won't they feel excluded if I only breastfeed?"	Family members wanting to feed the baby	Enlist father's/partner's help in supporting the nursing partner; fathers/partners can interact with their newborn by holding the baby skin-to-skin or taking the baby out while the mother sleeps After breastfeeding is well established, others can feed the baby expressed milk by bottle
	"I want to do combination feeding, or las dos cosas."	Desire for "the best of both worlds" by combination feeding	"Puro pecho," or only mother's own milk, provides greater health benefits and helps maintain an abundant milk supply
		Lack of knowledge about the importance of frequent and exclusive breastfeeding during the early postpartum weeks for establishing the mother's milk supply	If eligible, enrollment in the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) offers breastfeeding mothers a substantial food package, counseling, breast pumps, and peer counselors
Birth	"My friend says it is a good idea to ask the nurses to care for my baby at night so I can get some sleep."	Unrealistic expectations for the postbirth hospital stay	Promote immediate skin-to-skin contact after birth to facilitate the initiation of breastfeeding within the first hour
		Lack of prenatal education	Teach the mother to interpret her newborn's feeding cues and breastfeed as often as baby wants; advocate for no routine formula use in the system of care
		Frequent interruptions and excessive visitors deplete new mothers' energy	Advise the mother to request help in the hospital with breastfeeding to promote task mastery
		Increased risk of formula supplements for nighttime births from 9 PM to 6 AM	Encourage continuous rooming-in, where the mother can practice being with her baby in a controlled setting and learn to latch the baby comfortably and effectively

	"The yellow milk does not look like much. A little formula won't hurt, will it?"	Belief that the small amount of colostrum is insufficient until the "milk comes in"	Explain the potency and adequacy of colostrum and the rapid increase in milk production from 36 to 96 hours postpartum
3–5 days	"Now that we are home from the hospital, the baby seems to be feeding every hour. She or he doesn't seem satisfied with breast milk alone."	Lack of knowledge about normal frequency of feedings for breastfed newborns	Explain that 8–12 feedings in 24 hours are typical and necessary to establish an abundant milk supply
		Newborns typically begin feeding more frequently the second night after birth, when the baby is at home	Provide a hand-pump or teach hand expression, so the mother can see that she has milk
		Concern about whether the newborn is getting enough milk, due to the mother's inability to see what the newborn takes at the breast	Explain normal newborn elimination patterns once the mother's milk comes in (3–5 voids and 3–4 stools per day by 3–5 days; onset of yellow, seedy milk stools by 4–5 days)
		Sleepy newborn	Perform newborn test weights (before and after feeding) to reassure the mother about baby's milk intake at a feeding Teach the mother the difference between newborn "flutter sucking" or "nibbling" that results in only a trickle of milk at the breast versus "drinking" milk, with active sucking and regular swallowing Tickling under the axilla or holding a hand up can help keep baby on task at the breast; compressing the breast when the baby stops slow, deep sucking can deliver a spray of milk to entice him or her to start drinking again Anticipate a newborn appetite spurt at approximately 10–14 days of age
	"My nipples are sore and cracked. Can I take a break and give my baby a little formula?"	Sore nipples are usually attributable to incorrect latch-on technique and are a common reason that mothers discontinue breastfeeding early or start supplements	Observe a nursing session to evaluate latch; consider referring the mother to a lactation consultant for one-on-one assistance with latch
2 weeks	"My breasts do not feel very full anymore. I'm afraid my milk went away."	As postpartum breast engorgement resolves and the breasts adjust to making and releasing milk, mothers may perceive that they have insufficient milk supply	Expect the newborn to be above birth weight by 10–14 days and reassure the mother about the newborn's rate of weight gain since the 35-day visit Although the mother's breasts are less swollen than during postpartum engorgement, they should feel fuller before feedings and softer afterward
	"How can I know my baby is getting enough?"	The days 10–14 appetite spurt can cause the mother to doubt the adequacy of her milk supply	Consider performing test weights (before and after feeding) to reassure the mother about her newborn's intake Anticipate another appetite spurt at approximately 3 weeks of age
1 month	"My baby is crying a lot, and I am tired and need sleep."	Normal infant crying peaks at approximately 6 weeks (3–5 hours in 24 hours)	Congratulate the mother on a full month of breastfeeding!
		The mother may attribute infant crying to hunger or an adverse reaction to her milk	If the infant has gained weight appropriately, reassure the mother about the adequacy of her milk supply Offer coping strategies for infant crying, including holding the baby skin-to-skin; the Five S's[a] (swaddle with the baby's hands up near the head to help assess feeding cues); use of an infant carrier; going for a stroller or car ride; observing a period of "purple crying"
	"Nothing seems to calm my baby except the bottle."	If the infant drinks milk from a bottle that is offered, the mother may assume that her infant is not satisfied by breastfeeding	Explain that infant sucking is reflexive, and drinking from an offered bottle does not always mean that the baby is hungry; a baby "can't scream and suck at the same time," so the bottle may appear to calm the baby, just as a pacifier might If the mother desires to offer a bottle, use expressed milk as the supplement Forewarn the mother about cluster feedings (late afternoon/evening) and upcoming appetite spurts, occurring at about 6 weeks and 3 months
2 months	"My mother said that, if I give my baby rice cereal in a bottle before bedtime, he or she may sleep longer at night."	Parental sleep deprivation	Explain the lack of evidence that rice cereal or other solid foods increase infant sleep

		The mother may have already returned to work, which often increases fatigue and leads to a decrease in milk supply	Remind the mother that adding complementary foods is a project and increases the workload for parents Reinforce the benefits of exclusive breastfeeding for maternal–infant health and the mother's milk supply
	"I am going back to work and am worried that I do not have enough frozen stores of milk. Are there any herbs I can take to keep my milk supply strong?"	Lack of knowledge about the principles of milk production and unrealistic beliefs about the effectiveness of galactagogues	Enlist help from others, including support for returning to work Explain that there is no "magic pill" or special tea to increase the mother's milk supply; the key to ongoing milk production is frequent, effective milk removal (every 3–4 hours) Caution the mother to avoid going long intervals without draining her breasts
4 months	"My baby seems to eat for only a few minutes, and when I try to put her/him back to the breast, she or he refuses."	Misinterpretation of infant's efficiency in nursing causes concern about infant milk intake	Explain that infants become more efficient at breastfeeding, and by 3 months, they may drain the breast in 4–7 minutes Reinforce continuing to delay the introduction of solid foods
	"My baby seems more interested in everything around him or her than in nursing at the breast."	Normal infant distractibility causes the mother to believe her infant is self-weaning	Explain that distractibility is a normal developmental behavior at this age and that short, efficient feedings are common Nurse in a quiet, darkened room
6 months	"My baby is drooling and rubbing on her or his gums all the time. I do not think that I can continue to breastfeed because my baby might bite me."	Common myth that a mother needs to wean when her baby gets teeth to avoid being bitten while breastfeeding	Congratulate the mother on 6 months of exclusive breastfeeding! Explain that infants cannot bite and actively breastfeed at the same time. Biting tends to occur if the breast is offered when the infant is not interested or at the end of the feeding If the infant bites, say "No biting," touch the infant's lips, set the baby down, and briefly leave the room
	"My baby has refused to breastfeed for almost a whole day now. Is she or he ready to wean?"	Misinterpretation of sudden breastfeeding refusal ("nursing strike") to mean that a baby is self-weaning	Explain that some infants may suddenly refuse the breast between 4 and 7 months of age for no apparent reason; common causes include upper respiratory infection, ear infection, teething, regular exposure to bottle-feeding, use of a new soap/perfume, maternal stress, or a decrease in milk supply Because many infants will nurse while asleep, try offering the breast when the baby is drowsy or asleep Regularly express milk if the baby will not nurse, and feed the pumped milk until the infant resumes breastfeeding

[a] The Five S's consist of swaddling, side-lying, swaying, shushing, and sucking. The Five S's are interventions recommended to help an infant stop crying promoted by Dr. Harvey Karp. Of note, these interventions have not been the subject of research.

Reproduced with permission from Bunik M. The pediatrician's role in encouraging exclusive breastfeeding. *Pediatr Rev*. 2017;38(8):353-368.[81]

Table 35-13 Signs and Symptoms Associated with Common Breast Conditions in Women Who Are Lactating

Condition	Definition	Description	Systemic Symptoms	Fever	Pain	Unilateral vs. Bilateral	Other
Engorgement	Breast is engorged	Hard, enlarged, shiny, bilateral erythema	No, occasional transient fever "milk fever" that resolves within 24–48 hours	< 38.4°C	Yes	Bilateral	Associated with onset of lactogenesis II
Blocked ducts[a]	Lump of localized milk stasis	Single lumpy area; may have erythema	Unlikely	Unlikely	Tender or localized pain	Usually unilateral	May occur with a painful white bleb on the nipple[b]
Galactocele	A milk-retention cyst	Smooth, well-demarcated mass	No	No	Maybe	Usually unilateral	Compression of the cyst may cause milky fluid to exude from the nipple
Inflammatory mastitis	Secondary to ineffective and/or obstructed drainage of milk from the breast	Erythematous	Yes, in 50–66% of women	Mild: < 37.5 °C Acute: > 37.5°C and < 38.5°C Hyperacute: > 38°C	Yes, localized	Usually unilateral	See note[c]
Infectious mastitis	Results from untreated milk stasis and/or colonization with pathogenic bacteria	Hard, may be wedged shaped; may be erythematous	No, in 33–50% of women	Mild: < 37.5 °C Acute: > 37.4°C and < 38.5°C Hyperacute: > 38°C	Usually	Usually unilateral but may be bilateral	See note[c]
Breast abscess	A localized infection that has formed a barrier of granulation tissue	Swollen mass, possibly fluctuant, with erythema	May or may not be present; may follow mastitis	May or may not be present	Usually severe	Often unilateral but may be bilateral	

[a] May be referred to as "focal breast engorgement," "caked breast," or "plugged duct."
[b] The bleb may be opened up with warm water soaks, massage, or a sterile needle.
[c] The clinician cannot reliably differentiate noninfectious mastitis clinically from infectious mastitis. Treat the woman with antibiotics if she is acutely ill, there are severe or bilateral symptoms, there are nipple fissures, or symptom resolution does not occur within 12–24 hours of improved milk removal.

Modified with permission from Betzold CM. An update on the recognition and management of lactational breast inflammation. *J Midwifery Womens Health*. 2007;52:595-605.[82]
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Breast Engorgement

Breast engorgement is a nonspecific term that describes breast hardness and fullness that is associated with pain or discomfort. Primary engorgement occurs in most women at the onset of lactogenesis II and is believed to arise secondary to a combination of milk and interstitial edema.[12] Secondary engorgement can occur at any time if milk is not emptied from the breast. When engorgement is severe, the nipple may flatten and the infant may not be able to effectively latch onto the nipple.

Primary engorgement generally lasts approximately 48 hours and resolves spontaneously. The goals of treatment are to facilitate infant latch and decrease maternal discomfort. If the areola is involved, a small amount of milk can be expressed via hand or a breast pump to soften the areola. The breast does not need to be fully emptied. If more than two-thirds of the retained milk is removed, milk production will increase; conversely, if less than one-third of retained milk is removed, milk production will remain stable or decrease.[12] It is best to allow the infant to control the amount of milk removed from the breast.

Remedies for postpartum breast engorgement remain insufficiently tested.[83] Common treatments include wrapping the breasts tightly to facilitate comfort, ice packs, application of cold cabbage leaves, and use of analgesics such as ibuprofen. Although a popular home remedy, application of cold cabbage leaves does not appear to be more or less beneficial than application of cold packs.[84]

Delayed Lactogenesis II

Delayed lactogenesis describes a longer than expected interval before lactogenesis II occurs. Delayed lactogenesis is distinct from failed lactogenesis or secondary insufficiency of milk production. Each of these conditions is addressed in turn in this section.

The definition of delayed lactogenesis II used clinically and in many research protocols is no obvious signs of milk production 72 hours or more after birth.[85] Nevertheless, this definition may not reflect the normal variability in human physiology. Although most of the studies conducted on timing of lactogenesis II are several years old and rely on small sample sizes, some conclusions can be drawn. Neville et al. found the timing of lactogenesis in multiparous women occurs between 46 and 92 hours postpartum.[86] Arthur et al. reported women detected lactogenesis II between 38 and 92 hours postpartum, with the mean being 74 hours after birth.[87] A more recent study by Brownell et al. (n = 2491) surveyed women in the United States who gave birth to a healthy infant at 35 weeks' gestation or later. In this study, 11% of the participants experienced lactogenesis II at 1 day postpartum or sooner, 66% at 2 to 3 days postpartum, 15% at 4 days postpartum, and 8% after the fourth day postpartum.[88] In summary, the normal timing of the onset of lactogenesis II may occur later than previously believed.

Despite the lack of clear guidelines to define delayed lactogenesis II, some risk factors are well known. Primigravidas are likely to experience lactogenesis II later than multigravidas, with the range reported as approximately 24 hours.[89] Women who have cesarean births may also experience lactogenesis approximately 24 hours later than women who have vaginal births, but do not experience lower quantities of milk production.[88,89]

Delayed lactogenesis can be due to retained placental fragments, diabetes, thyroid disease, preeclampsia, pituitary tumors, Sheehan syndrome, theca-lutein cysts, polycystic ovary syndrome (PCOS), high maternal BMI, and gestational diabetes.[56,88–90] A review of medications that may reduce milk supply may be considered when a woman experiences such a delay. Recently, consuming encapsulated placenta has been suggested as a risk factor in delayed or failure of lactogenesis II.[91]

Obesity and Delayed Lactogenesis

Women with prepregnancy obesity are known to have lower breastfeeding initiation rates, delayed lactogenesis II, and shorter breastfeeding duration.[92] Many factors may contribute to lower rates of breastfeeding initiation in women with high BMIs, including increased use of Pitocin for prolonged labor and increased incidence of cesarean birth. The underlying mechanism of action may be related to higher levels of leptin in this population. Leptin, an adipokine produced in adipose tissue, is a tocolytic agent that may reduce oxytocin's action in the milk ejection reflex.

Milk expression or pumping after feedings in the early postpartum period, breast massage, and warm compresses may enhance early breastfeeding efforts for women with obesity. These women may also benefit from early breastfeeding education, lactation support visits or phone calls, and peer support groups.

Failed Lactogenesis II

Although rare, failed lactogenesis may occur secondary to mammary hypoplasia. While the incidence of mammary hypoplasia has not been studied rigorously, estimates are that fewer than 5% of women have underdeveloped mammary glandular tissue. During physical examination, breasts with insufficient mammary tissue may appear tubular, widely spaced (more than 1.5 inches between the breasts at the sternum), or markedly asymmetrical in size and shape. Breasts with insufficient mammary tissue do not grow—that is, bra cup size is unchanged—during pregnancy.[93]

Many women with mammary hypoplasia will give formula supplements following each feeding or change to exclusive formula-feeding. Women with mammary hypoplasia need reassurance and support as they determine the best choices for infant nutrition.

Insufficient Milk Supply

Women concerned about insufficient milk supply often report infant crying often and lack of fullness in the breasts. Supply and demand is the basic principle governing milk production: The emptier the breast, the faster milk is produced in the alveoli. When the breasts are emptiest, the rate of milk production is approximately 2 ounces per hour per breast. When the breasts are fullest, the rate drops to approximately

ounce per hour per breast, with an hourly average rate of 1 ounce per hour per breast.[94] Common etiologies of insufficient milk supply are not emptying the breast frequently enough and insufficient emptying by the newborn.

Assessment for insufficient milk supply includes obtaining a history of time intervals between nursing, duration of each feeding session, and use of medication that could lower milk production. The infant should be examined and weighed. Observation of a breastfeeding session can detect problems with infant latch. If the infant is nursing well, is gaining weight, and has a normal elimination pattern, insufficient milk supply is unlikely. In this case, sharing information about normal infant developmental growth spurts and expected hours of infant crying in a 24-hour period can help reframe and contextualize infant behavior. The woman can also weigh the infant before and after a feeding session to obtain data showing effective milk transfer. Manual expression or using breast pump after feeding can enhance milk production if necessary.

If the infant has lost weight or has signs of hyponatremia, consultation with a lactation consultant and/or pediatric provider is indicated.

Plugged Ducts and Galactoceles

Plugged ducts are usually attributed to restriction of milk flow in one section of the breast. Encourage women to feed their infants frequently, preferentially offering the affected breast first, to more thoroughly drain the plugged areas. Positioning the infant’s chin toward the blocked area may improve drainage. Manual massage toward the nipple may clear a plugged area. Plugged nipple pores (white bleb) on the tip of the nipple are pinpoint-tender and usually

related to a plugged duct. Gentle massage and warm water soaks will usually relieve the plug.

A galactocele is a milk retention cyst that results from a blocked duct and presents as a cystic, usually nontender mass. Galactoceles are uncommon during lactation, but rather are most likely to develop during the process of weaning. Although these cysts are benign, the differential diagnosis includes breast abscess, lipoma, fibrocystic changes, and breast malignancy. The diagnosis can be made on ultrasound and/or via aspiration. Very rarely, if the galactocele is quite large or painful, repeat aspiration or excision may be recommended.

Conditions That Affect the Nipple and Areola

Pain and dermatoses that affect the nipple and areola can appear in the first days after the infant starts breastfeeding, or they can appear later, after breastfeeding is well established.

Conditions That Appear with the Onset of Breastfeeding: Nipple Trauma

During the first 3 to 4 days of breastfeeding, nipple pain or discomfort is normal for the first 30–60 seconds of nursing. This initial pain dissipates once the infant is firmly latched and suckling effectively. Pain that persists throughout the breastfeeding session or pain that worsens over time needs further evaluation.

In such a case, the nipple should be inspected to identify cracks, abrasions, blisters, or signs of infection. The next step is to determine the etiology. Nipple pain is most commonly related to the infant's inability to effectively latch onto the nipple. If the infant latches on too close to the nipple tip, the nipple is compressed between the infant's tongue and hard palate, creating pressure and eventually friction damage. The remedy is a deeper latch that pulls the nipple farther into the infant's mouth so the nipple touches the soft palate and not the hard palate during suckling.

If there is no obvious nipple injury such as cracks or blisters and repositioning the infant does not relieve the pain, a disorganized or dysfunctional suck is the next likely cause. Immaturity, oral anatomy, and muscular tone can be factors that can be evaluated by a lactation consultant or an oral-motor specialist. Ankyloglossia can quickly inhibit effective milk intake and simultaneously damage a woman's nipples, although most infants with ankyloglossia do not have difficulties nursing. A lactation consultant and/or pediatric provider can evaluate the infant for ankyloglossia and consideration of frenotomy.

Several remedies have been proposed for treating nipple trauma that develops in the first days of breastfeeding, including anhydrous lanolin, nipple shields, and applications of breast milk. Application of breast milk to the injury and use of a breast shield to protect the area between feedings appears to be more effective than anhydrous lanolin (which must be wiped off before breastfeeding), breast shields, or glycerin gel dressings.[95,96]

Conditions That Appear After Breastfeeding Is Established

The list of differential diagnoses for women with late-onset or new-onset nipple pain after breastfeeding has been established is broad. Potential causes include (1) infection—fungal, bacterial, or, rarely, viral (herpes); (2) noninfectious nipple trauma, including eczema, atopic

dermatitis, psoriasis, vasospasm, and rarely Paget's disease of the nipple; and (3) Raynaud's syndrome.

Noninfectious Conditions of the Nipple/Areolar Complex. Noninfectious conditions such as dermatitis (atopic, irritant contact, or allergic contact) or psoriasis usually affect the areola. Acute eruptions may produce vesicles, crusting, and erosions, whereas chronic eruptions are characterized by a dry, erythematous, and scaling pattern. Women with a history of eczema may develop atopic dermatitis that starts at the nipple and spreads across the areola to the breast. Irritant contact dermatitis is the result of direct contact. Possible irritants include soaps, topical antibiotics, and new unwashed clothing. In such a case, the affected area is limited to sites on the breast that were in contact with the irritant. Allergic dermatitis is a hypersensitivity reaction to an irritant. Possible allergic irritants include lanolin, tannic acid from tea bags, vitamins A and E, aloe vera, and fragrances. Consultation with a dermatologist may be indicated.

Infectious Conditions of the Nipple/Areolar Complex: Bacterial Versus Candidal. Infections often follow or present concomitantly with injury to the skin; they may also occur secondary to bacterial or fungal colonization. Diagnosis of candidal versus bacterial infection can be challenging, as the same signs and symptoms are observed in both types of infections. Furthermore, there is controversy about obtaining results from breast milk cultures because breast milk has antibacterial properties and contains lactoferrin, which suppresses growth of *Candida albicans*. This problem has greatly hampered attempts to accurately diagnose the organism responsible for these conditions.[97]

Infection of the nipple/areolar complex is usually diagnosed on the basis of history and physical examination. Women with candidal infection may report a history of their infant having thrush or severe diaper rash, or the infant may have signs of thrush. The nipple and areola are often shiny and red, and there may be flaking of the skin around the nipple or cracks in the areola. The pain is frequently out of proportion compared to the physical findings and typically includes burning, itching, stinging, "piece of glass" feelings, or deep stabbing pain that radiates back toward the chest wall. The stabbing pain has been hypothesized to occur secondary to infection in the ductal system. Multiple symptoms have a higher predictive value than single symptoms for candidal infection, as shown in **Table 35-14**.[98,99]

Table 35-14	**Positive Predictive Value of Signs and Symptoms of *Candida* of the Breast in Lactating Women Between 2 and 9 Weeks Postpartum**
Sign/Symptom	**Positive Predictive Value**
Sore + burning + pain + stabbing + skin changes[a]	100%
Burning + pain + stabbing + skin changes	100%
Pain + stabbing + skin changes	100%

Sore + burning + pain + skin changes	80–91%
Burning + pain + skin changes	80–85%
Sore + burning + pain + stabbing	74%
Burning + pain + stabbing	63%
Pain + stabbing	57%

Most women with severe nipple/areolar pain and/or deep stabbing breast pain respond well to antibiotics or fluconazole (Diflucan), or both. If candidal infection is suspected, the infant should be simultaneously treated with a topical antifungal.[99,100] Empiric treatments often used for nipple/areolar infections are presented in **Table 35-15**.

Table 35-15 Treatment Options for Nipple and Breast Infections

Condition	Treatment
Nipple/areolar infection[a]	*If bacterial infection is suspected:* Mupirocin 2% ointment (not cream): 15 g. If no improvement, culture and treat based on sensitivity results or treat empirically for yeast or consider other etiologies. Polymyxin B sulfate (Bacitracin): Apply mupirocin or polymyxin after each nursing or pumping. *If candidal infection is suspected[b]:* Infant: Nystatin (Nilstat) 100,000 units/mL. Place 1 mL in cup, then swab the inside of the infant's mouth 4 times/day. 2 mL 4 times/day for older infants. Have the infant drink what is left. Gentian violet 1% in 10% alcohol, applied to the infant's mouth with a Q-tip before feeding, so that the mother's nipple will be coated during the feeding. It should be used daily for 4 days, although treatment duration may be extended to 7 days if there is improvement but no resolution. Mother: Topical treatment with nystatin (Nilstat) suspension. Miconazole (Monistat-derm) or clotrimazole (Lotrimin) may be preferred, as resistance to nystatin is increasing. Apply cream or suspension after each nursing. Allow suspension to air dry or, if using cream, rub in a small amount. Gentian violet 1% in 10% alcohol, applied to the nipple. It should be used daily for 4 days, although treatment duration may be extended to 7 days if there is improvement but no resolution. Anti-inflammatory cream and/or antibiotic cream to treat overgrowth with *Staphylococcus aureus*. The topical treatment (Dr. Newman's APNO) is applied sparingly after each feeding and used until nipple soreness has dissipated. APNO is compounded and contains the following ingredients: mupirocin 2% ointment (not cream): 15 g; betamethasone 0.1% ointment (not cream): 15 g; and miconazole powder to formulate a final concentration of 2% miconazole. If miconazole is unavailable, substitute clotrimazole powder added to a final concentration of 2%.
Ductal infection (deep shooting, burning pain)	If topical treatment does not resolve symptoms, systemic treatment for the mother may be considered. Some authors recommend use of fluconazole only after confirmation of candidal infection via breast milk culture or potassium hydroxide examination of skin scraping. Fluconazole (Diflucan): 400 mg loading dose, followed by 200 mg/day for 14 days. Considered safe for the breastfeeding infant.
Infectious mastitis	First-line choices: Dicloxacillin (Dynapen) 500 mg 4 times/day for 10–14 days *or*

	Cephalexin (Keflex) 500 mg 4 times/day for 10–14 days *or* If penicillin allergic, use clindamycin 300 mg 4 times/day or erythromycin 250 mg or 500 mg 4 times/day for 10–14 days Cultures of milk and nipple are indicated for maternal acute illness, failure to respond to treatment, high suspicion of MRSA, and bilateral mastitis. The infant may need to be treated for these conditions concurrently, particularly if infection with group A or B *Streptococcus* is suspected.
Recurrent mastitis	Culture and treat as appropriate for 14–30 days. Low-dose erythromycin or clindamycin may be given on a daily or weekly basis. Consider cultures and treatment with nasal mupirocin (Bactroban) if an *S. aureus* carrier state is suspected.
Ductal infections	Culture and treat as appropriate for at least 14 days. It may be prudent to treat the woman until her symptoms have been resolved for at least 1 week. Can empirically start treatment with clindamycin 300 mg 4 times/day or sulfamethoxazole–trimethoprim (Bactrim) double strength bid. Treat with antifungals if any signs of yeast infection appear in the infant. First-line: Fluconazole (Diflucan) 200–400 mg STAT, followed by 100–200 mg daily for 2–3 weeks or until symptoms have been resolved for 1 week. Ketoconazole (Nizoral) may also be used.
Breast abscess (puerperal)	Outpatient: Dicloxacillin (Dynapen): 500 mg orally 4 times/day for 10–14 days *or* Cephalexin: 500 mg 4 times/day for 10–14 days *or* If penicillin allergic: Clindamycin (Cleocin) 300–400 mg orally 4 times/day for 10–14 days *or* If risk for MRSA[c] and infant is beyond the neonatal period: Trimethoprim–sulfamethoxazole 1DS tablet orally 2 times/day or clindamycin 300 mg 3 times/day Inpatient: Nafcillin or oxacillin 2.0 g every 4 hours IV or cefazolin 1.0 g every 6 hours IV or vancomycin 1.0 g every 12 hours IV Incision and drainage, or use ultrasound-guided needle for aspiration. Send aspirate or discharge for culture. Infants may continue nursing on the affected side unless the area of incision involves the areola. May want to treat infant concurrently if the mother has *S. aureus* or streptococcal disease.

Abbreviations: APNO, All-Purpose Nipple Ointment; DS, double strength; IV, intravenous; MRSA, methicillin-resistant *Staphylococcus aureus*.

[a] All treatments for nipple/areolar pain and/or ductal infection are empiric. There is little evidence to indicate which therapy is superior.

[b] If Candida is suspected or diagnosed, both mother and infant must be treated concurrently, even if one is asymptomatic.

[c] Risk factors for MRSA include recent hospitalization, recent surgery, hemodialysis, HIV infection, injection drug use, prior antibiotic use (failed therapy), military service, sharing sports equipment, and sharing needles or razors.

Raynaud's Phenomenon. Nipple vasospasm may be the cause of pain following breastfeeding or when the nipples are exposed to cool air, such as after a shower, in cold temperatures, or with cold air blowing from automobile air-conditioning ducts. Blanching or purple color changes with sharp, burning pain may be experienced. Connective tissue diseases or Raynaud's phenomenon may be related and should be explored.[101] Some clinicians use nifedipine (a beta blocker) for nipple spasms unrelieved by avoiding cold exposure or warmth.[102]

Mastitis

Mastitis refers to a breast inflammation such that one or more segments of the breast are hot, red, and inflamed. Risk factors for infectious mastitis include unresolved milk stasis, nipple damage, and previous episodes of mastitis. Mastitis is common, affecting as many as 1 in 5 women. Approximately 11% of women with mastitis will develop a breast abscess if the mastitis is not treated, and approximately 3% will develop an abscess even when the mastitis is treated.[103] The evaluation of a woman with symptoms of mastitis includes a breast examination to rule out breast abscess.

The diagnosis of mastitis is made clinically on the basis of a localized, unilateral area of erythema with associated fever (38.5°C [101°F] or higher) and feelings of malaise, flu-like symptoms, and fatigue. The infective agents most commonly implicated in this type of mastitis include *Staphylococcus aureus* and coagulase-negative *Staphylococcus* (CoNS), although *Candida albicans*, *Escherichia coli*, *Enterobacteriaceae*, and *Mycobacterium tuberculosis* have also been cultured from infected breasts. Bilateral mastitis, although rare, is likely to be associated with *Streptococcus* infection. Methicillin-resistant *S. aureus* (MRSA) mastitis has been noted in communities with a high prevalence of these bacteria.[104] Most symptoms resolve within 48 hours with sufficient and frequent breast emptying, adequate fluids and nutrition, and antibiotic therapy.

If symptoms do not resolve within 48 hours of initiating antibiotics, or if symptoms worsen, the milk should be cultured and consultation obtained to determine the best choice of alternative antibiotic. Rarely, persistent unresolved mastitis may be an early sign of inflammatory carcinoma.

Breast Abscess

An abscess is a localized collection of pus in the breast, formed by disintegrated cells and surrounded by an inflamed area (**Figure 35-4**). The infecting organism is most often *S. aureus*, although MRSA is increasingly being identified in such cases. The differential diagnosis includes mastitis (without abscess) and galactocele. Diagnosis is based on physical examination and confirmed via ultrasound.



Figure 35-4 Breast abscess.

Reproduced with permission from International Lactation Consultant Association; Mannel R, Martens PJ, Walker M, eds. *Core Curriculum for Lactation Consultant Practice.* 3rd ed. Burlington, MA: Jones & Bartlett Learning; 2013.[68]

Breast abscesses are treated with surgical drainage or needle aspiration to allow drainage to the exterior. Antibiotics may also be recommended. A woman who develops a breast abscess is referred for physician care but may be collaboratively managed by a midwife to help support ongoing breastfeeding.

Special Populations

There are many situations in which breastfeeding is desired but may be challenging. This section reviews care of individuals who want to breastfeed but may need additional support especially to meet the infant's nutritional needs.

Breast Reduction Surgery and Breast Augmentation

Lactation is not generally impaired in women who had a breast reduction procedure that used a pedicle technique. Conversely, breastfeeding is not likely to be possible for women who had a breast reduction procedure that involved removal of the nipple.[105] Although lactation insufficiency is common among women who had a breast augmentation, outcomes are unpredictable. Some women are able to exclusively breastfeed without difficulty.[106] All women who have undergone breast surgery should be monitored for insufficient milk production and may be best supported by a lactation consultant who has experience in this area.

Maternal–Newborn Separation Secondary to Illness

Women and newborns may be separated after birth secondary to maternal or newborn ill health. Premature newborns can have short suck cycles, weak or uncoordinated suck–swallow–breathe rhythms, small oral cavities, and respiratory conditions that inhibit breastfeeding. If a newborn cannot effectively breastfeed, the goal for the mother is to express milk approximately as often as a newborn would normally feed. Most newborns nurse approximately 8 times in a 24-hour period. Milk can be expressed via hand, manual pump, or electric pump.[107] Some hospitals have milk banks, and donor milk can be used to feed the newborn. Donors are screened for HIV-1, HIV-2, human T-cell leukemia virus 1 and 2, hepatitis B, hepatitis C, and syphilis. Low-heat processing of donor milk reduces cellular components, growth factors, and nutrients in the breast milk; therefore, banked milk for preterm infants is fortified with essential nutrients.

Many different technologies and techniques may be used to assist an individual who cannot breastfeed her infant.[108] A midwife who has additional training in lactation can be part of the healthcare team helping a family in this situation. The Resources section at the end of this chapter identifies sources that may help midwives who need to offer this specialized care if a lactation consultant or pediatric specialist is not available.

Expressing Milk for Bottle-Feeding

There are many reasons why women might need to express breast milk so their infant can receive that milk via bottle. General guidance for this situation includes knowledge about ways to effectively express milk and handle expressed milk. Pumped milk maintains its quality at room temperature for 4–6 hours in environments at 66–78°F.[109] Coolers with ice or gel packs with a temperature lower than 59°F will keep milk safely for 24 hours. Breast milk can also be

stored in a refrigerator for 3–8 days (32–39°F) and in a 4°F freezer/refrigerator combination unit for 3–4 months.[109] Breast milk can be thawed overnight in a refrigerator. Once thawed, it should not be refrozen.[109]

Adoption and Relactation

Although many case reports of relactation (resuming lactation after it had stopped) and induced lactation for an adopted infant have been published, effective techniques and success rates are not well known for these techniques. Additional support by a lactation consultant is recommended for these situations.

Nutrition for Lactating Women

Lactating women use approximately 500–700 calories more per day than do nonpregnant women, with this energy being devoted to production of approximately 750–800 mL of breast milk per day once lactation is established. The recommended daily reference intakes for lactating women for macronutrients, micronutrients, and vitamins are listed in the *Nutrition* chapter. Routine supplementation is not recommended for all women, as the increased needs specific to lactation can be easily met by consuming a healthy diet. That said, women who follow a vegetarian or vegan diet, women who have undergone bariatric surgery, and women who have malabsorption disorders may need supplementation of specific micronutrients. The micronutrients particularly needed during lactation are vitamin D, long-chain polyunsaturated fatty acids, and vitamin B_{12}.

Vitamin D levels in breast milk are constitutionally low in all women. Furthermore, typical vitamin D levels in breast milk are lower than the recommended daily intake values for exclusively breastfed infants. Hence it is recommended that all breastfed infants be given an oral supplement of vitamin D 400 IU per day.

Docosahexaenoic acid (DHA), a long-chain polyunsaturated fatty acid, is abundant in the developing brain of a fetus, newborn, and infant. These fatty acids are "essential" because they are not made by the body and must be obtained from the maternal diet. Women whose diets have higher concentrations of DHA also have higher concentrations of this fatty acid in their breast milk. Although some studies have found that DHA supplementation during breastfeeding improves infant cognition, visual acuity, and development,[110] a Cochrane meta-analysis of this body of research did not find any significant benefit in women who took DHA supplements during lactation.[111] The American Academy of Pediatrics has recommended that breastfeeding women whose diet is deficient in DHA take a supplement.[1] Sufficient DHA can be obtained by consuming two servings of fish per week. Thus, women who follow a strict vegan diet are those who are most likely to benefit from a supplement that contains DHA.

Vitamin B_{12} plays an essential role in DNA synthesis and is obtained via consumption of meat or dairy products. Vitamin B_{12} deficiency in infants is associated with developmental abnormalities and anemia. Women who have undergone bariatric surgery and those who follow a vegan or lacto-ovo vegetarian diets are advised to take supplemental vitamin B_{12} in the range of 2.8 mcg per day.[112]

Infants at high risk for allergies and atopic eczema have a decreased incidence of these conditions if they are breastfed exclusively for 4 to 6 months. There is no evidence that maternal avoidance of certain foods during pregnancy decreases the risk of allergies in infants, although some evidence does suggest that antigen avoidance in high-risk mothers during lactation may reduce atopic disease.[113]

Weight Loss During Breastfeeding

Postpartum weight retention can be an important health problem for women. The evidence on the role of breastfeeding in postpartum weight loss is mixed, probably reflecting studies' poor control for the intensity and duration of lactation. However, among high-quality studies, the

data support an association, with breastfeeding accounting for a 1-kg weight loss at one year postpartum.[114] A summary review of breastfeeding among women in developed countries found that women who were breastfeeding exclusively reached their prepregnant weight approximately 6 months earlier than women who were bottle-feeding; a slowing of weight loss was observed when lactation was stopped.[6]

Women should be counseled about ways to lose their pregnancy weight after the birth. Even lactating women can safely exercise (45 minutes per day, 4 days per week) and decrease their daily intake by 500 calories to achieve a safe weight loss of 1 pound per week with no ill effects on their infant's health.[115]

Formula-Feeding

Breast milk substitutes are available for women who choose not to feed their newborn with human milk or if breastfeeding is not possible. Breast milk substitutes based on cow's milk are commonly suggested for formula-fed infants, except those with a known allergy to cow's milk. These products are formulated either as powders that are mixed with water or as premixed liquids. Incorrectly reconstituted formula risks under-nutrition or gastrointestinal irritation of the infant; therefore, liquid, ready-to-feed products may be the best option for some families. Cow's milk–based formulas are designed to approximate human milk, with similar whey/casein protein ratios. These formulations also approximate the calorie content of human milk: 19–20 calories per fluid ounce. The usual intake for a healthy term newborn is 100–110 kCal per kilogram per day. Additives found in such formulas typically include iron, vitamin D, lutein, DHA, and arachidonic acid (ARA).

Formulas that are developed from cow's milk–based hydrolysates are the first option for infants who exhibit signs of cow's milk allergies. Hydrolysates are produced by heating cow casein and whey with enzymes that split proteins into peptide chains and free amino acids. Newly developed rice hydrolysates also show promise as a first treatment in cow's milk allergy. Soy-based formulas are second-choice options, as 10% to 15% of infants who are allergic to cow's milk also react to soy products.[116]

Infants should always be fed on cue and not be pressed to consume specific amounts of formula. They should never be fed "propped" bottles or given a bottle in the crib to hold, because doing so increases the risk of choking and aspiration.

Conclusion

Most women in the United States now initiate breastfeeding, and approximately half continue breastfeeding for at least the first 6 months. Most breastfeeding-related problems have relatively easy-to-implement solutions that are within the scope of midwifery practice. Referrals to specialized lactation assistance when needed can preserve the breastfeeding relationship. A woman or primary caregiver who chooses not to breastfeed should be supported without judgment. These women and those who cannot breastfeed can be assured that their infants will receive essential nutrition via breast milk substitute formulas.

Resources

Article		
Turner-Maffei C. Lactation resources for clinicians. *J Midwifery Womens Health*. 2007;52:e57-e65. https://doi.org/10.1016/j.jmwh.2007.03.024.		
Organization	**Description**	**Webpage**
Academy of Breastfeeding Medicine (ABM)	Protocols in English, Spanish, and other languages for management of many breastfeeding problems.	http://www.bfmed.org/Resources/Protocols.aspx
Baby-Friendly Hospital Initiative (BFHI)	Baby-Friendly USA is the accrediting body for the BFHI in the United States.	https://www.babyfriendlyusa.org
La Leche League International (LLLI)	Community-based support for women.	http://www.llli.org
World Alliance for Breastfeeding Action (WABA)	A worldwide coalition of organizations to improve breastfeeding protection and support.	http://waba.org.my
Centers for Disease Control and Prevention (CDC)	U.S. breastfeeding data, policies, and information for women and healthcare professionals.	https://www.cdc.gov/breastfeeding/resources/us-breastfeeding-rates.html
	Strategies to Prevent Obesity and Other Chronic Diseases: The CDC Guide to Strategies to Support Breastfeeding Mothers and Infants. Atlanta, GA: Department of Health and Human Services; 2013 This guide presents evidence-based maternity care practices, professional support, peer-support programs, and breastfeeding education programs.	https://www.cdc.gov/obesity/downloads/fandv_2011_web_tag508.pdf
Human Milk Banking Association of North America (HMBANA)	Guidelines for establishing a milk bank and locations of milk banks in the United States and Canada	https://www.hmbana.org
International Lactation Consultant Association (ILCA)	Directory of lactation consultants (IBCLC). Continuing education in breastfeeding and lactation.	http://www.ilca.org/home
National Library of Medicine	Drugs and Lactation Database: database for	https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm

(NLM)	checking safety of medication use during breastfeeding.	
Office on Women's Health (OWH)	Resource for breastfeeding women who are returning to work.	https://www.womenshealth.gov/breastfeeding
World Health Organization (WHO) and United Nations Children's Fund (UNICEF)	*Breastfeeding and Maternal Medication: Recommendations for Drugs in the Eleventh WHO Model List of Essential Drugs*: rates drugs on a 5-point scale that moves from compatible with breastfeeding, through compatible with side effects, to avoid. Extremely comprehensive and easy-to-use reference.	http://apps.who.int/iris/bitstream/10665/62435/1/55732.pdf
United States Breastfeeding Committee (USBC)	Coalition of professional, educational, and governmental organizations. Publishes core competencies on breastfeeding for healthcare professionals. These competencies are endorsed by the American College of Nurse-Midwives.	http://www.usbreastfeeding.org http://www.usbreastfeeding.org/p/cm/ld/fid=170

Organization	Description	Webpage
Resources for Medication Use During Breastfeeding		
American Academy of Pediatrics (AAP)	The AAP Committee on Drugs publishes a list of drugs with rating for safety during breastfeeding.	http://www.aap.org/breastfeeding
LactMed from National Institutes of Health (NIH)	Database that summarizes information on drugs and other chemicals to which breastfeeding women can be exposed. Reviews studies of pharmacokinetics, and recommends alternatives where appropriate. Data are well referenced. App is available.	http://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm
Motherisk	Service of the Hospital for Sick Children in Toronto. This site has a wealth of reviews on current drug topics of interest for breastfeeding. Motherisk counselors are available for phone contact during the week at 1-877-439-2744.	http://www.motherisk.org/prof/breastfeeding.jsp
Organization of Teratology Information	MotherToBaby service of OTIS provides fact sheets and counseling about	https://mothertobaby.org/fact-sheets-parent/

Specialists (OTIS)	medications and other exposures.

References

1. American Academy of Pediatrics. Breastfeeding and the use of human milk. *Pediatrics*. 2012;129(3):e827-e841.
2. World Health Organization. Global strategy for infant and young child feeding. 2003. Available at: http://www.who.int/maternal_child_adolescent/documents/9241562218/en/. Accessed August 30, 2017.
3. U.S. Department of Health and Human Services. *The Surgeon General's Call to Action to Support Breastfeeding*. Washington, DC: U.S. Department of Health and Human Services Office of the Surgeon General; 2011. Available at: https://www.surgeongeneral.gov/library/calls/breastfeeding/index.html. Accessed August 28, 2017.
4. American College of Nurse-Midwives. Position statement on breastfeeding. 2016. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000248/Breastfeeding-statement-Feb-2016.pdf. Accessed August 29, 2017.
5. Chowdhury R, Sinha B, Sankar MJ, et al. Breastfeeding and maternal health outcomes: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:96-113.
6. Ip S, Chung M, Raman G, et al. Breastfeeding and maternal and infant health outcomes in developed countries. *Evid Rep Technol Assess*. 2007;153:1-186.
7. Victoria CG, Bahl R, Barros A, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet*. 2016;387:475-490.
8. World Health Organization. Indicators for assessing infant and young child feeding practices: Part 1 definitions. 2008. Available at: http://whqlibdoc.who.int/publications/2008/9789241596664_eng.pdf. Accessed August 31, 2017.
9. Centers for Disease Control and Prevention. Breastfeeding report card 2016. Available at: https://www.cdc.gov/breastfeeding/pdf/2016breastfeedingreportcard.pdf. Accessed August 28, 2017.
10. *Healthy People 2020 Breastfeeding Goals*. Washington, DC: U.S. Department of Health and Human Services, Office o Disease Prevention and Health Promotion. 2014. Available at: https://www.healthypeople.gov/2020/topics-objectives/topic/maternal-infant-and-child-health/objectives. Accessed August 29, 2017.
11. Anstey EH, Chen J, Elam-Evans LD, Perrine CG. Racial and geographic differences in breastfeeding—United States, 2011–2015. *MMWR*. 2017;66(27):723-727.
12. Kent JC. How breastfeeding works. *J Midwifery Womens Health*. 2007;52(6):564-570.
13. Ballard O, Morrow AL. Human milk composition: nutrients and bioactive factors. *Ped Clin North Am*. 2013;60(1):49-74.
14. Andreas NJ, Kampmann B, Le-Doare KM. Human breast milk; a review on its composition and bioactivity. *Early Hum Develop*. 2015;91:629-635.
15. Gomez-Gallego C, Garcia-Mantrana I, Salminen S, Collabo MC. The human milk microbiome and factors influencing its composition and activity. *Semin Fetal Matern Med*. 2016;21:400-405.
16. Allen-Blevins CR, Sela DA, Hinde K. Milk bioactives may manipulate microbes to mediate parent–offspring conflict. *Evo Med Public Health*. 2015(1):106-121.
17. Gomez-Gallego C, Garcia-Mantrana I, Salminen S, Collado MC. The human milk microbiome and factors influencing its composition and activity. *Semin Fetal Neonatal Med*. 2016;21(6):400-405.
18. Sankar MJ, Sinha B, Chowdhury R, et al. Optimal breastfeeding practices and infant and child mortality: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:3-13.
19. Lodge CJ, Tan DJ, Lau MXZ, et al. Breastfeeding and asthma and allergies: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:38-53.
20. Tham R, Bowatte G, Dharmage SC, et al. Breastfeeding and the risk of dental caries: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:62-84.
21. Peres KG, Cascaes AM, Nascimento GG, Victora CG. Effect of breastfeeding on malocclusions: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:54-61.
22. Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Does breastfeeding influence risk of type 2 diabetes in later life? A quantitative analysis of published evidence. *Am J Clin Nutr*. 2006;84(5):1043.
23. Carlin RF, Moon RY. Risk factors, protective factors, and current recommendations to reduce sudden infant death syndrome: a review. *JAMA Pediatr*. 2017;171(2):175-180.
24. Moon RY. Task Force on Sudden Infant Death Syndrome. SIDS and other sleep-related infant deaths: evidence base for 2016 updated recommendations for a safe infant sleeping environment. *Pediatrics*. 2016:138(5). pii: e20162940.
25. Koh K. Maternal breastfeeding and children's cognitive development. *Soc Sci Med*. 2017;187:101-108.
26. Huang J, Peters KE, Vaughn MG, Witko C. Breastfeeding and trajectories of children's cognitive development. *Develop Sci*. 2014;17(3):452-461.
27. Der G, Batty GD, Deary IJ. Effect of breast feeding on intelligence in children: prospective study, sibling pairs analysis, an meta-analysis. *BMJ*. 2006;333(7575):945.
28. Kramer MS, Aboud F, Mironova E, et al.; Promotion of Breastfeeding Intervention Trial (PROBIT) Study Group. Breastfeeding and child cognitive development: new evidence from a large randomized trial. *Arch Gen Psychiatry*. 2008;65(5):578-584.

29. Perrine CG, Nelson JM, Corbelli J, Scanlon KS. Lactation and maternal cardio-metabolic health. *Annu Rev Nutri*. 2016;36:627-645.
30. Karlson EW, Mandl LA, Hankinson SE, Grodstein F. Do breast-feeding and other reproductive factors influence future ris of rheumatoid arthritis? Results from the Nurses' Health Study. *Arthritis Rheum*. 2004;50(11):3458-3467.
31. Schwarz EB, Brown JS, Creasman JM, et al. Lactation and maternal risk of type 2 diabetes: a population-based study. *Am J Med*. 2010;123(9):863.e1-863.e6.
32. Pope CJ, Mazmanian D. Breastfeeding and postpartum depression: an overview and methodological recommendations for future research. *Depression Res Treat*. 2016;2016:4765310.
33. Bell AF, Erickson EN, Carter CS. Beyond labor: the role of natural and synthetic oxytocin in the transition to motherhood. *Midwifery Womens Health*. 2014;59:35-42.
34. Cox EQ, Stuebe A, Peirson B, Grewen K, Rubinow D, Meltzer-Brody S. Oxytocin and HPA stress axis reactivity in postpartum women. *Psychoneuroendocrinol*. 2015;55:164-172.
35. Strathearn L, Mamun AA, Najman JM, O'Callaghan MJ. Does breastfeeding protect against substantiated child abuse an neglect? A 15-year cohort study. *Pediatrics*. 2009;123(2):483-493.
36. Patnode CD, Henninger ML, Senger CA, Perdue LA, Whitlock EP. Primary care interventions to support breastfeeding updated evidence report and systematic review for the U.S. Preventive Services Task Force. *JAMA*. 2016;316(16):1694-1705.
37. Centers for Disease Control and Prevention. *Strategies to Prevent Obesity and Other Chronic Diseases: The CDC Guide to Strategies to Support Breastfeeding Mothers and Infants*. Atlanta, GA: Department of Health and Human Services; 2013. Available at: https://www.cdc.gov/obesity/downloads/fandv_2011_web_tag508.pdf. Accessed August 29, 2017.
38. World Health Organization, United Nations Childrens Fund. *National Implementation of the Baby-Friendly Hospital Initiative 2017*. Geneva, Switzerland: World Health Organization; 2017. Available at: http://apps.who.int/iris/bitstream/10665/255197/1/9789241512381-eng.pdf?ua=1. Accessed August 29, 2017.
39. Smith LJ. Impact of birthing practices on the breastfeeding dyad. *J Midwifery Womens Health*. 2007;52(6):621-630.
40. de Barros Duarte L, Dantas Móises EC, Cavalli RC, Lanchote VL, Duarte G, da Cunha SP. Distribution of bupivacaine enantiomers and lidocaine and its metabolite in the placental intervillous space and in the different maternal and fetal compartments in term pregnant women. *J Clin Pharmacol*. 2011;51(2):212-217.
41. de Barros Duarte L, Moisés EC, Carvalho Cavalli R, Lanchote VL, Duarte G, da Cunha SP. Distribution of fentanyl in the placental intervillous space and in the different maternal and fetal compartments in term pregnant women. *Eur J Clin Pharmacol*. 2009;65(8):803-808.
42. Wang K, Cao L, Deng Q, et al. The effects of epidural/spinal opioids in labour analgesia on neonatal outcomes: a meta-analysis of randomized controlled trials. *Can J Anaesth* 2014; 61:695.
43. Beilin Y, Bodian CA, Weiser J, et al. Effect of labor epidural analgesia with and without fentanyl on infant breast-feeding: prospective, randomized, double-blind study. *Anesthesiology*. 2005;103(6):1211-1217.
44. Moore ER, Bergman N, Anderson GC, Medley N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;11:CD003519. doi:10.1002/14651858.CD003519.pub4.
45. Hung KJ, Berg O. Early skin-to-skin after cesarean to improve breastfeeding. *Matern Child Nurs*. 2011;36(5):318-324.
46. Schafer R, Watson Genna C. Physiologic breastfeeding: a contemporary approach to breastfeeding initiation. *J Midwifery Womens Health*. 2015;60:546-553.
47. American College of Obstetricians and Gynecologists' Committee on Obstetric Practice; Breastfeeding Expert Work Group. Committee Opinion No. 658: optimizing support for breastfeeding as part of obstetric practice. *Obstet Gynecol*. 2016;127(2):e86-e92.
48. Uscher-Pines L, Mehrotra A, Bogen DL. The emergence and promise of telelactation. *Am J Obstet Gynecol*. 2017;217(2):176-178.
49. Howe-Heyman A, Lutenbacher M. The Baby-Friendly Hospital Initiative as an intervention to improve breastfeeding rate A review of the literature. *J Midwifery Womens Health*. 2016;61:77-102.
50. Centers for Disease Control and Prevention. When should a mother avoid breastfeeding? Available at: https://www.cdc.gov/breastfeeding/disease/index.htm. Accessed March 14, 2017.
51. U.S. Food and Drug Administration. FDA Drug Safety Communication: FDA restricts use of prescription codeine pain an cough medicines and tramadol pain medicines in children; recommends against use in breastfeeding women. Silver Spring (MD): FDA; 2017. Available at: https://www.fda.gov/Drugs/DrugSafety/ucm549679.htm. Retrieved April 24, 2017
52. American College of Obstetricians and Gynecologists. Practice advisory on codeine and tramadol in breastfeeding women April 27, 2017. Available at: https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-on-Codeine-and-Tramadol-for-Breastfeeding-Women. Accessed December 28, 2017.
53. American Academy of Pediatrics, Committee on Drugs. Transfer of drugs and other chemicals into human milk. *Pediatrics*. 2001;108(3):776-789.
54. Haastrup MB, Pottegard A, Damkier P. Alcohol and breastfeeding. *Basic Clin Pharmacol Toxicol*. 2014;114(2):168-173.

55. Foeller ME, Lyell DJ. Marijuana use in pregnancy: concerns in an evolving era. *J Midwifery Womens Health*. 2017;62(3):363-367.
56. Newton ER, Hale TW. Drugs in breast milk. *Clin Obstet Gynecol*. 2015;58(4):868-884.
57. Sachs HC; Committee on Drugs. The transfer of drugs and therapeutics into human breast milk: an update on selected topics. *Pediatrics*. 2013;132(3):e796-e809.
58. Moretti ME, Lee A, Ito S. Which drugs are contraindicated during breastfeeding: Mortherisk Update. *Can Fam Physician* 2000;46:1753-1757. Available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2145042/pdf/canfamphys00031-0055.pdf. Accessed September 2, 2017.
59. Buschimi CS, Weiner CP. Medications in pregnancy and lactation. *Obstet Gynecol*. 2009;113:166-188.
60. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep*. 2016;65(RR-3):1-104.
61. Oladapo O, Fawole B. Treatments for suppression of lactation. *Cochrane Database Syst Rev.* 2012 (9):CD005937. doi:10.1002/14651858.CD005937.pub3.
62. The Academy of Breastfeeding Medicine Protocol C. ABM Clinical Protocol #9: use of galactogogues in initiating or augmenting the rate of maternal milk secretion (first revision January 2011). *Breastfeeding Med*. 2011;6(1):41-49.
63. Osadchy A, Moretti ME, Koren G. Effect of domperidone on insufficient lactation in puerperal women: a systematic review and meta-analysis of randomized controlled trials. *Obstet Gynecol Int.* 2012;2012:642893.
64. Bazzano AN, Hofer R, Thibeau S, Gillispie V, Jacobs M, Theall KP. A review of herbal and pharmaceutical galactagogues for breast-feeding. *Ochsner J*. 2016;16(4):511-524.
65. Mortel M, Mehta SD. Systematic review of the efficacy of herbal galactogogues. *J Hum Lact*. 2013;29(2):154-162.
66. Kelly LE, Chaudhry SA, Rieder MJ, et al. A clinical tool for reducing central nervous system depression among neonates exposed to codeine through breast milk. *PLoS One*. 2013;8(7):e70073. doi:10.1371/journal.pone.0070073.
67. Payne JL. Psychopharmacology in pregnancy and breastfeeding. *Psychiatr Clin North Am.* 2017;40(2):217-238.
68. International Lactation Consultant Association; Mannel R, Martens PJ, Walker M, eds. *Core Curriculum for Lactation Consultant Practice*. 3rd ed. Burlington, MA: Jones & Bartlett; 2013.
69. Hill PD, Johnson TS. Assessment of breastfeeding and infant growth. *J Midwifery Womens Health*. 2007;52(6):571-578.
70. Cadwell K. Latching-on and suckling of the healthy term neonate: breastfeeding assessment. *J Midwifery Womens Health*. 2007;52(6):638-642.
71. Hetherington MM. Understanding infant eating behaviour: lessons learned from observation. *Physiol Behav*. 2017;176:117-124.
72. Colson SD, Meek JH, Hawdon JM. Optimal positions for the release of primitive neonatal reflexes stimulating breastfeeding. *Early Hum Develop*. 2008;84(7):441-449.
73. Wambach KA, Riordan J. *Breastfeeding and Human Lactation*. 5th ed. Burlington, MA: Jones & Bartlett Learning; 2016.
74. Jacobs LA, Dickinson JE, Hart PD, Doherty DA, Faulkner SJ. Normal nipple position in term infants measured on breastfeeding ultrasound. *J Hum Lact*. 2007;23(1):52-59.
75. Sakalidis VS, Geddes DT. Suck–swallow–breathe dynamics in breastfed infants. *J Hum Lact*. 2016;32(2):201-211.
76. Bergman NJ. Neonatal stomach volume and physiology suggest feeding at 1-h intervals. *Acta Paediatr*. 2013;102(8):773-777.
77. Kent JC, Mitoulas LR, Cregan MD, Ramsay DT, Doherty DA, Hartmann PE. Volume and frequency of breastfeedings and fat content of breast milk throughout the day. *Pediatrics*. 2006;117(3):e387-e395.
78. Nommsen-Rivers LA, Heinig MJ, Cohen RJ, Dewey KG. Newborn wet and soiled diaper counts and timing of onset of lactation as indicators of breastfeeding inadequacy. *J Hum Lact*. 2008;24(1):27-33.
79. Wagner EA, Chantry CJ, Dewey KG, Nommsen-Rivers LA. Breastfeeding concerns at 3 and 7 days postpartum and feeding status at 2 months. *Pediatrics*. 2013. 132(4):e865-e875.
80. Ahluwalia IB, Morrow B, Hsia J. Why do women stop breastfeeding: findings from the pregnancy risk assessment monitoring system. *Pediatrics*. 2005;116(6):1408-1412.
81. Bunik M. The pediatrician's role in encouraging exclusive breastfeeding. *Pediatr Rev*. 2017;38(8):353-368.
82. Betzold CM. An update on the recognition and management of lactational breast inflammation. *J Midwifery Womens Health*. 2007;52:595-605.
83. Mangesi L, Zakarija-Grkovic I. Treatments for breast engorgement during lactation. *Cochrane Database Syst Rev.* 2016;28(6):CD006946. doi:10.1002/14651858.CD006946.pub3.
84. Roberts KL. A comparison of chilled cabbage leaves and chilled gelpaks in reducing breast engorgement. *J Hum Lact*. 1995;11(1):17-20.
85. Brownell E, Howard CR, Lawrence RA, Dozier AM. Does delayed onset lactogenesis II predict the cessation of any or exclusive breastfeeding? *J Pediatr*. 2012;161(4):608-614.
86. Neville MC, Allen JC, Archer PC, et al. Studies in human lactation: milk volume and nutrient composition during weaning and lactogenesis. *Am J Clin Nutr*. 1991;54(1):81-92.

87. Arthur PG, Smith M, Hartmann PE. Milk lactose, citrate, and glucose as markers of lactogenesis in normal and diabetic women. *J Pediatr Gastroenterol Nutr*. 1989;9(4):488-496.
88. Scott JA, Binns CW, Oddy WH. Predictors of delayed onset of lactation. *Matern Child Nutr*. 2007;3(3):186-193.
89. Vestermark V, Hogdall CK, Birch M, Plenov G, Toftager-Larsen K. Influence of the mode of delivery on initiation of breast-feeding. *Eur J Obstet Gynecol Reprod Biol*. 1991;38:33-38.
90. Hurst NM. Recognizing and treating delayed or failed lactogenesis II. *J Midwifery Womens Health*. 2007;52(6):588-594.
91. Joseph R, Giovinazzo M, Brown M. A literature review on the practice of placentophagia. *Nurs Womens Health*. 2016;20(5):476-483.
92. Bever Babendure J, Reifsnider E, Mendias E, Moramarco M, Davila Y. Reduced breastfeeding rates among obese mothers: a review of contributing factors, clinical considerations and future directions. *Int Breastfeed*. 2015;10:21. doi:10.1186/s13006-015-0046-5.
93. Arbour MW, Kessler JL. Mammary hypoplasia: not every breast can produce sufficient milk. *J Midwifery Womens Health*. 2013;58(4):457-461.
94. Kent JC, Prime DK, Garbin CP. Principles for maintaining or increasing breast milk production. *J Obstet Gynecol Neonatal Nurs*. 2012;41(1):114-121.
95. Vieira F, Mota DD, Castral TC, Guimarães JV, Salge AKM, Bachion MM. Effects of anhydrous lanolin versus breast milk combined with a breast shell for the treatment of nipple trauma and pain during breastfeeding: a randomized clinical trial. *J Midwifery Womens Health*. 2017;62(5):572-579.
96. Dennis CL, Jackson K, Watson J. Interventions for treating painful nipples among breastfeeding women. *Cochrane Database Syst Rev*. 2014;12:CD007366. doi:10.1002/14651858.CD007366.pub2.
97. Betzold CM. Results of microbial testing exploring the etiology of deep breast pain during lactation: a systematic review and meta-analysis of nonrandomized trials. *J Midwifery Womens Health*. 2012;57(4):353-364.
98. Francis-Morrill J, Heinig MJ, Pappagianis D, Dewey KG. Diagnostic value of signs and symptoms of mammary candidosis among lactating women. *J Hum Lact*. 2004;20(3):288-295; quiz 296-289.
99. Wiener S. Diagnosis and management of *Candida* of the nipple and breast. *J Midwifery Womens Health*. 2006;51(2):125-128.
100. Amir LH, Donath SM, Garland SM, et al. Does *Candida* and/or *Staphylococcus* play a role in nipple and breast pain in lactation? A cohort study in Melbourne, Australia. *BMJ Open*. 2013;3(3). pii: e002351. doi:10.1136/bmjopen-2012-002351.
101. Barrett ME, Heller MM, Stone HF, Murase JE. Raynaud phenomenon of the nipple in breastfeeding mothers: an underdiagnosed cause of nipple pain. *JAMA Dermatol*. 2013;149(3):300-306.
102. Garrison CP. Nipple vasospasms, raynaud's syndrome, and nifedipine. *J Hum Lact*. 2002;18(4):382-385.
103. Amir LH, Forstere D, McLachlan H, Lumley J. Incidence of breast abscess in lactating women: a report from an Australian cohort. *BJOG*. 2004;111(12):1378-1381.
104. Berens P, Swaim L, Peterson B. Incidence of methicillin-resistant *Staphylococcus aureus* in postpartum breast abscesses. *Breastfeed Med*. 2010;5(3):113-115.
105. Thibaudeau S, Sinno H, Williams B. The effects of breast reduction on successful breastfeeding: a systematic review. *J Plast Reconstr Aesthet Surg*. 2010;63:1688.
106. Michalopoulos K. The effects of breast augmentation surgery on future ability to lactate. *Breast J*. 2007;13:1:62-74.
107. Becker GE, Smith HA, Cooney F. Methods of milk expression for lactating women. *Cochrane Database Syst Rev*. 2016;9:CD006170. doi:10.1002/14651858.CD006170.pub5.
108. Flasherman VJ, Lee HC. "Breastfeeding" by feeding expressed mother's milk. *Pediatr Clin North Am*. 2013;60:227-246.
109. La Leche League International. What are the LLLI guidelines for storing my pumped milk? Available at: http://www.llli.org/faq/milkstorage.html. Accessed August 27, 2017.
110. Carlson SE. Docosahexaenoic acid supplementation in pregnancy and lactation. *Am J Clin Nutr*. 2009;89(2):678S-684S.
111. Delgado-Noguera MF, Calvache JA, Bonfill Cosp X, Kotanidou EP, Galli-Tsinopoulou A. Supplementation with long chain polyunsaturated fatty acids (LCPUFA) to breastfeeding mothers for improving child growth and development. *Cochrane Database Syst Rev*. 2015;7:CD007901. doi:10.1002/14651858.CD007901.pub3.
112. Penney DS, Miller KG. Nutritional counseling for vegetarians during pregnancy and lactation. *J Midwifery Womens Health*. 2008;53(1):37-44.
113. Kramer MS, Kakuma R. Maternal dietary antigen avoidance during pregnancy or lactation, or both, for preventing or treating atopic disease in the child. *Cochrane Database Syst Rev*. 2012;9:CD000133.
114. Neville CE, McKinley MC, Holmes VA, Spence D, Woodside JV. The relationship between breastfeeding and postpartum weight change: a systematic review and critical evaluation. *Int J Obesity*. 2014;38(4):577-590.
115. Lovelady CA, Garner KE, Moreno KL, Williams JP. The effect of weight loss in overweight, lactating women on the growth of their infants. *New Eng J Med*. 2000;342(7):449-453.
116. Vandenplas Y. Prevention and management of cow's milk allergy in non-exclusively breastfed infants. *Nutrients*. 2017;9(7):731.

PART VII

Newborn

LILY HSIA

Welcoming the Newborn

The moment a newborn enters the world, a lusty cry brings an instant, unconditional joy and great happiness to the family. That initial, spontaneous wail reassures the newborn's mother, father, and midwife, as well as others, that this new life has just been launched onto an uncharted journey, with immense uncertainty but also great promise. Only a few seconds or minutes after the birth, the parents already have their hopes for the baby. Everyone present at the birth can now take a deep breath and feel at ease as congratulations are extended all around.

At birth, the baby has limited functional and structural adaptability. The first hour is often called the "golden hour of life," in recognition of the fact that the newborn's intrauterine to extrauterine transition is both dramatic and critical. The midwife needs to provide a safe environment to ease the transition of the neonate, whose functional and physiologic changes in respiratory and circulatory systems, thermoregulation, and glucose metabolism must begin immediately so that that the baby can rapidly assume physiologic independent without delay (discussed in the *Anatomy and Physiology of the Newborn* chapter).

As the newborn and mother bond soon after birth, one of the important functions of the midwife is to perform an initial head-to-toe examination of the newborn in front of the family. The midwife needs to be familiar with the woman's prenatal, labor, and birth history and to note any problems such as intrauterine fetal development, perinatal, genetic, family, social, and environmental factors that may adversely affect the well-being of the newborn. This immediate systematic newborn assessment by the midwife seeks to determine whether the infant's appearance and behavior are within normal limits and, if deviation from normal is found, whether neonatal and/or pediatric consultation should be sought, along with further follow-up and evaluation of any neonatal problems as required (discussed in the *Examination of the*

Newborn and *Neonatal Care* chapters).

Midwives caring for women and newborns during the first 28 days of life must maintain currency in the basic knowledge necessary for systematic evaluation and management of neonates, including a solid grounding in the physiology of the transition from intrauterine to extrauterine life and provision of rapid and supportive care when necessary, using the reliable, validated, evidence-based practices and current standards for evaluation and management described in this text.

The midwife is privileged to serve women and families at some of the most vulnerable times in their lives. To serve competently, compassionately, and with respect, the midwife seeks to strengthen the family by providing them with advocacy, resources, trust, and understanding that enable them to have a fulfilling experience during pregnancy and birth, and that assist them in making informed choices. Benefiting from the midwife's commitment and support in the form of educational and emotional nourishment, the family can move forward freely and securely in their adaptive process as they grow and flourish.

Lily Hsia

CHAPTER

36

Anatomy and Physiology of the Newborn

CECILIA M. JEVITT

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The first few moments and hours of extrauterine life are among the most dynamic of any that occur during the entire life cycle. At birth, the newborn moves from complete dependence to physiologic independence. Some organs, such as the lungs, make rapid changes that are fully accomplished within days of birth. Other organ systems, such as the hepatic system, take longer to convert to extrauterine function. Overall, the transition to extrauterine life is an ongoing process and part of a continuum that starts with conception and extends through infancy.

Extrauterine transition can be profoundly influenced by prenatal factors as well as intrapartumevents. At each birth, the midwife considers prenatal or intrapartum factors that could cause compromise in the first extrauterine hours of newborn life. Events during conception and the prenatal period that can cause compromise include fetal growth restriction, congenital malformations, genetic disorders, prenatal infections (especially viral), chronic malnutrition, prematurity or postmaturity, maternal chronic disease (e.g., cardiac, renal, hepatic, diabetes), the residual effects of environmental or workplace teratogens, and substance use by the mother. Labor and birth events that can adversely influence extrauterine transition include prolonged labor, maternal fever, hypoxic uterine conditions, birth trauma, infection, passage of meconium, fetal blood loss, use of medications during labor, difficult or operative birth, and any condition causing excessive blood loss. Accurate risk prediction is essential for selection of the appropriate site and personnel for the birth.

The most dramatic and rapid extrauterine transitions that happen immediately after birth occur in four areas: the respiratory system, the circulatory system, thermoregulation, and glucose metabolism. These four areas of transition are interdependent—failure of one can lead to impairment of others, and all must occur successfully for neonatal transition to proceed smoothly.

Respiratory Changes

Prior to birth, the fetus receives oxygen from the maternal circulation via the placenta and umbilical vein. Lung development progresses through distinct stages during gestation.[1] Initially, the airways develop into branches, as do the pulmonary capillaries, although the two systems are separated by a mesenchyme. Terminal alveolar sacs appear during the second canalicular stage, which occurs between 16 and 24 weeks' gestation. During the third, saccular stage that occurs between approximately 28 and 36 weeks' gestation, the space between the pulmonary capillaries and the alveoli diminishes so that capillaries are in direct contact with the alveolar basement membrane. Primitive alveoli develop and become lined with two types of alveolar cells. Gas exchange occurs across the very thin type I epithelial alveolar cells. Interspersed are cuboidal-shaped type II epithelial alveolar cells that produce and store surfactant. Surfactant is the complex phospholipid that lines the alveoli and keeps them partially open during exhalation via its surface-acting properties. The alveolar period starts at 36 weeks and continues throughout childhood. During this phase, more alveoli form, creating a larger surface area for gas exchange. Approximately one-third of the 300 million alveoli are mature at birth; the rest mature after birth.

The primary events that occur in the lung after birth are (1) clearance of alveolar fluid, (2) lung expansion, and (3) circulatory changes that increase pulmonary perfusion. Prior to labor, the lungs are full of fluid, which is secreted in Type II alveolar cells into the alveolar space as a secondary effect of chloride ion secretion. The term fetus begins to experience a decrease in lung fluid in the days before labor, which in turn reduces the amount of fluid that needs to be cleared during the neonate's initial respirations. Clearance of alveolar fluid is facilitated by increased production and activity of epithelial sodium channels (ENaCs). When the ENaCs become active, sodium (Na^+) ions enter the cell from the fetal lung fluid and are actively extruded from the cell into the interstitial space. This process creates an electrochemical gradient that lung fluid and chloride (Cl^-) ions follow, which results in clearing the alveoli of lung fluid (**Figure 36-1**).[2-5] During labor, fetal plasma levels of catecholamines and cortisol increase; these hormones further stimulate reabsorption of lung fluid during labor by up-regulating the genes that produce ENaCs.[3,6] This overall process causes the lungs to change from active fluid secretion to active fluid absorption.[7] Activation of ENaCs is correlated with gestational age. Preterm and near-term infants have less activation of ENaCs after birth and more lung fluid compared to term newborns, which increases the incidence of respiratory distress in these neonates.[2,3]

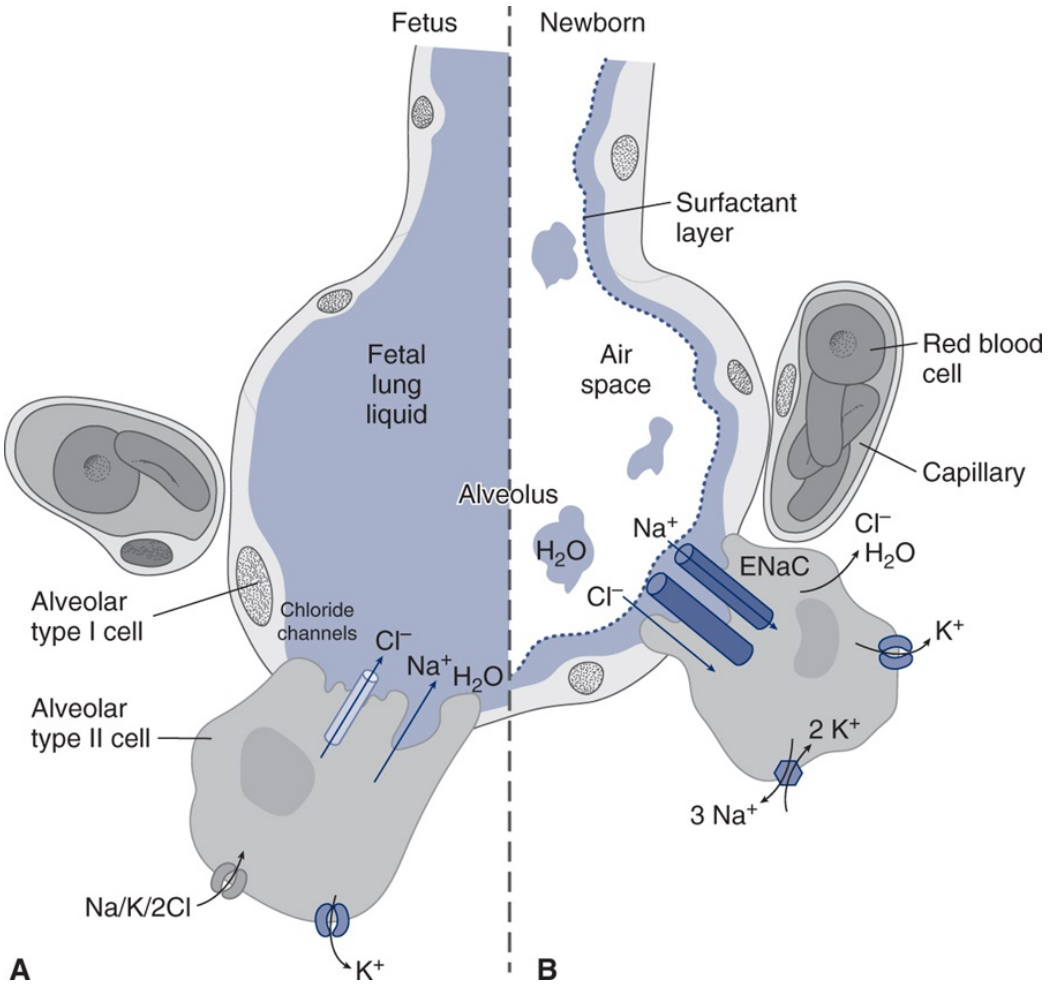


Figure 36-1 A. Fetal alveoli: Cl^- is actively excreted by the type II alveolar cell. NA^+ and water follow passively thereby secreting fluid into the alveolar space. **B.** Newborn alveoli: Epithelial sodium channels (ENaCs) upregulate in the apical surface of the alveolar cell. Na^+ enters the cell through the ENaCs. The increase in cell Na stimulates Na, K^+-ATPase activity on the basolateral aspect of the cell membrane, which drives out 3 Na^+ ions in exchange for 2 K^+ ions. When the net ion movement is from the apical surface to the interstitium an osmotic gradient is created, directing water in the same direction via either aquaporins or diffusion. This process empties the alveolar fluid into the interstitial space and pulmonary circulation.

Abbreviations: CL^-, chloride; ENaCs, epithelial sodium channels; H_2O, water; K^+, potassium, Na^+, sodium.

Based on Keene SD, Bland RD, Jain L. Lung fluid balance in developing lungs and its role in neonatal transition. In: Oh W, Guinard J-P, Baumgart S, eds. *Nephrology and Fluid/Electrolyte Physiology: Neonatology Questions and Controversies.* 2nd ed. Philadelphia, PA: Saunders Elsevier; 2012:221-232.[4]

The squeezing of the thorax that occurs in the last minutes of second-stage labor when the fetus is in the birth canal helps extrude some upper airway fluid, although this effect is small.[6,7] Neonates who experience a cesarean section birth, especially in the absence of labor, do not experience all the processes that contribute to diminution of lung fluid and have an increased risk for transient tachypnea of the newborn (TTN) or respiratory distress syndrome (RDS).

Despite decades of investigation, the phenomena that stimulate the neonate to take the first breath are only partially understood. Several biochemical and mechanical events have been

implicated in the processes. For example, the relative hypoxia of the end of labor and physical stimuli to which the neonate is subjected, such as cold, gravity, pain, light, and noise, cause excitation of the respiratory center. The release of the thoracic pressure and rebound chest expansion at birth may help stimulate a breath. With the neonate's first breath, fluid filling the mouth and trachea is partially released, and air begins to fill the tracheal column. The first active breaths set in motion a seamless chain of events that (1) assists with the conversion from fetal to adult circulation, (2) further empties the lungs of liquid, (3) establishes neonatal lung volume and the characteristics of pulmonary function in the newly born infant, and (4) decreases pulmonary artery pressure.

When the head is born, mucus drains from the nares and mouth. Many newborns gasp and even cry at this time. With the first few breaths, room air starts to fill the large airways of the neonate's trachea and bronchi. Fetal lung fluid remaining in the alveoli is pushed to the lung periphery, where it is absorbed. The first few breaths require positive pressure because room air is flowing into a fluid-filled space.[8] Newborns who are exhausted and compromised by the birth process may need assistance in clearing the fluid and mucus from the upper airway. Use of a suctioning device such as a bulb syringe or wall suction should be limited to situations in which the newborn's respiratory efforts are diminished.[9]

All of the alveoli expand with air over time. The maximum function of the alveoli occurs in the presence of adequate surfactant and adequate blood flow through the pulmonary microcirculation. After 34 weeks' gestation, sufficient quantities of surfactant help to stabilize the walls of the alveoli so that they do not collapse onto themselves at the end of each breath taken by the newborn. This surfactant effect reduces the pressure needed to expand the alveoli during subsequent breaths, thereby decreasing the workload of breathing. Adequate oxygenation is a critical factor in *maintenance* of adequate air exchange. In the presence of hypoxia, the pulmonary vasculature vasoconstricts and oxygen in the alveoli cannot be transported into blood vessels for oxygenation of other areas of the body.

If the first breaths are of adequate volume and pressure, they establish the functional residual capacity (FRC) of the neonate—that is, the gas volume left in the lungs at the end of each expiration. Crying has been found to have a functional purpose.[10] In a crying neonate, expiration is slowed by resistance from the narrowed glottis, which helps maintain end-expiratory pressure within the lungs. The muscles of the diaphragm also slow the rate of lung deflation. Control of expiration ensures a slower lung deflation that minimizes the chance of alveolar collapse and loss of the FRC.[11]

Continuation of breathing occurs secondary to central brain stem activity and the actions of specialized cells within the carotid body. In the term newborn, the chemoreceptors in the carotid body initiate increases in ventilatory effort in response to acidosis and hypercapnia, and decreases in ventilatory effort in response to hypocapnia. Respirations fluctuate—they are not stable and rhythmic for any extended period of time in the neonate. Breathing in a newborn may sound noisy and wet during this period of transition. Additionally, a newborn often experiences short periods of apnea or sighing.[12] Lack of persistent rhythm and occasional periodic breathing (irregular rhythm with short periods of apnea) are normal and more common in a near-term preterm newborn.

There are, however, certain abnormal responses to look for in the newborn. A respiratory

rate consistently faster than 60 breaths per minute, with or without nasal flaring, grunting, or chest retractions associated with breaths, is clearly abnormal at 2 hours of life. Flaring, grunting, and retracting are classic signs of respiratory distress and warrant prompt evaluation by a pediatric healthcare provider. Significant episodes of periodic breathing signal the need for medical intervention before major apnea occurs. Other normal and abnormal responses are summarized in **Table 36-1**.

Table 36-1 **Normal and Abnormal Respiratory Characteristics in the Newborn**

Characteristic	Normal	Abnormal
Respiratory rate	40–60 breaths/minute	< 30 breaths/minute or > 60 breaths/minute
Diaphragm	Diaphragmatic and abdominal breathing	Grunting with expiration
Nasal	Obligate nasal breather	Nasal flaring with breaths
Chest	No retractions	Visible suprasternal or subcostal retraction with breaths

When a newborn needs resuscitation and respiratory support, room air—rather than 100% oxygen—should be used initially.[13] The switch to 100% oxygen should be made only if room air is not sufficient. Although historically 100% oxygen has been used for respiratory resuscitation, it is now known that 100% oxygen can create free oxygen radicals that exacerbate reperfusion injury and decrease cerebral perfusion. Therefore, room air is preferred.

Circulatory Changes

The blood flow from the placenta stops with the clamping of the umbilical cord. This action eliminates the maternal supply of oxygen to the newborn and triggers a subsequent series of reactions. These reactions are complemented by those occurring in the lungs in response to the first breath.

Fetal circulation is characterized by three shunts: (1) the foramen ovale, (2) the ductus arteriosus, and (3) the ductus venosus (**Figure 36-2**). Because the lungs are a closed, fluid-filled organ, they need minimal blood flow; therefore, the pulmonary circulation has a high vascular resistance. Oxygenated blood bypasses the lungs by flowing through the opening between the right and left atria—that is, the foramen ovale—and then into the descending aorta. In the second shunt, blood in the right ventricle does not preferentially flow very far into the pulmonary arteries because of the high-pressure pulmonary vasculature, but rather flows through the ductus arteriosus directly into the descending aorta. Placental prostaglandin, prostacyclin, and low oxygen tension keep the ductus arteriosus open.[14] The third fetal shunt, the ductus venosus, connects the umbilical vein to the inferior vena cava, which allows a portion of fetal blood to bypass the circulation through the liver.

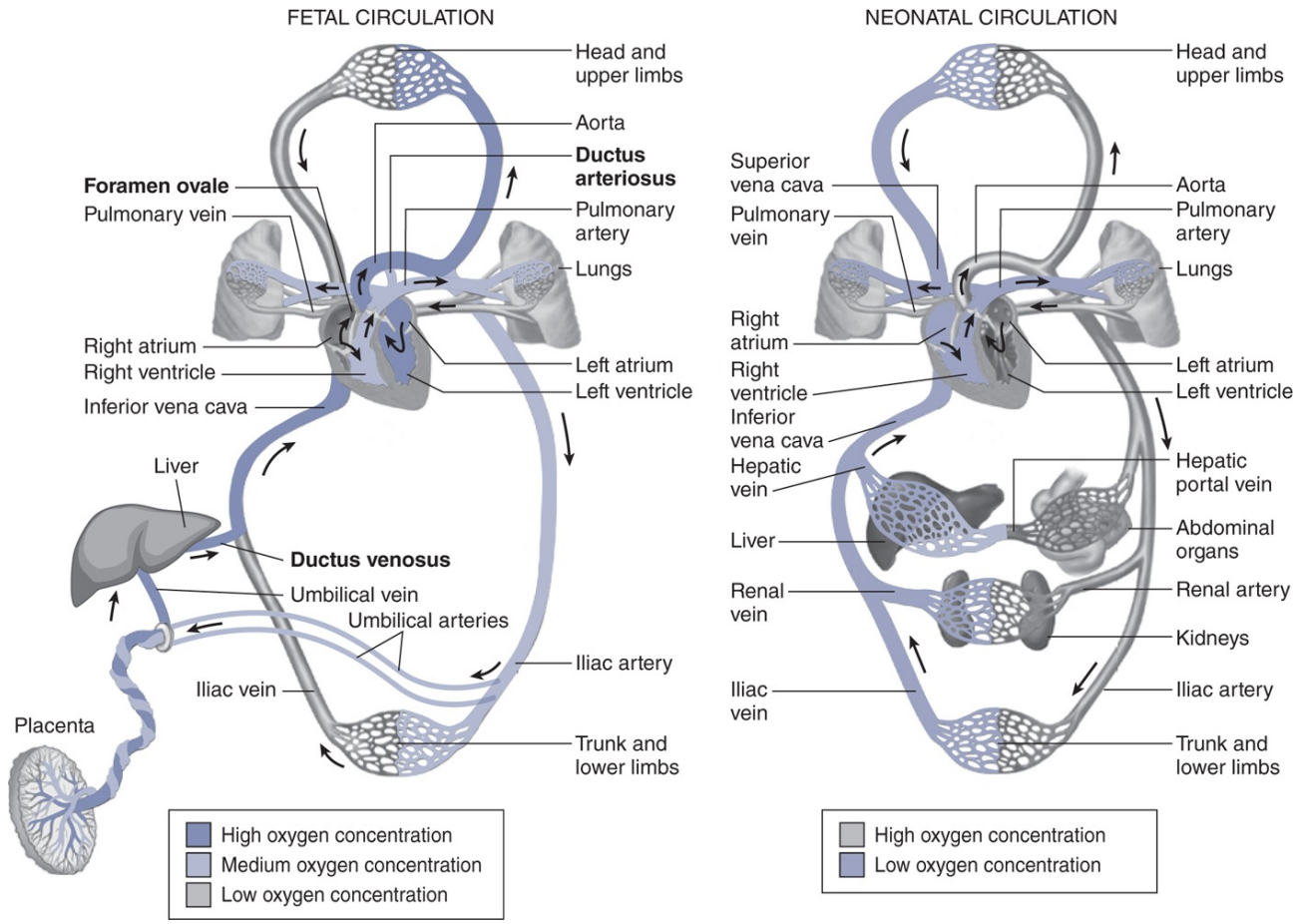


Figure 36-2 Fetal and neonatal circulation.

At birth, oxygen tension rises, and placental prostaglandins and prostacyclin no longer enter the system via the umbilical vein. This causes constriction of the ductus arteriosus. Blood from

the right ventricle is then able to enter the pulmonary circulation, and the oxygenated blood now routinely passing by the ductus arteriosus also facilitates closure of this shunt. Within 48 hours, the ductus arteriosus functionally closes.

Clamping the umbilical cord shuts down the low-pressure system that characterizes the fetal– placental circulatory unit, so that the newborn circulatory system becomes a freestanding, closed system. The immediate effect of cord-clamping is a rise in systemic vascular resistance (SVR). Most importantly, this rise occurs at the same time the newborn takes the initial breaths. The oxygen in those breaths causes the pulmonary vasculature to relax and open. The pulmonary vasculature resistance (PVR) decreases just as the SVR increases, and this shift in pressure, in combination with closure of the ductus arteriosus, encourages blood flow into the pulmonary system. A transitional period ensues during which kinked pulmonary vessels straighten which decreases resistance to blood flow. Over time, the development of elongated and thinner cell walls also increases vessel flexibility.[15]

The combination of increased SVR and lower PVR also changes the pressure of blood flow within the heart. As blood flow increases in the left side of the heart, the foramen ovale closes. The results of the change in SVR and PVR and the closure of the three fetal shunts complete the radical changes in the anatomy and physiology of the heart. Deoxygenated blood enters the neonatal heart, becomes fully oxygenated in the lungs, and is distributed to all other body tissues.

Clinical evidence of these tremendous changes in circulation is manifested as increasingly pink color, normal heart rate, and strong pulses. Although these changes are not anatomically complete for weeks, the functional closure of the foramen ovale and the ductus arteriosus occurs soon after birth. The change from fetal to newborn circulation is intimately related to adequate respiratory function and oxygenation. The term newborn who is stressed by hypoxia, hypoglycemia, cold, or infection may not make the transition smoothly and can develop persistent pulmonary hypertension.[6]

Thermoregulation

The newborn can become rapidly stressed by changes in environmental temperature. The fetal temperature is typically 0.6°C higher than the maternal temperature. Factors that contribute to heat loss in the newborn include the large surface area of a newborn (the ratio of surface area to body weight ratio in a neonate is two times higher than that of an adult), limited subcutaneous fat insulation, and a limited ability to generate heat via shivering.

Prevention of Hypothermia

Newborns can lose heat through four mechanisms: (1) convection, (2) conduction, (3) radiation, and (4) evaporation. Skin-to-skin contact between the neonate and mother is recommended as the initial method for maintaining neonatal body temperature for healthy term infants.[16] Skin-to-skin contact minimizes heat loss via conduction and results in better short-term temperature stability than bundling or placing the neonate under a warmer. This approach should be the first line of treatment for hypothermia, especially in low-resource environments.[17,18]

Coping with Cold Stress

The neonate can create heat in three ways: voluntary muscle activity, shivering, and nonshivering thermogenesis. Muscle activity can generate heat but is of limited benefit in increasing temperature, even in term infants with sufficient muscle strength. Shivering in term neonates is seen only in the most severe cold stress.

Nonshivering thermogenesis refers to utilization of brown fat for heat production. Brown adipose tissue contains more lipids, mitochondria, and capillaries than does white adipose tissue. The primary purpose of brown adipose tissue is to generate heat; such tissue is a nonrenewable resource of the newborn. Brown adipose tissue is located in and around the upper spine, the clavicles and sternum, the kidneys, and major blood vessels. The amount of brown adipose tissue depends on gestational age and is relatively sparse in growth-restricted newborns and preterm infants.

Creation of heat through utilization of brown adipose stores starts at birth, with a surge of catecholamines and withdrawal of the placental suppressors prostaglandin and adenosine. The cold stimulus of leaving the mother's warm body triggers activity in the newborn's hypothalamus that leads to oxidation of stored lipids in brown adipose and subsequent production of heat.[19] Through the mediation of glucose and glycogen, the brown adipose cells convert multiple small intracellular fat vacuoles into heat energy. The heat produced warms the blood before it circulates to peripheral areas of the neonate's body and ensures a stable temperature for other biochemical reactions.[20] In a newborn experiencing hypoglycemia or thyroid dysfunction, however, the use of brown adipose stores does not proceed efficiently.

The sequelae of heat loss in the neonate can cascade quickly into hypoglycemia, hypoxia, acidosis, and respiratory distress (**Figure 36-3**). Pulmonary vasoconstriction occurs during hypothermia, which then causes respiratory distress. These adverse effects are also

consequences of the increased metabolic demands that result from the newborn's attempts to create a neutral thermal zone.

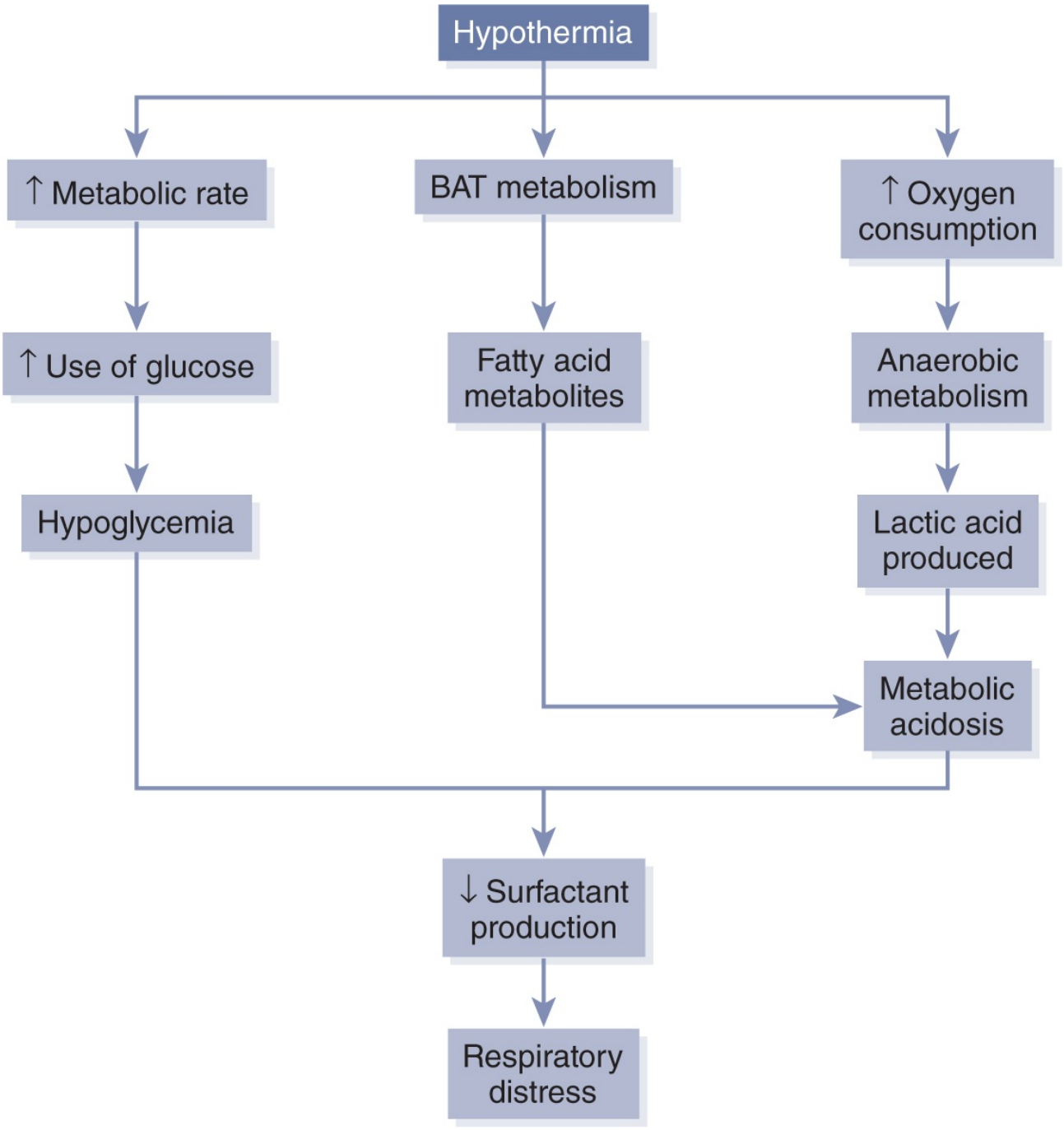


Figure 36-3 Hypothermia in the newborn.

Abbreviation: BAT, brown adipose tissue.

The World Health Organization defines hypothermia as a core temperature less than 36.5°C (97.7°F). Clinical symptoms of hypothermia may be subtle and include a low core temperature and cold skin, pale skin, pallor, hypotonia, lethargy or irritability, poor feeding or vomiting tachypnea, and an increased heart rate.[17] Any newborn who is hypothermic should be evaluated for hypoglycemia because of the high caloric demands associated with nonshivering thermogenesis.[21]

Glucose Regulation

Prior to birth, the fetus is exposed to a nearly constant blood glucose supply; the levels of glucose in fetal blood are approximately 60% to 70% of the maternal levels. In preparation for extrauterine life, the healthy fetus stores glucose as glycogen, especially in the liver. Most glycogen storage occurs in the third trimester. Although any infant can develop symptomatic or asymptomatic hypoglycemia, newborns who are growth restricted, preterm infants, post-term infants, and newborns who have experienced some form of distress prior to birth are at particular risk for hypoglycemia in the newborn period.[22] In all of these neonates, glycogen stores may be smaller than in normal term newborns. The large for gestational age (LGA) neonate is also at risk for hypoglycemia due to a different mechanism which involves increased calorie consumption to maintain body temperature. This occurs in LGA infants whether or not the mother has diabetes, with the newborn's risk increasing as birth weight increases.[23]

The moment the cord stops pulsating or is clamped, the newborn must find a way to maintain a serum glucose level sufficient for normal neonatal brain function. Blood glucose levels initially fall to a nadir at approximately 1 to 2 hours after birth. The physiologic low occurs at approximately 1 to 1.5 hours after birth, with levels subsequently stabilizing at 3 to 4 hours. In a meta-analysis of term newborns, Alkalay et al. recommended that low thresholds for plasma glucose levels in full-term newborns be designated as 28 mg/dL at 1 to 2 hours, 40 mg/dL at 3 to 23 hours, 41 mg/dL at 24 to 47 hours, and 48 mg/dL at 48 to 72 hours.[24] Their analysis included newborns fed via both bottle and breast. These researchers also noted that further study is needed to determine normal blood glucose levels for exclusively breastfed newborns.

The healthy newborn maintains blood glucose levels in one of three ways and generates glucose in the amount of approximately 4 to 8 mg/kg/minute: (1) utilization of breast milk or a substitute; (2) utilization of the glycogen stores (glycogenolysis); or (3) creation of glucose (gluconeogenesis). The newborn should be encouraged to feed as soon as possible after birth. Many newborns are active feeders during the first period of reactivity. This is an ideal time to imprint the infant with the physiologic tasks of breastfeeding. Newborns who are exhausted or stressed from long labor may show minimal interest in feeding and are therefore at increased risk for hypoglycemia.

Newborns who cannot ingest adequate nutrients will create glucose from glycogen. However, glycogenolysis can occur only if the infant has adequate stores of glycogen. Although it is possible for the newborn to create glucose from fats or proteins, the process of gluconeogenesis is inefficient and creates many metabolic by-products.

Routine blood glucose testing of term newborns after an uncomplicated birth is not recommended.[25,26] Nevertheless, all newborns should be monitored for signs of hypoglycemia. Hypoglycemia may be asymptomatic at first. Symptoms, when they occur, can be vague and nonspecific and include jitteriness, cyanosis, apnea, weak cry, lethargy, limpness, and refusal to feed. Long-term sequelae of persistent hypoglycemia include damage to the occipital area of the brain manifesting as seizures, intellectual disability, and attention-deficit disorder.[27] Damage is exacerbated when the hypoglycemia is accompanied by neonatal asphyxia.

Hematologic System

Normal values for a term newborn's blood components are listed in **Table 36-2**. Newborn blood cell indices vary by weeks of gestation, vaginal and cesarean birth without labor, and sex of the newborn.[28,29] At birth, newborns have higher values for hemoglobin and hematocrit compared to adult values. Neonatal blood contains both fetal hemoglobin (HbF), which contains two alpha subunits and two gamma subunits, and adult hemoglobin (HbA), which contains two alpha subunits and two beta subunits. HbF has a higher affinity for oxygen than does HbA, is more stable in an acidic environment, and has a shorter lifespan. After birth, HbA gradually replaces HbF. Because HbF breaks down faster than erythropoiesis, however, newborns experience a physiologic anemia approximately 6 to 8 weeks after birth. The hemoglobin level decreases slowly over the first 7 to 9 weeks after birth, reaching an average of 12.0 g/dL in a 2-month-old infant.

Table 36-2 **Hematologic Values of a Full-Term Newborn**

Component	Optimal Range
Hemoglobin concentration	14.0–20.0 g/dL
RBC count	4,200,000–5,800,000/mm^3
Hematocrit	43–63%
Mean cell diameter	8.0–8.3 µm
MCV	100–120 $µm^3$
MCH	32–40 pg
MCHC	30–34%
Reticulocyte count	3–7%
Nucleated red blood cell count	200–600/mm^3
White blood cell count	10,000–30,000/mm^3
Granulocytes	40–80%
Lymphocytes	20–40%
Monocytes	3–10%
Platelet count	150,000–350,000/mm^3
Serum iron concentration	125–225 mcg/dL
Total iron-binding capacity	150–350 mcg/dL

Abbreviations: MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell.

Based on Henry E, Christensen R. Reference intervals in neonatal hematology. *Clin Perinatol.* 2015;42:483-497[28]; Christensen RD, Henry E, Jopling J, Wiedmeier SE. The CBC: reference ranges for neonates. *Semin Perinatol.* 2009;33:3-11.[29]

Delayed Cord-Clamping

The initial hemoglobin/hematocrit values in a newborn can be influenced by the timing of clamping of the cord and the newborn's position immediately after birth. Delay in clamping the umbilical cord after birth allows a passive placental transfusion, which increases the neonate's blood volume.[30] The physiology of delayed cord-clamping is reviewed in the *Umbilical Cord-Clamping at Birth* appendix in the *Second Stage of Labor and Birth* chapter. The placental transfusion can continue for several minutes after birth to deliver approximately 30% more blood volume and 50% more iron-rich blood cells compared to immediate clamping.[30] Delaying cord-clamping has several benefits: (1) The neonate receives a bolus of oxygenated blood during the first breaths, (2) the volume expansion promotes pulmonary capillary perfusion in the newborn, (3) the more rapid achievement of adequate oxygenation leads to closure of fetal structures such as the ductus arteriosus, and (4) the infant has a decreased risk of iron-deficiency anemia at 4 to 6 months of life.[30-33] Delaying cord-clamping until cord pulsation ceases has been shown to be safe and is recommended to increase neonatal iron storage at birth despite being associated with a slightly increased risk for hyperbilirubinemia.[34,35]

To support the physiologic transfusion that occurs over the first minutes of life, the newborn is placed on the mother's abdomen (skin-to-skin) with the cord intact. This position promotes modest amounts of blood flow to the newborn without the potential danger of a forced and large bolus of blood. By 5 minutes, most of the blood flow from the cord to the neonate is usually accomplished.

Red Blood Cells and Hyperbilirubinemia

The newborn's red blood cells (RBCs) have a short lifespan of 80 days on average, compared to the adult RBC lifespan, which averages 120 days. This rapid cell turnover creates more by-products of cell breakdown that must be metabolized, including bilirubin. Newborns also have a reduction in the activity of the protein uridine diphosphate glucuronosyltransferase (UGT), which helps to conjugate bilirubin and prepare it for excretion. These two factors are most responsible for the physiologic jaundice seen in many newborns.[36] Total serum bilirubin peaks at the approximately 3 days of postnatal life. Ninety-six percent of term newborns will have a total serum bilirubin of less than 17 mg/dL at 96 hours of age, so levels higher than this concentration are considered pathological.[36] Significant jaundice before post-natal day 3 may be pathological, and any visible jaundice in the first 24 hours of life is of significant concern. There is a correspondingly high reticulocyte count in newborns, reflecting the high rate of formation of new red blood cells.

White Blood Cells

The average white blood cell (WBC) levels in a newborn range from 10,000 to 26,000/mm^3. There can be a further elevation in the WBC count in a normal newborn during the first 24 hours of life, with this level then gradually decreasing over the next 3 to 5 days. Because of these physiologic shifts, it is more useful to focus on neutrophils as a marker for infection. Limits for normal neutrophil shifts in term newborns have recently been changed to reflect a

normal peak of 28,500/mm^3 at 8 hours of age and 13,000/mm^3 at 72 hours.[37] Labor, meconium aspiration, prolonged periods of crying, and maternal fever can cause a shift upward in the neutrophil count.

Platelets and Clotting Factors

Platelet counts in newborns should be within the same range as adult values. Levels lower than 150,000/mm^3 should prompt further medical evaluation. However, platelet aggregation can vary in term newborns. In the first month of life, there is an overall deficiency of many of the clotting factors that are dependent on vitamin K. Because of this general deficiency, it is customary to give newborns supplementary vitamin K after birth. The most common route of administration for vitamin K is intramuscular, as studies have not demonstrated a reliably effective oral vitamin K dose.[38,39] Prothrombin time, thrombin time, and partial thromboplastin time are slightly prolonged.

The Newborn Immune System

The neonatal immune system is immature in a number of ways, including having incomplete physical and chemical barriers in the gastrointestinal system, limited and delayed secretory immunoglobulin A (IgA) production, incomplete complement cascade function, and decreased anti-inflammatory mechanisms within the respiratory and gastrointestinal (GI) tract. This functional immaturity renders the neonate vulnerable to many infections and allergic responses. Innate immunity is the primary line of defense for the neonate.[40]

Innate Immunity

Innate immunity consists of cellular and noncellular first responders to infection. Some examples of innate immunity include (1) the barrier protection offered by the skin, mucous membranes, and intestinal mucosa; (2) the sieve-like action of the respiratory passages; (3) the colonization of skin and intestine by protective microbes; (4) the chemical protection offered by the acidic environment of the stomach; and (5) the actions of breast milk that support neonatal innate immune function.

Skin and mucous membranes are colonized as the fetus passes through the vaginal canal. The vernix caseosa contains antimicrobial proteins and peptides (APPs) and forms a "microbial shield" on the newborn's body.[41,42] The presence of vernix also moistens the skin and increases its acidity, which inhibits growth of pathogenic bacteria.[43] The inflammatory skin reaction of normal newborns called erythema toxicum or "newborn rash" contains the antimicrobial peptide LL-37, indicating that this skin condition may be a manifestation of bacterial colonization.[40,42,44]

Colostrum that is ingested in the first few days of life and breastfeeding in the first month of life are powerful stimulants of the infant's innate immune response. Breast milk contains many bioactive substances that facilitate development of the infant's immune system. In the infant's gastrointestinal tract, breast milk inhibits binding of many toxins, inhibits inflammatory responses, and facilitates colonization of commensal bacteria that will make up the microbiome.[44] Breast milk influences expression of immune signaling genes in intestinal epithelial cells. These toll-like receptors (TLRs) play a central role in immune signaling by recognizing patterns of pathogen-associated molecular signals.[44]

Innate immunity is also provided at the cellular level. Three cell types—polymorphonuclear neutrophils (PMNs), monocytes, and macrophages—act via phagocytosis to engulf and kill foreign antigens. The PMNs will eventually become the primary phagocytes in the defense of the host. However, in the neonate, the PMN is compromised both in ability to mobilize, move in the right direction, and adhere to inflammatory sites.[42,45] These deficiencies do not reflect inadequate numbers of the PMN cells, but rather inadequate chemotaxis and opsonization.

Chemotaxis refers to the organized movement of a phagocytic cell—that is, its actual travel around the body. Opsonization is the alteration, or "marking," of the cell surface of a foreign microbe or antigen; it promotes the destruction of foreign cells. The immaturity of chemotaxis and opsonization in the newborn decreases the effectiveness of the phagocytic response and leads to one of the main weaknesses of the neonatal immune system: the inability to localize

infection. Thus, the risk for systemic infection is higher in newborns than in older children and adults.

The innate immune system also includes numerous noncellular proteins, such as complement molecules, acute-phase reactants, cytokines, chemokines, coagulation proteins, and vasoactive substances. The process of phagocytosis is enhanced if the foreign cells are prepared by complement components. Natural killer (NK) cells contribute to innate immunity, but their function is compromised in the neonate, as they have limited cytolytic ability.[46]

Signs of infection can be subtle in the newborn; changes in activity, tone, color, or feeding, for example, may be difficult to recognize in the early stages of an infection. In addition, lack of fever does not exclude the possibility of infection. Thus, observation for symptoms of systemic infection is one of the most important reasons for healthcare professional assessments of the newborn.

Adaptive Immunity

Adaptive immunity is the acquired response to specific pathogens. It is divided into two components: cellular and antibody mediated (humoral). The cellular adaptive immune response is modulated through T-cell and B-cell lymphocytes. T cells have two main roles: killing infected cells and aiding other cells by stimulation of cytokines. These cells are found in high levels in the newborn, but are slow to respond and require increased stimulus to generate their activity compared to later in life.[45] The T cells that develop immunological memory—that is, CD8 and CD4 cells—are present in low levels in the neonate; their populations increase slowly throughout childhood. Until the newborn has been exposed to some antigens, these T cells do not offer significant protection. Thus, it is not until 4 to 6 weeks after birth that the infant will experience minimal protection through the activity of these cells.[47]

The activation of the antibody-mediated B cells is dependent on T-cell activity. Thus, as a consequence of diminished T-cell functionality in the newborn, B-cell responses are also deficient, having a delayed onset and shorter duration. Bacteria may also trigger B-cell development. Recent evidence indicates that B cells are activated by maternal intestinal bacteria during early newborn intestinal colonization.

Passive Immunity

The neonate is born with passive immunity to viruses and bacteria via transplacental passage of immunoglobulin G (IgG) from the mother. Antibodies passed transplacentally to the fetus during the last trimester of pregnancy continue to protect the infant for the first few months after birth.[45] The newborn is dependent on this form of passive immunity until approximately 6 months of age when significant amounts of IgG are produced by the infant. Other antibody immunoglobulins, such as IgM and IgA, are larger than IgG and do not cross the placenta. Breast milk, however, contains a significant amount of secretory IgA that protects the infant from microbes in the maternal intestinal tract.

Gradually, the young infant will begin to produce adequate circulating antibodies of the IgG

class. This process takes time, and mounting the full antibody response to a foreign antigen is not possible until well into early childhood.

In summary, because of a variety of deficiencies in both natural and acquired immunities, the neonate is vulnerable to infection. Neonatal response to infection is sluggish and inadequate, leading to a predisposition to systemic rather than localized infections. Vaginal birth and breastfeeding both help establish healthy microbial colonization, an effect that persists throughout infancy. Breastfeeding has additional direct and indirect mechanisms that support and stimulate development of immunity in the newborn.

The Newborn Gastrointestinal Tract

The gastrointestinal system in a term newborn exhibits varying levels of maturity. Prior to birth, the term fetus exhibits sucking and swallowing motions. Mature gag and cough reflexes are intact at birth. Meconium, although sterile, contains debris from the amniotic fluid, which confirms that the fetus ingests amniotic fluid and that the fluid passes through the gastrointestinal tract.

The ability of the term newborn to ingest and digest exogenous food sources is limited secondary to varying amounts of digestive enzymes and hormones in all portions of the gastrointestinal tract. Newborns are less able to digest proteins and fats than are adults. Carbohydrate absorption is relatively efficient but still less than the adult ability. The newborn is particularly efficient in absorbing monosaccharides such as glucose, as long as the amount of glucose is not too large.

The cardiac sphincter—the juncture of the lower esophagus and the stomach—is incomplete at birth, which contributes to frequent regurgitation of stomach contents in newborns and young infants. The capacity of the stomach itself is limited, holding less than 30 mL in a term newborn.

The newborn's intestines are also relatively immature. Prior to birth, this organ is semi-dormant. Normal extrauterine transition includes a striking increase in blood flow and a decrease in vascular resistance that results in increased blood flow to the neonate's rapidly growing intestines.[48] The underlying musculature is thinner and less efficient in the newborn than in the adult, leading to unpredictable peristaltic waves. The folds and villi of the intestinal wall are not fully developed. The epithelial cells lining the newborn's small intestine do not have the rapid cell turnover that promotes most effective absorption. This immaturity of the intestinal epithelium affects the ability of the intestine to be protected from harmful substances, and the decreased vascular resistance increases the risk of damage subsequent to hypoxia, should hypoxia occur.[48]

During early infancy, the newborn faces the substantial task of "gut closure," the process by which the epithelial surfaces of the intestine become impermeable to antigens. Prior to gut closure, the infant is vulnerable to bacterial/viral infection as well as to allergenic stimulation through intestinal absorption of large molecules.

Among the defenses present in the GI tract are the chemical barriers of increased acidity, the digestive enzymes that break down large molecules, and the secretory IgA that lines the small intestine. Secretory IgA dampens inappropriate immune activation and binds with harmful antigens.[49,50] In the breastfed newborn, maternal secretory IgA restricts immune activation and bacterial attachments.[48] Unlike other types of immunoglobulin, secretory IgA is not absorbed and works at the local level in the intestine.[51]

The beginning of oral feedings stimulates the intestinal lining to mature via promotion of rapid cell turnover and production of microvillus enzymes such as amylase, trypsin, and pancreatic lipase. All enteral feedings, even in very small amounts, lead to helpful surges in gastrointestinal trophic factors—mainly hormones that lead to a full maturation in functioning.[52] The colon in the newborn conserves water less efficiently than that in the adult, which predisposes the newborn to complications from water loss. This makes a diarrheal

illness potentially serious in a young infant.

The Newborn Microbiome

In humans, the entire gastrointestinal tract serves as a component of the natural immune system, which collectively represents a system of defense for the host. Ongoing research on maternal and newborn microbiomes is replacing former theories of newborn bacterial colonization. Microbiomes flourish on the skin, in the mouth, and in the gut, and appear to influence digestion, the immune system, endocrine function, and possibly neurologic signaling.[53] At birth, the rapid colonization of the sterile fetal intestine with commensal bacteria performs valuable functions and prevents pathogenic bacteria from dominating this microbiota.

Following a vaginal birth, the first colonization is with maternal vaginal bacteria. Following a cesarean section birth, the first colonization is with bacteria from the operating room environment. Thus, after birth, the intestinal epithelium is exposed to trillions of bacteria. This diverse population of microbes is then involved in creating an effective barrier against bacteria in a process known as mucosal host–microbial homeostasis. The initial mucosal host–microbial interaction begins development of an intestinal microbiota that recognizes healthy bacteria and repels harmful ones.[49] These microorganisms also assist in digestion and breakdown of toxins, and the synthesis of vitamins.

Many factors have the potential to change neonatal microbiomes: maternal stress and diet, antibiotic exposure, cesarean birth, and infant diet. Differences have been demonstrated between preterm and term neonate microbiomes, between the microbiomes of newborns born vaginally and by cesarean section, between breastfed and formula-fed neonates, and between those exposed to antibiotics before and after birth and those unexposed to antibiotics. Research is being conducted on ideal ways of seeding the neonatal microbiome, such as exposing newborns born by cesarean section to maternal vaginal secretions. The outcomes of this emerging research are not conclusive enough yet to affect current newborn care practices.[53]

The Renal System

The term newborn has some structural and functional immaturity within the renal system, most of which resolves spontaneously during the first month of life.[54] The newborn kidney has less renal blood flow and a lower glomerular filtration rate compared to that of an adult. Tubular function is immature, which can lead to large sodium losses and other electrolyte imbalances.[55] The newborn is unable to concentrate urine very well, which is reflected in the low specific gravity (as low as 1.004) and osmolality of urine. All of these renal limitations are accentuated in the preterm infant. These aspects of renal function are typically only a problem for sick or stressed newborns. For example, if a newborn needs intravenous fluids or medications, the limitations of the still developing renal system increase the risk of iatrogenic fluid overload.[55]

The newborn excretes a small quantity of urine in the first 48 hours of life, often as little as 30 to 60 mL. This urine should not include any protein or blood. Large amounts of cellular debris may indicate injury or irritation within the renal system.

Conclusion

Newborn physiology comprises fascinating, dynamic, and rapidly changing systems that both protect the newborn during adjustment to extrauterine life and exhibit significant vulnerabilities to certain stressors such as cold and infection. Basic knowledge of newborn physiology can help midwives care for newborns and teach parents how to care for their infants as well.

Resources

Organization	Description	Webpage
Global Health Media	Training video on newborn heel stick procedure. These training videos have been provided by the Global Health Media Project and are based on standards of care described in *Care of the Newborn Reference Manual*, Save the Children, 2004; *Managing Newborn Problems*, World Health Organization, 2003; and *Integrated Management of Childhood Illnesses Chart Booklet*, World Health Organization, 2011.	https://youtu.be/m6MBM0pylpA?list=PLXeu9kWMOoFLMXFrdo67Xx06HiCa3Q6Vv
Global Library of Women's Medicine (GLOWM)	Korones S. Anatomic aspects of fetal development. This online site has detailed descriptions of fetal anatomy, growth, and development.	http://editorial.glowm.com/ch075/framesets/006f.html?p=glowm.cml/section_view&articleid=102
Quizlet	This site provides online reviews, including flashcards and quizzes on newborn lab values such as hemoglobin and hematocrit.	https://quizlet.com/4042928/normal-newborn-blood-lab-values-flash-cards/

References

1. Laudy JA, Wladimiroff JW. The fetal lung 1: developmental aspects. *Ultrasound Obstet Gynecol.* 2000;16(3):284-290.
2. Matalon S, Bartoszewski R, Collawn J. Role of epithelial sodium channels in the regulation of lung fluid homeostasis. *Am J Physiol Lung Cell Mol Physiol.* 2015;309(11):L1229-L1238.
3. Katz C, Bentur L, Elias N. Clinical implication of lung fluid balance in the perinatal period. *J Perinatol.* 2011;31(4):230-23
4. Keene SD, Bland RD, Jain L. Lung fluid balance in developing lungs and its role in neonatal transition. In: Oh W, Guinard P, Baumgart S, eds. *Nephrology and Fluid/Electrolyte Physiology: Neonatology Questions and Controversies*. 2nd ed. Philadelphia, PA: Saunders Elsevier; 2012:221-232.
5. Graves BW, Haley MM. Newborn transition. *J Midwifery Womens Health*. 2013;58(6):662-678.
6. Swanson J, Sinkin R. Transition from fetus to newborn. *Pedatr Clin North Am.* 2015;62:329-343.
7. Morton S, Brodsky D. Fetal physiology and the transition to extrauterine life. *Clin Perinatol.* 2016;43:395-407.
8. Vyas H, Field D, Milner AD, Hopkin IE. Determinants of the first inspiratory volume and functional residual capacity at birth. *Pedatr Pulmonol.* 1986;2:189-193.
9. Evans M. Does medical evidence support routine oronasopharyngeal suction at delivery? *J Okla State Med Assoc*. 2016;109(4-5):140-142.
10. Te Pas AB, Wong C, Kamlin CF, Dawson JA, Morley CJ, Davis PG. Breathing patterns in preterm and term infants immediately after birth. *Pediatr Res*. 2009;3(65):352-356.
11. Hutten GJ, van Eykern LA, Latzin P, Kyburz M, van Aalderen WM, Frey U. Relative impact of respiratory muscle activity on tidal flow and end expiratory volume in healthy neonates. *Pediatr Pulmonol.* 2008;43(9):882-891.
12. MacLean J, Fitzgerald D, Waters K. Development changes in sleep and breathing across infancy and childhood. *Paediatr Resp Rev.* 2015;16:276-284.
13. Wyckoff MH, Aziz K, Escobedo MB, et al. Part 13: neonatal resuscitation: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(18 suppl 2):S543-S560.
14. Backer CL, Eltayeb O, Monge MC, Mazwi ML, Costello JM. Shunt lesions part I: patent ductus arteriosus, atrial septal defect, ventricular septal defect, and atrioventricular septal defect. *Pedatr Crit Care Med*. 2016;17(8S1):S302-S309.
15. Lakshminrushimha S, Sauqstad OD. The fetal circulation, pathophysiology of hypoxemic respiratory failure and pulmonary hypertension in neonates and the role of oxygen therapy. *J Perinatol*. 2016;36(S2):S3-S11.
16. Association for Women's Health, Obstetric and Neonatal Nurses. Immediate and sustained skin-to-skin contact for the healthy term newborn after with AWHONN Practice Brief Number 5. *JOGN*. 2016;45(6):842-844.
17. Lunze K, Hamer DH. Thermal protection of the newborn in resource-limited environments. *J Perinatol.* 2012;32(5):317-324.
18. World Health Organization, United Nations Population Fund, UNICEF. *Pregnancy, Childbirth, Postpartum and Newborn Care: A Guide for Essential Practice*. 3rd ed. Geneva, Switzerland: World Health Organization; 2010.
19. Ravussin E, Galgani JE. The implication of brown adipose tissue for humans. *Annu Rev Nutr.* 2012;31(1):33-47.
20. Tews D, Wabitsch M. Renaissance of brown adipose tissue. *Hormone Res Pediatr.* 2011;75:231-239.
21. Soll RF. Heat loss prevention in neonates. *J Perinatol.* 2008;28(suppl 1):S57-S59.
22. Rozance P, Hay W. New approaches to management of neonatal hypoglycemia. *Matern Health Neonatol Perinatol.* 2016;2:3.
23. Weissmann-Brenner A, Simchen MJ, Zilberberg E, et al. Maternal and neonatal outcomes of large for gestational age pregnancies. *Acta Obstet Gynecol Scand*. 2012;91(7):844-849.
24. Alkalay A, Sarnat H, Flores-Sarnat L, Elashoff J, Farber S, Simmons C. Population meta-analysis of low plasma glucose thresholds in full-term normal newborns. *Am J Perinatol.* 2006;2(23):115-119.
25. Adamkin D. Metabolic screening and postnatal glucose homeostasis in the newborn. *Pediatr Clin North Am.* 2015;62:385-409.
26. Stanley C, Rozance PJ, Thornton PS, et al. Re-evaluating "transitional neonatal hypoglycemia": mechanism and implications for management. *J Pediatr.* 2015;166(6):1520-1525.
27. Jobe A. Transitional neonatal hypoglycemia. *J Pediatr.* 2015;166(6):1329-1332.
28. Henry E, Christensen R. Reference intervals in neonatal hematology. *Clin Perinatol.* 2015;42:483-497.
29. Christensen RD, Henry E, Jopling J, Wiedmeier SE. The CBC: reference ranges for neonates. *Semin Perinatol.* 2009;33:3-11.
30. Katheria AC, Lakshminrusimha S, Rabe H, McAdams R, Mercer JS. Placental transfusion: a review. *J Perinatol.* 2017;37(2):105-111.
31. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on maternal and neonatal outcomes. *Cochrane Database Syst Rev.* 2013;7:CD004074. doi:10.1002/14651858.CD004074.pub3.
32. Mercer JS, Skovgaard RL. Neonatal transitional physiology: a new paradigm. *J Perinatal Neonatal Nurs*. 2002;15(4):56-75.

33. Chaparro CM. Timing of umbilical cord clamping: effect on iron endowment of the newborn and later iron status. *Nutr Re* 2011;69:S30-S36.
34. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on materna and neonatal outcomes. *Evid Based Child Health*. 2014;9(2):303-397.
35. Reiser DJ. Neonatal jaundice: physiologic variation or pathologic process. *Crit Care Nurs Clin North Am.* 2004;16(2):257 269.
36. Muchowski KE. Evaluation and treatment of neonatal hyperbilirubinemia. *Am Fam Physician*. 2014;89(11):873-878.
37. Prosser A, Hibbert J, Strunk T, et al. Phagocytosis of neonatal pathogens by peripheral blood neutrophils and monocytes from newborn preterm and term infants. *Pediatr Res*. 2013;74(5):503-510.
38. Phillippi J, Holley SL, Morad A, Collins MR. Prevention of vitamin K deficiency bleeding. *J Midwifery Womens Health*. 2016;51(5):632-636.
39. Ipema HJ. Use of oral vitamin K for prevention of late vitamin K deficiency bleeding in neonates when injectable vitamin K is not available. *Ann Pharmacother*. 2012;46(6):879-893.
40. Gil A, Kenney L, Mishra R, Watkin L, Aslan N, Selin L. Vaccination and heterologous immunity: educating the immune system. *Trans R Soc Trop Med Hyg*. 2015;109:62-69.
41. Tollin M, Bergsson G, Kai-Larsen Y, et al. Vernix caseosa as a multi-component defence system based on polypeptides, lipids and their interactions. *Cell Molecul Life Sci*. 2005;62(19):2390-2399.
42. Kumar S, Bhat B. Distinct mechanisms of the newborn innate immunity. *Immunol Lett*. 2016;173:42-54.
43. Visscher MO, Narendran V, Pickens W, et al. Vernix caseosa in neonatal adaptation. *J Perinatol*. 2005;25(7):440-446.
44. He Y, Lawlor NT, Newburg DS. Human milk components modulate toll-like receptor–mediated inflammation. *Adv Nutr*. 2016;7(1):102-111.
45. Ygberg S, Nilsson A. The developing immune system: from foetus to toddler. *Acta Paediatr*. 2012;101(2):120-127.
46. Lee Y, Lin S. Neonatal natural killer cell function: relevance to antiviral immune defense. *Clin Devel Immunol*. 2013;2013:427696. doi:10.1155/2013/427696.
47. Lusyati S, Hulzebos CV, Zandvoort J, Sauer PJ. Levels of 25 cytokines in the first seven days of life in newborn infants. *BMC Res Notes*. 2013;2013;6:547. doi:10.1186/1756-0500-6-547.
48. Walker A. Intestinal colonization and programming of the intestinal immune response. *J Clin Gastroenterol*. 2014;48(S1):S8-S11.
49. Nuriel-Ohayon M, Neuman H, Koren O. Microbial changes during pregnancy, birth and infancy. *Front Microbiol*. 2016;14(7):1031.
50. Turfkruyer M, Verehasselt V. Breast milk and its impact on maturation of the neonatal immune system. *Curr Opin Infect Dis*. 2015;28(3);199-206.
51. Harris NL, Spoerri I, Schopfer JF, et al. Mechanisms of neonatal mucosal antibody protection. *J Immunol*. 2006;177(9):6256-6262.
52. Neu J. Gastrointestinal maturation and implications for infant feeding. *Early Hum Develop*. 2007;83:767-775.
53. Yang I, Corwin E, Brennan P, Jordan S, Murphy J, Dunlop A. The infant microbiome: implications for infant health and neurocognitive development. *Nurs Res*. 2016;65(1):76-88.
54. Saint-Faust M, Boubred F, Simeoni U. Renal development and neonatal adaptation. *Am J Perinatol*. 2014;31(9):773-780.
55. Sulemnji M, Vakili K. Neonatal renal physiology. *Semin Pediatr Surg*. 2013;22:195-198.

CHAPTER

37

Examination of the Newborn

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Introduction

The complete newborn examination is composed of three parts: (1) a newborn history, (2) the gestational age assessment, and (3) the physical examination. Based on the results of this examination, the midwife will ascertain a newborn's level of wellness and identify potential or existing problems. A plan of care appropriate for that newborn and family can then be developed. This chapter reviews the basic newborn examination. An extensive guide to the content and findings of the newborn physical examination is provided in **Appendix 37A**.

Appreciation for the role of the newborn history and the gestational age assessment has grown in recent years. With new knowledge of genetic inheritance, genomics, fetal development, and the effect of maternal conditions on fetal well-being, it is now recognized that birth is merely a continuation of a life already influenced by many variables. An accurate history of these variables is a critical piece of the full assessment of the newborn.

Approach to the Newborn Examination

The goal in examination of the newborn is to maximize the amount of information gathered while minimizing distress to the newborn and parents. Parents usually show appropriate concern about any handling or examinations that cause their child to cry. It is best to examine the newborn approximately 1 hour after a feeding, when the infant is most likely to be content. The general order of the examination also should be modified based on infant status at the time of the examination. For example, if the infant is asleep or in a quiet alert phase, collection of assessments that benefit from this status—specifically heart rate and respiratory rate—should be performed first. Once these data are collected, more potentially upsetting examination techniques can follow.

Prior to starting a physical examination and gestational age assessment, the maternal records during pregnancy, labor and birth records, and neonatal records should be reviewed. The examination should be performed in an environment that provides proper lighting, cleanliness, and warmth. All equipment needed for the examination should be organized before beginning, including gestational age and physical examination forms (or access to an electronic health record), measuring tape and length growth chart, stethoscope, and ophthalmoscope. Familiarity with the correct use of equipment and the guidelines for accurate measurement is essential. As a case in point, a study published in 2009 reported that of 200 children admitted to a large, urban hospital, only 24% had their linear measurement plotted correctly.[1]

Prior to making any physical contact with the newborn, the goal and purpose of the examination is explained to the mother and/or family members and permission to handle the newborn is obtained. The midwife then performs a 20-second scrub of hands and forearms with a bactericidal soap. Nonsterile gloves also frequently are used by midwives to perform the examination, especially in hospital settings. During the examination, feedback can be offered to the parents about the strengths and attributes of their newborn as well as any significant findings. As part of this exchange, the midwife can provide the parents with information and further assess their readiness for infant care.

History

The prenatal and family history of a newborn is gathered through chart review and interviews with the mother and possibly other members of the newborn's family. As the information is gathered, the midwife develops a mental list of areas of potential concern, including environmental, genetic, social, maternal medical, perinatal, and neonatal factors.

Environmental and Genetic Factors

Environmental factors that influence the newborn can include occupational exposure of the mother or father to hazardous materials such as X rays, other radiation, solvents, infectious agents, fumes, and gases. In the home, environmental factors that can cause fetal or neonatal damage or illness include unsafe drinking water, ingestion of raw or undercooked meats, exposure to paints or solvents, infections transmitted by house pets or gardening activities, poorly ventilated heaters, living near a hazardous waste site, and exposure to toxins brought to the home from a family member's workplace.

Inquiring about the well-being of other children in the home and their ages is important to assess for exposure to illness that could affect the fetus prenatally such as varicella, cytomegalovirus (CMV), parvovirus, or other viral illness.

The genetic history should include information about any family members, alive or deceased, with physical or mental disorders or inherited diseases. Any family members with unexplained multiple pregnancy losses also should be noted. Current health status of the newborn's parents, siblings, and second-degree relatives (aunts, uncles) should be included.

Social Factors

The social history includes information about the woman's place of residence, access to transportation, and social supports. Information gathered includes who will be the designated caregiver and who lives in the home. If the woman has other children, the midwife should determine whether they live with her and, if not, the reason for their absence. Inquiring about the health and well-being of the children, paying particular attention to any accident patterns that may indicate abuse or neglect, is important as well. A key question used in the past to indicate the status of care received by other children in the home was those children's immunization status. As more families choose among various immunization schedules, the decision making regarding this topic should be further explored. Is the family making an informed decision regarding the immunization of their children, or is a lack of immunization due to neglect or another problem, such as lack of access to healthcare services?

Obtaining a description of the social environment is an opportune time to screen for substance use by the mother and other members of the newborn's household. Maternal exposure to even passive cigarette smoke may affect fetal development and growth,[2] while newborns and small children exposed to passive smoke are at risk for respiratory illness, including sudden infant death syndrome (SIDS).[3] Newborns living in a home where there is regular illicit substance use or overuse of prescription medications may also be vulnerable to

neglect. In addition, the possibility for exposure to violence, either direct or secondary, is increased in such a setting.

Maternal Medical and Perinatal Factors

Basic data about the newborn's mother include maternal age, last normal menstrual period, and expected date of birth. The adequacy of prenatal care should be noted, along with any prenatal problems listed in the prenatal record. All laboratory values and prenatal testing, including ultrasound reports, as listed in **Table 37-1** are reviewed.

Table 37-1 Prenatal Tests Reviewed Prior to Newborn Examination

Purpose of Test	Prenatal Screening Tests
Blood type	ABO blood type and Rhesus (Rh) type, antibody screen for isoimmunization
Maternal infection	Hepatitis B surface antigen, *Chlamydia trachomatis* screen, syphilis, group B streptococcus colonization test, human immunodeficiency virus (HIV) screen, *Neisseria gonorrhoeae*
Diabetes (including gestational diabetes)	Plasma glucose level as a screen for gestational diabetes, or hemoglobin A1c (HbA1c) if screened for type 2 diabetes
Birth defects	Carrier screening (e.g., cystic fibrosis), chromosomal anomaly screening (e.g., Down syndrome screening in first and second trimester), to diagnostic test (e.g., chorionic villus sampling or amniocentesis)
Fetal ultrasound	Fetal survey for morphology, amniotic fluid volume, gestational age
Substance abuse	Toxicology screen

A wide variety of maternal medical, prenatal, and intrapartum conditions can adversely affect the newborn's health and well-being. **Table 37-2** presents a selected list of maternal medical conditions and fetal and newborn implications that may be associated with those conditions.[4-13]

Table 37-2 Neonatal Complications Associated with Maternal Medical and Perinatal Conditions

Factor	Possible Newborn Complications
Maternal Medical Conditions	
Cardiac disease	Chronic intrauterine hypoxia
Diabetes (type 1 or type 2)[a]	Neonatal risks are directly related to the onset, duration, and degree of glucose dysregulation in the mother. Risks include congenital anomalies; miscarriage or stillbirth; perinatal asphyxia; intrauterine growth restriction; large for gestational age; polyhydramnios; birth injury, especially brachial plexus injury following shoulder dystocia; polycythemia; hyperbilirubinemia; hypoglycemia; and respiratory distress.
	Long-term: The infant has an increased risk of diabetes, obesity, and metabolic syndrome.
Hypertensive disorders[a]	Neonatal risks are directly related to the onset, duration, and degree of the mother's hypertension. Neonatal complications include intrauterine growth restriction, prematurity, abruptio placentae, stillbirth, and respiratory distress.

Renal disease	Prematurity, intrauterine growth restriction
Rh or other isoimmunization	Anemia, jaundice, hydrops fetalis
Seizure disorder requiring anticonvulsant therapy	Increased risk for congenital anomalies associated with some anticonvulsant drugs. Seizure disorder may independently increase the risk for numerous newborn morbidities including stillbirth, preterm birth, placental abruption, and preeclampsia.
Sexually transmitted infections	Perinatal transmission
Substance abuse	Neonatal abstinence syndrome
Prenatal Conditions	
Assisted vaginal delivery	Cephalohematoma, bruising, subgaleal hemorrhage
Gestational diabetes[a]	Neonatal risks are directly related to the onset, duration, and degree of glucose dysregulation in the mother. Risks include congenital anomalies; miscarriage or stillbirth; perinatal asphyxia; intrauterine growth restriction; large for gestational age; polyhydramnios; birth injury, especially brachial plexus injury following shoulder dystocia; polycythemia; hyperbilirubinemia; hypoglycemia; and respiratory distress. Long-term: The infant has an increased risk of diabetes, obesity, and metabolic syndrome.
Infection	Perinatal transmission
Oligohydramnios	Fetal growth restriction, amniotic band defects, dehydration syndromes, neonatal kidney/bladder abnormalities
Polyhydramnios	Neonatal kidney problems, tracheoesophageal fistula, other congenital anomalies
Size–dates discrepancy	Fetal growth restriction, macrosomia, birth trauma
Perinatal Events	
Abnormal presentation or position of fetus	Birth trauma
Acute fetal bradycardia (e.g., placental abruption, uterine rupture, prolapsed cord)	Intrauterine fetal demise, neonatal encephalopathy, neonatal hypovolemia, metabolic acidemia at birth (base excess [pH > 12]), cerebral palsy[b]
Cesarean birth	Fetal laceration, respiratory distress
Fetal intolerance of labor	Metabolic acidemia at birth (base excess [pH > 12]), neonatal encephalopathy
Intra-amniotic infection or maternal fever in labor	Neonatal sepsis
Meconium-stained fluid	Meconium aspiration pneumonia
Preterm birth	Neonatal risks are related to the degree of prematurity. Selected problems include both short-term and long-term morbidities such as neonatal intensive care unit admission, respiratory distress syndrome, and cerebral palsy.
Prolonged rupture	Neonatal sepsis

of membranes, term or preterm	
Postdates birth (> 42 weeks' gestation)	Postmaturity syndrome, meconium aspiration syndrome, neonatal intensive care unit admission
Operative vaginal birth: vacuum or forceps	Vacuum: Cephalohematoma, intracranial hemorrhage, subgaleal hemorrhage, retinal hemorrhage, shoulder dystocia
	Forceps: Skull fracture, facial nerve palsy, intracranial hemorrhage, subgaleal hemorrhage
Shoulder dystocia	Erb's palsy, fractured clavicle

Neonatal Factors

Valuable data from the immediate neonatal period include the Apgar scores at 1 and 5 minutes, umbilical cord artery pH and base excess values if obtained, and the duration of any resuscitation efforts performed immediately after the birth. Information that indicates an increased risk for perinatal asphyxia is important, as is evidence suggesting an increased risk for hypothermia/hyperthermia and hypoglycemia.

Newborn behaviors (noted in the health record) indicative of a healthy newborn transition include sucking, the ability to feed, alertness, voiding, and passage of meconium. Behaviors of concern include jitteriness, lethargy, poor or absent sucking, and unusual cry.

The results of laboratory tests performed in the birth room should be noted in the newborn history. Typically, these tests include peripheral glucose samples, hematocrit, and umbilical cord blood gases. The anatomic site from which blood samples were obtained should be noted, as this site will be assessed during the physical examination.

Gestational Age Assessment

Key components of both the gestational age assessment and the physical examination can be achieved by observation alone. Observation is particularly powerful and useful in newborn assessment. Judicious use of observation minimizes handling of the newborn and subsequent newborn agitation. If the newborn is ill, minimal touching and handling will allow the infant to conserve oxygen and glucose.

Scales for Gestational Age Assessment

Standardized forms of gestational assessment achieved a wide level of use after the publication of research by Dubowitz in 1970, which described a detailed examination of gestational age–related newborn attributes.[14] The Dubowitz examination combined assessment of both physical and neurologic characteristics.

In 1979, Ballard published a simplified method for assessing gestational age based on physical examination and neuromuscular assessment.[15] The Ballard Scale, based on the work of Dubowitz and others, involves less handling of ill newborns and relies less on a quiet, rested newborn for accurate results. This scale was revised in 1991 in an attempt to more accurately assess extremely premature newborns and provide more accuracy for term newborns.[16] It is now known as the New Ballard Scale, and has become the universal scale used to assess the gestational age of the newborn.

The New Ballard Scale (**Figure 37-1**) is accurate within a range of 2 weeks and can determine the gestational age of newborns as early as 20 weeks. This test is standardized and best performed by a trained examiner. Older studies have found that prior knowledge of the obstetric assessment of gestational age does not bias the examiner performing the New Ballard Scale.[17] Other studies have validated the New Ballard Scale as a reliable clinical tool until day 7 in the life of the moderately preterm infant, with neurologic signs being more reliable than strictly physical ones.[18] The New Ballard Scale does have some limitations. Most notably, it may overestimate gestational age compared with ultrasound or last menstrual period among infants with lower weight or gestational age, and among those newborns whose mothers received prenatal corticosteroid therapy.[19]

NEUROMUSCULAR MATURITY

	−1	0	1	2	3	4	5
Posture							
Square window (wrist)	> 90°	90°	60°	45°	30°	0°	
Arm recoil		180°	140°–180°	110°–140°	90°–110°	< 90°	
Popliteal angle	180°	160°	140°	120°	100°	90°	< 90°
Scarf sign							
Heel to ear							

PHYSICAL MATURITY

Skin	Sticky, friable, transparent	Gelatinous, red, translucent	Smooth, pink, visible veins	Superficial peeling and/or rash, few veins	Cracking, pale areas, rare veins	Parchment, deep cracking, no vessels	Leathery, cracked, wrinkled
Lanugo	None	Sparse	Abundant	Thinning	Bald areas	Mostly bald	
Plantar surface	Heel-toe 40–50 mm: −1 < 40 mm: −2	> 50 mm, no crease	Faint red marks	Anterior transverse crease only	Creases anterior two-thirds	Creases over entire sole	
Breast	Imperceptible	Barely perceptible	Flat areola, no bud	Stippled areola, 1–2 mm bud	Raised areola, 3–4 mm bud	Full areola, 5–10 mm bud	
Eye/Ear	Lids fused loosely: −1 tightly: −2	Lids open; pinna flat, stays folded	Slightly curved pinna, soft, slow recoil	Well-curved pinna, soft but ready recoil	Pinna formed and firm, instant recoil	Thick cartilage, ear stiff	
Genitals (male)	Scrotum flat, smooth	Scrotum empty, faint rugae	Testes in upper canal, rare rugae	Testes descending, few rugae	Testes down, good rugae	Testes pendulous, deep rugae	
Genitals (female)	Clitoris prominent, labia flat	Clitoris prominent, labia minora small	Clitoris prominent, labia minora enlarged	Labia majora and minora equally prominent	Labia majora large, labia minora small	Labia majora cover clitoris and labia minora	

MATURITY RATING

Score	Weeks
−10	20
−5	22
0	24
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

Figure 37-1 The New Ballard Scale (NBS).

Reproduced with permission from Ballard J, Khoury J, Wedig K, Wang L, Eilers-Walsman BL, Lipp R. New Ballard Scale, expanded to include extremely premature infants. *J Pediatr*. 1991;119:417.[16]

The New Ballard Scale takes approximately 2 or 3 minutes to perform, and its results should be recorded directly on the standardized form. It is best to record findings as the assessment is made, rather than relying on memory. After scores for each category of physical and neuromuscular maturity have been assigned, they are summed and the aggregate score is plotted to obtain gestational age.

The proper procedure for both the neuromuscular evaluation and the assessment of physical maturity is included in Appendix 37A. Accurate assessment using the New Ballard Scale

requires practice, and new examiners should have their examinations validated by a preceptor for several gestational age assessments with infants of different gestational ages.

Gestational Age Categories

When the physical maturity score and the neuromuscular maturity score on the New Ballard Scale form are added, the sum yields a gestational age determination. The newborn will then be classified into one of the following categories:[20-24]

1. *Preterm*: less than 37 *completed weeks' gestation*

Extremely preterm: less than 27 6/7 completed weeks' gestation

Very preterm: 28 to 31 6/7 completed weeks' gestation

Moderate preterm: 32 to 33 6/7 completed weeks' gestation

Late preterm: 34 0/7 to 36 6/7 completed weeks' gestation

2. *Term*: 37 to 42 completed weeks' gestation

Early term: 37 0/7 through 38 6/7 completed weeks' gestation

Full term: 39 0/7 through 40 6/7 completed weeks' gestation

Late term: 41 0/7 through 41 6/7 completed weeks' gestation

3. *Post-term*: more than 42 completed weeks' gestation

Small, Appropriate, and Large for Gestational Age

It is possible to estimate newborn health risks solely based on gestational age. By comparison, a more sophisticated relationship exists between gestational age and birth weight.[23] Within each of the categories of gestational age, there will be some newborns who are larger and some who are smaller. The extremes of accelerated and diminished growth are associated with certain newborn problems and predispositions.

At any gestational age, birth weights vary with altitude, race, country of origin, and socioeconomic class.[25] Thus, there is no international standard for normal birth weights for specific gestational ages.

The older charts that graph weight in relation to gestational age have limited utility because they determine gestational age based solely on the mother's menstrual history. More recent research has studied birth weight in comparison to a gestational age assigned by Ballard scores and ultrasound. Assessing birth weight in relation to gestational age will characterize the newborn as being in one of three categories:

1. Small for gestational age (SGA)
2. Appropriate for gestational age (AGA)
3. Large for gestational age (LGA)

After accurately categorizing the newborn into a gestational age and size-for-gestational age category, a plan can be developed to assess and manage potential problems that are associated with each of these categories.

Midwives generally care for term infants but may be involved in collaborative care of late preterm newborns (34 to 36 6/7 weeks' gestation) and early term newborns (37 to 38 6/7 weeks' gestation). Infants born in both of these gestational age categories are at higher risk for multiple respiratory conditions, problems with feeding, thermoregulation issues, jaundice, and death compared to infants born at term.[26,27]

Physical Examination of the Newborn

The physical examination of the newborn is designed to screen for physical variations, malformations, and the overall health of the newborn. There are many differences between the components of an adult physical examination and that of a newborn.

During examination of the newborn, the four basic techniques of physical examination—inspection, palpation, auscultation, and percussion—are used. The complete examination involves three types of evaluation: (1) anthropomorphic measurements, (2) evaluation of organ systems, and (3) neurologic evaluation.

When documenting findings, note any variations present, and avoid use of the nondescript phrase "within normal." Many minor newborn physical and behavioral variations fall within the normal range, so the specific terms and descriptions for newborn characteristics should be used where appropriate.

Approach to the Examination

Most textbooks and physical examination records assume an examination that proceeds from head to toe. This approach is not necessary, however, for a newborn. For example, if the newborn is quiet or sleeping, many examiners start the hands-on examination with auscultation of heart and bowel sounds and palpation of the femoral pulses. In an awake, alert newborn, the examiner may check the red reflex first.[28] A crying newborn presents the opportunity for inspection of the mouth and throat. Excessive handling can over-stimulate the newborn. The newborn may be calmed during an examination by being given a non-latex gloved finger or pacifier to suck on if the mother gives permission.

Anthropomorphic Measurements

Anthropomorphic measurements include the infant's length, as well as the chest and head circumferences. The newborn body has a unique appearance. Normally, the head circumference is larger than the chest circumference, the abdomen is protuberant, and the tone is flexed (**Figure 37-2**). Measurements should be performed using a standardized technique. The newborn length is most accurately assessed if the head of the newborn is flush against a firm surface. A mark often is made on the table paper to designate the top of the neonate's head. The newborn's legs are then extended and a mark made on the table paper where the heels rest when the legs are completely extended. After the newborn is moved, the distance between the mark from the top of the head and the mark placed at the heel is measured and documented in centimeters.



Figure 37-2 Normal newborn body proportions.

The newborn head circumference is measured around the head at the level of the occiput, parietal bosses, and just above the eyebrows. This measurement may decrease in the first week of life as caput succedaneum, cephalohematoma, or soft facial tissue swelling from birth recedes. Chest circumference is measured under the arms and across the nipple line.

The newborn's weight should be assessed on a scale with a drape between the newborn and the metal or plastic. This covering prevents conductive heat loss and minimizes infection from cross-contamination. The scale should be calibrated to account for the weight of the drape. **Table 37-3** lists mean birth weights, lengths, and head circumferences of term newborns (37–42 weeks) born in North America.[29] Newborn measurements vary by gestational age, sex, and parental ethnicity, with newborns having Asian heritage being the smallest at birth. Using correct measurement standards can prevent unnecessary testing for newborns incorrectly categorized as small for gestational age.

Table 37-3 Mean (and Standard Deviation) for Birth Weight, Length, and Head Circumference by Sex, Gestational Age at Birth, and Ethnicity

Gestational Age (weeks)	European Descent		Chinese Descent		South Asian Descent	
	Weight (g) Mean (SD)	Length (cm) Mean (SD)	Weight (g) Mean (SD)	Length (cm) Mean (SD)	Weight (g) Mean (SD)	Length (cm) Mean (SD)
Male						
37	3336.4 (434.3)	49.4 (2.0)	3119.0 (361.9)	48.3 (1.6)	3060.0 (346.1)	48.7 (3.9)
38	3359.4 (416.3)	49.4 (1.7)	3209.5 (356.5)	48.4 (2.0)	3201.8 (397.8)	50.2 (2.2)
39	3586.0 (460.3)	50.5 (1.9)	3359.0 (390.4)	49.4 (2.0)	3361.2 (383.5)	50.2 (1.7)
40	3687.6 (410.5)	50.8 (1.7)	3500.4 (343.5)	50.1 (1.6)	3452.5 (399.1)	50.4 (2.5)
41	3877.1 (384.4)	51.4 (2.2)	3724.1 (522.3)	50.9 (2.9)	3674.7 (363.1)	51.4 (1.5)
Female						
37	3050.7 (414.1)	47.4 (2.5)	3048.4 (402.9)	47.6 (1.7)	2956.8 (436.3)	48.2 (2.2)
38	3993.5 (423.3)	48.6 (1.8)	3190.2 (377.1)	48.2 (1.6)	3118.4 (371.4)	48.8 (1.8)
39	3455.8 (434.0)	49.4 (1.7)	3271.0 (424.1)	48.7 (1.7)	3311.7 (458.5)	49.5 (1.9)
40	3639.5 (458.2)	50.2 (1.8)	3373.3 (362.3)	49.2 (2.2)	3376.1 (389.4)	50.0 (2.6)
41	3696.8 (448.0)	50.5 (1.6)	3451.0 (344.5)	49.4 (2.7)	3411.4 (401.6)	50.6 (1.6)

Modified with permission from Janssen PA, Thiessen P, Klein MC, Whitfield MF, Macnab YC, Cullis-Kuhl SC. Standards for the measurement of birth weight, length and head circumference at term in neonates of European, Chinese and South Asian ancestry. *Open Med*. 2007;1(2):e74-e88.[29] Table 2.29. Available at: http://www.openmedicine.ca/article/view/47/45. Reprinted by permission.

Assessment for Birth Injuries

Birth injuries may occur during a long, protracted labor or a difficult birth. For example, they can be associated with situations in which the fetus is large or the fetal presentation/position is abnormal. Physical examination findings related to birth trauma include small scalp abrasions, bruises that correspond to forceps placement along the side of the face, brachial plexus injury to the arm, and fractures to bones, most often clavicle or extremities.

The skull bones are not fused in an infant and therefore they are somewhat mobile and able to conform to pressures from the maternal pelvic bones during the passage through the vaginal canal. A newborn skull that is not exactly round is a normal finding. Parents can be reassured that the skull will assume a normal newborn shape within a few days. Possible physical examination findings of the newborn head are depicted in **Figure 37-3**. There is often a soft tissue swelling (edematous collection of serosanguineous and subcutaneous fluid) called *caput succedaneum* that is visible on the area of the fetal head, which was the presenting part. Caput succedaneum has poorly defined margins, may cross suture lines, and is not markedly tense. In contrast, a *cephalohematoma* is a collection of blood under the periosteum of one of the cranial bones, usually the parietal bone. Unlike caput succedaneum or general edema, the blood does not cross the suture lines of the newborn skull because the bleeding is under the periosteum. Some cephalohematomas occur with linear skull fractures, most of which heal well. A *subgaleal hemorrhage* is an accumulation of blood below the scalp but above the periosteum. It is a rare but life-threatening complication because this space is large and the newborn can lose a great deal of blood very quickly. Signs include a diffuse swelling in the head that shifts independent of movement. In addition, the newborn will display symptoms of shock and hypovolemia.

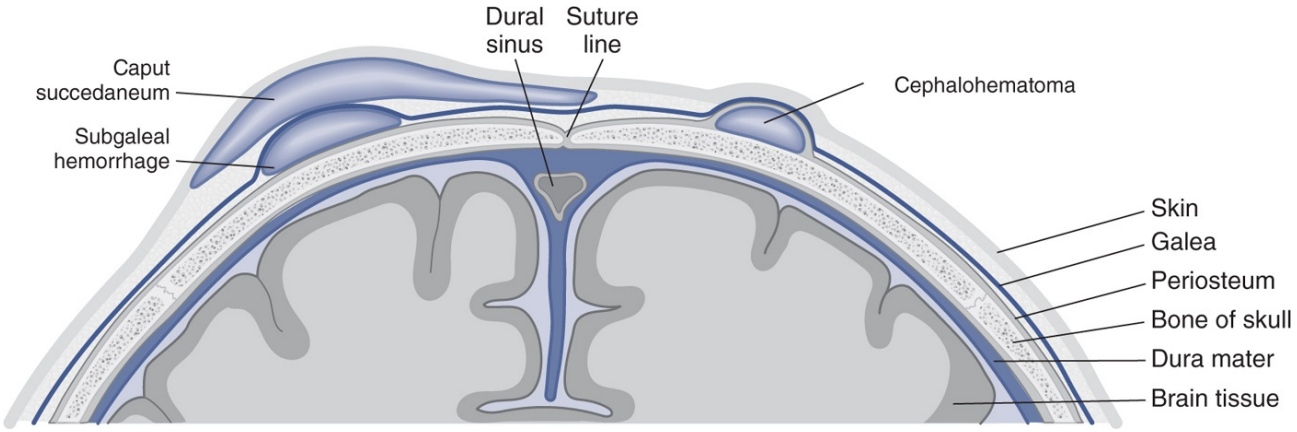


Figure 37-3 Anatomy of caput succedaneum, cephalohematoma, and subgaleal hemorrhage.

The signs of facial palsy—a temporary condition—include asymmetry of the face. The newborn with a brachial plexus injury may be in pain. The manifestations of the injury will depend on the nerve root that was injured and the degree of the injury. Nerve roots involved can include the cervical roots C5 and C6 (Erb-Duchenne paralysis), roots C8 and T1 (Klumpke's paralysis), or both.[30] The physical signs of Erb-Duchenne paralysis include a generalized loss of movement in the affected arm with an adduction of the lower part of the arm. This leads to the characteristic "waiter's tip" sign, involving an internal rotation of the lower portion of the arm with the finger and wrist flexed.[30] The grasp reflex is intact, but the Moro reflex is weak on the affected side. In Klumpke's paralysis, the grasp reflex is absent and the infant's hand is kept in a claw-like posture.[30]

Pressure and manipulation during the birth process may cause fractures of bones, although occasionally intrauterine fractures may occur. The bones most frequently affected include the clavicles and the extremities. Signs of bone fracture include swelling, skin discoloration, lack of movement, abnormal positioning, and pain with movement.[31] Crepitus occasionally can be elicited on palpation. The Moro reflex will be asymmetric.

Assessment for Birth Defects

Variations in physical appearance may be due to inconsequential human variation or familial traits. However, approximately 3% to 4% of live births involve some congenital defect—most minor, but some serious. Many genetic, chromosomal, multifactorial disorders are initially suspected or even diagnosed based on the newborn's physical appearance.

Many parents believe that normal prenatal tests provide assurance that their fetus is normal. Unfortunately, all prenatal tests have limitations, and it is important to explain to new parents that most prenatal tests are screening tools meant to identify an increased risk for specific common disorders rather than being either diagnostic or able to detect all possible birth defects. The newborn examiner must inform parents about any potential problems detected during the examination and refer to a specialist for more extensive evaluation.

Most minor malformations (**Table 37-4**), when found in isolation, are simple physical variants. However, the presence of three or more minor malformations is suggestive of a major

underlying malformation, and requires further evaluation.[30] For example, epicanthal folds may be normal by themselves, especially for infants of Asiatic descent. In combination with a transverse palmar crease and low-set ears, however, genetic evaluation for a possible trisomy is warranted.

Table 37-4 Common Minor Malformations Noted on a Newborn Examination

Large fontanel
Epicanthal folds
Hair whorls
Widow's peak
Low posterior hair line
Preauricular tags and pits
Minor ear anomalies: protruding, rotated, low-set
Darwinian tubercle (thickening of ear on helix at junction of upper and lower thirds)
Digital anomalies: clinodactyly (curved finger); camptodactyly (bent finger); syndactyly (webbed finger)
Transverse palmar crease
Shawl scrotum (scrotum surrounds penis)
Redundant umbilicus
Widespread nipples
Supernumerary nipples

With the routine use of prenatal ultrasound, many physical malformations of the fetus may already have been detected during the prenatal period. However, the accuracy of ultrasonography can be compromised by numerous factors (e.g., technician experience level, fetal position during sonogram), which may lead to many subtle malformations going undetected until the infant's birth. **Table 37-5** lists visual clues that may be noted during physical examination and the serious conditions that they may indicate.[30] Early evaluation and intervention can help prevent serious sequelae of malformations such as infection, intestinal perforation, spinal cord injury, or intellectual disability.

Table 37-5 Visual Clues That Suggest Birth Defects and Genetic Conditions

Diagnosis	Possible Visual Clues
Autosomal Dominant (Mendelian Inheritance)	
Neurofibromatosis I	Café-au-lait spots
Tuberous sclerosis	Ash leaf spot: hypopigmented areas of skin
Myotonic dystrophy	Myopathic facies
Waardenburg syndrome	Facial abnormalities, with diminished coloration of hair, skin, and iris
Van der Woude syndrome	Cleft palate, cleft lip, and depression of lower lip
Holt–Oram syndrome	"Heart-hand syndrome": heart and upper limb abnormalities
Autosomal Recessive (Mendelian Inheritance)	
Tay–Sachs disease	Cherry red spot in macula of eye
Galactosemia	Cataracts and neonatal jaundice
Cystic fibrosis	Meconium ileus, rectal prolapse
Congenital adrenal hyperplasia	Ambiguous genitalia
Mucopolysaccharidoses	Corneal clouding, joint contracture
Meckel–Gruber syndrome	Polydactyly, encephalocele
Rhizomelia chondrodysplasia	Short proximal limbs, cataracts
X-Linked Recessive (Mendelian Inheritance)	
Fragile X syndrome	Large testes
Duchenne muscular dystrophy	Large calf muscle
Menke kinky hair syndrome	Steel-wool-like hair
Usher syndrome	Retinitis pigmentosa
Lesch-Nyhan syndrome	Gravel urine (sediment and calcifications in urine)
Mitochondrial (Non-Mendelian Inheritance)	
Kearns–Sayre syndrome	Muscle weakness, ophthalmoplegia (paralysis or weakness of eye muscles)
Leber's hereditary optic neuropathy	Muscle weakness, ophthalmoplegia
MELAS syndrome	Muscle weakness, ophthalmoplegia
MERRF syndrome	Muscle weakness, ophthalmoplegia
Uniparental Disomy (Non-Mendelian Inheritance)	
Prader–Willi syndrome	Obesity, small hands and feet
Gonadal Mosaicism (Non-Mendelian Inheritance)	
Osteogenesis imperfecta	Blue sclera

Diagnosis	Possible Visual Clues	Diagnosis	Possible Visual Clues
Teratogen(s)		**Chromosomal Abnormalities**	
Alcohol	Microcephaly, short palpebral fissures	Trisomy 21	Hypotonia, single palmar crease, prominent tongue
Nicotine (smoking)	Small for gestational age	Trisomy 18	Finger and joint contracture, webbed neck
Dilantin (Phenytoin)	Nail hypoplasia	Trisomy 13	Polydactyly, cleft palate, scalp defects
Carbamazepine (Tegretol)	Neural tube defects	Turner syndrome (45 XO)	Short stature, webbed neck
Warfarin (Coumadin)	Flat nasal bridge	Klinefelter syndrome (47XXY)	Small testes, gynecomastia
Isotretinoin (Accutane)	Facial and limb anomalies	Cri-du-chat syndrome (chromosome 5p deletion syndrome)	Natal mewing
Multifactorial		**Sporadic Multiple Pattern Syndrome**	
Clubfoot	Twisted foot	Cornelia de Lange syndrome	Synophrys (single eyebrow meeting in middle of face), phocomelia (underdeveloped limbs)
Cleft lip/palate	Nonjunction of lip or palate or both	Rubinstein–Taby syndrome	Large thumbs and great toes
Neural tube defects	Spina bifida, anencephaly, encephalocele	Williams syndrome	Elfin facies
Dislocated hip	Legs of different length, hip click on examination but may be asymptomatic	Sturge–Weber syndrome	Nevus flammeus
Congenital heart defect	Heart murmur, acrocyanosis, circumoral cyanosis	VACTERL association or VATER syndrome[a]	Tracheoesophageal fistula, imperforate anus, limb defects
Hypospadias	Urethral opening is on underside of penis		

Abbreviations: MELAS, mitochondrial encephalopathy, lactic acidosis, and stroke-like seizures; MERRF, myoclonic epilepsy with ragged red fibers.

[a] VACTERL and VATER refer to the types of defects that may be apparent at birth: vertebral, anus, cardiac, tracheoesophageal, renal, and limb.

Modified with permission from Wardinsky T. Visual clues to diagnosis of birth defects and genetic disease. *J Pediatr Health Care.* 1994;8(2):63-73.[30]

Newborns with ambiguous genitalia present a challenge. These newborns have external genital characteristics of both males and females. If the examiner has doubts about the genitalia, an immediate consultation with a neonatal team is indicated. In some instances, ambiguous genitalia are associated with congenital adrenal hyperplasia—a condition that will cause life-threatening dehydration shortly after birth. The team will measure the clitoris or phallus. This measurement will be used, along with other physical characteristics of the genitalia, to describe the virilization as in categorized in Prader staging.[32] The findings and arrangement for follow-up care will be discussed with the family. In some cases, these families will be encouraged to avoid assigning a sex to the newborn.

Complete assessment of an infant with physical variations should be performed by a team that includes specialists such as a neonatologist, a geneticist, an endocrinologist, and the nurse who is caring for the child. Some common abnormalities may be confirmed with rapid testing within 48 hours, but unfortunately it may take weeks before a comprehensive genetic karyotype is complete. Extensive additional testing may be necessary to evaluate the type and extent of the malformation. When a physical variation of concern is detected, the midwife's role is to obtain consultation and support the family.

The Neurologic Examination

The procedure of assessing reflexes, cranial nerves, and special senses is integrated into the overall physical examination outlined in Appendix 37A. The neurologic examination is an indicator of the integrity of the nervous system. Both diminished (hypo) and accentuated (hyper) responses are cause for concern. Diminished response can result from a congenital absence of a nerve, damage to sensory or motor pathways, or infection. Diminished or accentuated response to stimulation can also reflect a central neurologic deficit.

During the neurologic examination, assessments are made of the newborn's senses, including sight, hearing, and smell. Poor or absent response to oral, auditory, or olfactory stimulation may indicate nerve damage.

An absent or diminished response to an elicited reflex may be an indication of a variety of conditions.[33,34] Sometimes the reflex is partial or diminished because of newborn depression secondary to medications or infection. In other cases, the response may not be optimal because the sensory pathway conducting the stimulation has been damaged, reflecting a spinal or central nervous system lesion. Sometimes a reflex cannot be elicited because of temporary motor nerve damage that prevents the muscles in the affected area from responding to stimulation, as with a weak palmar grasp after a breech delivery or facial nerve paralysis after a forceps delivery (**Figure 37-4**). Sometimes damage is permanent, as in some brachial plexus injuries and some spinal cord defects. The following reflexes are elicited as part of the physical examination and are described in detail in Appendix 37A:

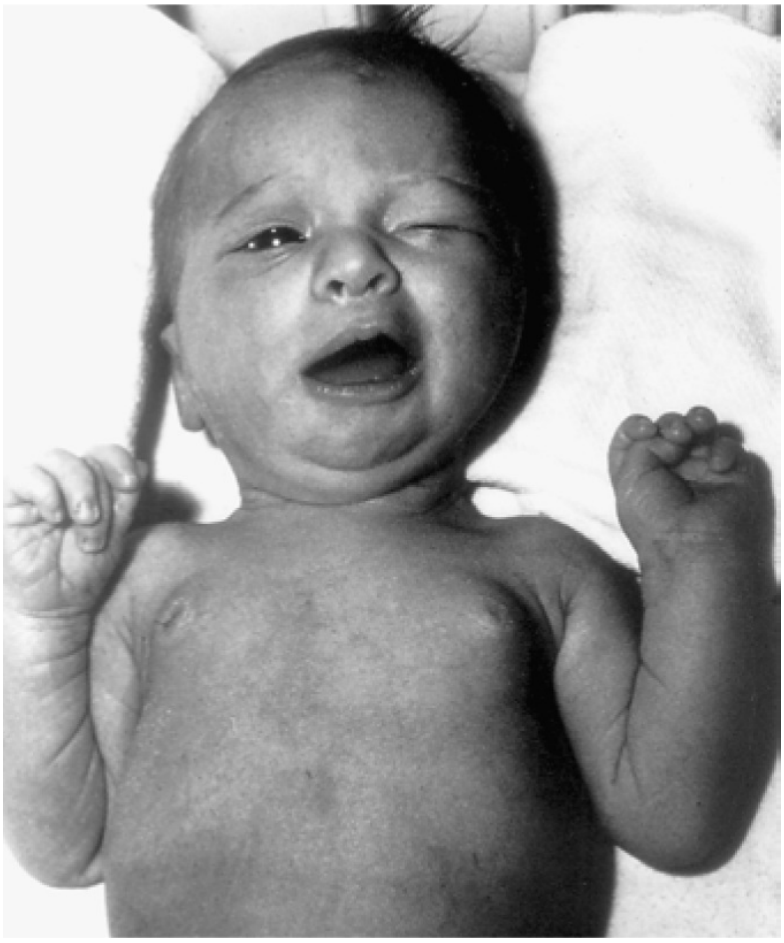


Figure 37-4 Facial nerve paralysis.

1. *Eyes*: pupillary reflex, red reflex, doll's eye reflex, blink reflex
2. *Upper extremities*: palmar grasp reflex
3. *Lower extremities*: patellar reflex, plantar reflex, Babinski reflex (**Figures 37-5** and **37-6**)



Figure 37-5 Plantar reflex.



Figure 37-6 Babinski reflex.

4. *Torso*: anal wink, tonic neck reflex

Absent, markedly diminished, or accentuated reflexes and asymmetrical reflexes should be noted on the physical examination form, and consultation with a pediatric provider about further testing and follow-up initiated.

One of the most commonly used evaluations of the neurologic status of the newborn is the Moro reflex, also known as the embracing reflex. As is true for the other primitive reflexes, the

Moro reflex is mediated by the brain stem and becomes apparent at approximately 25 to 26 weeks' gestational age.[34] Once thought to reflect an infant's effort to cling to or "embrace" its caregiver, it is now more commonly seen as an involuntary motor response meant to protect the infant from sudden changes in body displacements.[34-36] In normal infants, the response is symmetrical and disappears by 3 to 4 months. The Moro reflex consists predominantly of abduction and extension of the arms with hands open, and the thumb and index finger semiflexed to form a "C". Leg movements may occur, but they are not as uniform as the arm movements. With return of the arms toward the body, the infant either relaxes or cries. In comparison, a "startle pattern" (or reaction) is primarily a flexion rather than an extension response to sudden stimulation, and becomes more pronounced and regular as the infant matures, rather than diminishing.[33,34] Acceptable ways to elicit a Moro reflex that avoid eliciting a "startle pattern" include the following:

1. While the child is in a semi-sitting position, allow the infant to fall backward onto the examiner's open hand from an angle of 30 degrees.
2. While holding the infant in a horizontal position with both hands, quickly lower the infant approximately 10 to 20 cm, and come to sudden halt.[33]

As one of the primary primitive reflexes, any deviations found in the Moro reflex necessitate a pediatric consultation at a minimum and possibly a full neurologic work-up, especially if deviations in other reflex tests are also found. Sample deviations of the Moro reflex and their possible etiologies include the following:

- Absence of the reflex may indicate intracranial lesions.
- Asymmetrical response may indicate birth injury involving the brachial plexus, clavicle, or humerus.
- Abnormal persistence of embrace gesture may indicate hypertonicity.
- Persistence of the entire Moro reflex after 4 months may indicate delay in neurologic maturation.

Becoming Confident at Newborn Physical Examination

Many intricacies and nuances are involved in conducting and assessing a complete newborn examination. Recognition of normal as well as possible deviations is an essential skill for any clinician involved in the care of infants. While many academic sources are available for reference and review,[37,38] they cannot substitute for multiple clinical experiences involving the observation and examination of many newborns.

Resources

Organization	Description	Webpage
The New Ballard Score	This site explains gestational age estimation using the Ballard Score with drawings, photos, and video clips.	http://www.ballardscore.com
Global Health Media	Training video on examination of the newborn. These training videos have been provided by the Global Health Media Project and are based on standards of care described in *Care of the Newborn Reference Manual*, Save the Children, 2004; *Managing Newborn Problems*, World Health Organization, 2003; and *Integrated Management of Childhood Illnesses Chart Booklet*, World Health Organization, 2011.	https://youtu.be/m6MBM0pylpA?list=PLXeu9kWMOoFLMXFrdo67Xx06HiCa3Q6Vv

References

1. Lipman TH, Euler D, Markowitz GR, Ratcliffe SJ. Evaluation of linear measurement and growth plotting in an in-patient pediatric setting. *J Pediatr Nurs*. 2009;24(4):323-329.
2. Holbrook BD. The effects of nicotine on the human fetal development. *Birth Defects Res C Embryo Today*. 2016;108(2):181-192.
3. Zhang K, Want X. Maternal smoking and increased risk of sudden infant death syndrome: a meta-analysis. *Leg Med*. 2013;15(3):115-121.
4. Farrar D, Simmonds M, Bryant M, et al. Hyperglycaemia and risk of adverse perinatal outcomes: systematic review and meta-analysis. *BMJ*. 2016;354:i4694.
5. Friedmann I, Dahdouh EM, Kugler P, Mimran G, Balayla J. Maternal and obstetrical predictors of sudden infant death syndrome (SIDS). *J Matern Fetal Neonatal Med*. 2017;30(19): 2315-2323.
6. Manuck TA, Rice MM, Bailit JL, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Developme Maternal–Fetal Medicine Units Network. Preterm neonatal morbidity and mortality by gestational age: a contemporary cohort. *Am J Obstet Gynecol*. 2016;215(1):103.e1-103.e14.
7. Sjaus A, McKeen DM, George RB. Hypertensive disorders of pregnancy. *Can J Anaesth*. 2016;63(9):1075-1097.
8. Tucker J, McGuire W. Epidemiology of preterm birth. *BMJ*. 2004;329(7467):675-678.
9. Mitanchez D, Yzydorczyk C, Siddeek B, Boubred F, Benahmed M, Simeoni U. The offspring of the diabetic mother: short and long-term implications. *Best Pract Res Clin Obstet Gynaecol*. 2015;29:256-259.
10. Fahey JO. The recognition and management of intrapartum fetal heart rate emergencies: beyond definitions and classifications. *J Midwifery Womens Health*. 2014;59(6):616-623.
11. American College of Obstetricians and Gynecologists. Practice Bulletin No. 146: management of late-term and postterm pregnancies. *Obstet Gynecol*. 201;124(2 pt 1):390-396.
12. Razaz N, Tomson T, Wikström AK, Cnattingius S. Association between pregnancy and perinatal outcomes among women with epilepsy. *JAMA Neurol*. 2017;74(8):983-991.
13. Caughey AB, Sandberg PL, Zlatnik MG, et al. Forceps compared with vacuum: rates of neonatal and maternal morbidity. *Obstet Gynecol*, 2005;106(5 pt 1):908-912.
14. Dubowitz L, Dubowitz V, Goldberg C. Clinical assessment of gestational age in the newborn infant. *Pediatrics*. 1970;77:1-10.
15. Ballard J, Novak K, Driver M. A simplified score for assessment of fetal maturation of newly born infants. *J Pediatr*. 1979;95(5 pt 1):769-774.
16. Ballard J, Khoury J, Wedig K, Wang L, Eilers-Walsman BL, Lipp R. New Ballard Scale, expanded to include extremely premature infants. *J Pediatr*. 1991;119(3):417-423.
17. Smith LN, Dayal VH, Monga M. Prior knowledge of obstetrical gestational age and possible bias of Ballard score. *Obstet Gynecol*. 1999;93(5):712-714.
18. Sasidharan K, Dutta S, Narang A. Validity of New Ballard Score until 7th day of postnatal life in moderately preterm neonates. *Arch Dis Child Fetal Neonatal Ed*. 2009;94:39-44.
19. Donovan EF, Tyson JE, Ehrenkranz RA, Verter J, et al.; National Institute of Child Health and Human Development Neonatal Research Network. Inaccuracy of Ballard scores before 28 weeks' gestation. *J Pediatr*. 1999;135(2 pt 1):147-152.
20. American College of Obstetricians and Gynecologists. reVITALize obstetric data definitions. Available at: https://www.acog.org/About-ACOG/ACOG-Departments/Patient-Safety-and-Quality-Improvement/reVITALize-Obstetric-Data-Definitions. Accessed September 28, 2017.
21. Spong CY. Defining "term" pregnancy: recommendations from the Defining "Term" Pregnancy Workgroup. *JAMA*. 2013;309(23):2445-2446.
22. Raju TNK. Moderately preterm, late preterm and early term infants: research needs. *Clin Perinatol*. 2013;40(4):10.1016.
23. Engle WA. A recommendation for the definition of "late preterm" (new term) and the birth weight-gestational age classification system. *Semin Perinatol*. 2006;30(1):2-7.
24. American College of Obstetricians and Gynecologists. Committee Opinion No. 579: definition of term pregnancy. Available at: http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/Definition-of-Term-Pregnancy. Accessed September 28, 2017. [Reaffirmed 2015].
25. Soria R, Julian CG, Vargas E, Moore LG, Giussani DA. Graduated effects of high-altitude hypoxia and highland ancestry on birth size. *Pediatr Res*. 2013;74(6):633-638.
26. Celik IH, Demirel G, Canpolat FE, Dilmen U. A common problem for neonatal intensive care units: late preterm infants, a prospective study with term controls in a large perinatal center. *J Matern Fetal Neonatal Med*. 2013;26(5):459-462.
27. Pulver LS, Guest-Warnick G, Stoddard GJ, Byington CL, Young PC. Weight for gestational age affects the mortality of lat preterm infants. *Pediatrics*. 2009;123:1072-1077.
28. Cagini C, Tosi G, Stracci F, Rinaldi VE, Verrotti A. Red reflex examination in neonates: evaluation of 3 years of screening.

Int Ophthalmol. 2016;7. [Epub ahead of print]. doi:10.10007/s10792-016-0393-2.
29. Janssen PA, Thiessen P, Klein MC, Whitfield MF, Macnab YC, Cullis-Kuhl SC. Standards for the measurement of birth weight, length and head circumference at term in neonates of European, Chinese and South Asian ancestry. *Open Med*. 2007;1(2):e74-e88.
30. Wardinsky T. Visual clues to diagnosis of birth defects and genetic disease. *J Pediatr Health Care*. 1994;8(2):63-73.
31. Doumouchtsis SK, Arulkumaran S. Head injuries after instrumental vaginal deliveries. *Curr Opin Obstet Gynecol*. 2006;18(2):129-134.
32. Murphy C, Allen L, Jamieson MA. Ambiguous genitalia in the newborn: an overview and teaching tool. *J Pediatr Adolesc Gynecol*. 2011;24(5):236-250.
33. Futagi Y, Toribe Y, Suzuki Y. The grasp reflex and Moro reflex in infants: hierarchy of primitive reflex responses. *Int J Pediatr*. 2012;2012:191562. http://dx.doi.org/10.1155/2012/191562
34. Lewis M. A comprehensive newborn examination: Part II. Skin trunk, extremities, neurologic. *Am Fam Physician*. 2014;90(5):297-302.
35. Goldstein K, Landis C, Hunt W, Clarke FM. Moro reflex and startle pattern. *Arch Neurol Psychiatry*. 1938;40(2):322-327
36. Sohn M, Ahn Y, Lee S. Assessment of primitive reflexes in high-risk newborns. *J Clin Med Res*. 2011;3(6): 285-290.
37. Jones K. *Smith's Recognizable Patterns of Human Malformation*. 6th ed. Philadelphia, PA: Elsevier Saunders; 2006.
38. Zitelli BJ, McIntire SC, Nowalk AJ. *Zitelli and Davis' Atlas of Pediatric Physical Diagnosis*. 6th ed. Philadelphia, PA: Elsevier Saunders; 2012.

APPENDIX

37A

Physical Examination of the Newborn

CECILIA M. JEVITT

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Findings	Significance	MOE	Notes
Color			Indicative of overall status of infant, especially cardiopulmonary system
In light-skinned infants: Body and mucous membranes should be pink In dark-skinned infants: Mucous membranes should be pink	No cardiopulmonary compromise	I	
Acrocyanosis of hands and feet during first 24 hours of life	Sluggish peripheral circulation caused by transition to the cool extrauterine life	I	*Definition:* Acrocyanosis—a blue or purple mottled discoloration of the extremities, especially the fingers, toes, or nose
Reddish hue	Adjustment in oxygen levels in extrauterine environment	I	Especially noted immediately after birth
Ecchymosis over presenting part	Pressure over presenting part, causing bruising and trapping of blood in external tissue layers	I	*Definition:* Ecchymosis—the escape of blood into the tissues from ruptured blood vessels marked by a livid black-and-blue or purple spot or area *Technique:* To differentiate between cyanosis and ecchymosis, apply pressure to darkened area; cyanotic area will blanche, ecchymotic area will remain dark
Mongolian spots over buttocks, with possible extension to sacral region	Hyperpigmentation	I	*Normal variation*, especially if found in dark-skinned infants
Jaundice after first 48 hours of age, receding by days 4–5	Physiologic jaundice	I	*Definition:* Physiologic jaundice—transition of blood supply to liver, increased RBC count with decreased lifespan of cells, decreased plasma protein level, and decreased glucuronyl transferase that aids in bilirubin conjugation *Prematurity:* Peak in jaundice may occur later

General Appearance			Indicative of nutritional status, infant maturity, and general well-being
Well-formed and rounded, with presence of subcutaneous tissue No obvious anomalies	Good nutritional status; generally healthy	I	
Vernix	Increases with gestational age	I	*Definition:* Vernix—a pasty covering chiefly of dead cells and sebaceous secretions that protects the skin of the fetus
Lanugo	Decreases with gestational age	I	*Definition:* Lanugo—a dense cottony or downy growth of hair; specifically, the soft downy hair that covers the fetus of some mammals *Technique (NBS):* Check lanugo on the back with a direct light to get a clear view
Posture			
Newborn position: • Fists clenched • Arms adducted, flexed • Hips abducted • Knees flexed • Flexion moves upward to arms • Spinal column straight		I	*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1 *Prematurity:* "Frog position" of legs
Movement: • Spontaneous, symmetrical, may be slightly tremulous • Flexion and extension should be equal bilaterally	Full-term newborn activity	I	*Definition:* Tremulous—jerky motions, unequal movement *Prematurity:* No movement or asymmetrical, irregular, tremulous
Muscle Strength and Tone			
Strength and tone: Strong	May be full term	I	*Prematurity:* Strength and tone weak, hypotonia, or flaccid
Palmar grasp: Strong	Good overall strength; may be full term	I	*Prematurity*: Palmar grasp weak
Alertness and Cry			
Mood: • Ranges from quiet to alert • Consolable when upset	Normal newborn activity	I	*Prematurity:* Not easily aroused, not very alert
Cry: Strong	No increased intracranial pressure	I	
Moro (embracing) reflex: • Symmetrical • Disappears by 2–4 months		I/Pa	*Definition:* Consists predominantly of abduction and extension of the arms with hands open and the thumb and index finger semiflexed to form a "C". Leg movements may occur, but they are not as uniform as the arm movements. With return of the arms toward the body, the infant either relaxes or cries.

Alertness and Cry			
			Technique: Acceptable ways to elicit a Moro reflex include the following: • While the child is in a semi-sitting position, allow the infant to fall backward onto the examiner's open hand from an angle of 30 degrees • While holding the infant in a horizontal position with both hands, quickly lower the infant approximately 10–20 cm and come to sudden halt
Cardiopulmonary System			
Respiratory effort: • Easy, unlabored rhythm • May be irregular, but periods of apnea < 15 seconds Abdominal breathing	No respiratory distress or difficulty	I	
Respiratory rate: 40–60 breaths per minute	Normal rate	I	*Variation on normal:* Tachypnea in cesarean section or in full-term infants may be transient from retention of lung fluid *Prematurity:* Apnea > 15 seconds and accompanied by duskiness, cyanosis, or respiratory rate > 60 breaths per minute
Thorax: Symmetrical excursion	Normal respiratory pattern	I	*Definition:* Excursion—one complete movement of expansion and contraction of the lungs and their membranes (as in breathing)
Anteroposterior diameter: Normal	Normal respiratory pattern	I	
Breath sounds: • Clear • Equal bilaterally, anteriorly, and posteriorly • Occasional rales in first 24 hours	Clear lung fields	A	*Variation on normal:* Occasional rales may be present the first few hours after birth because of residual fetal lung fluid; no color changes or cyanosis should accompany this finding
Heart rate: • 100–160 bpm • Regular rate and rhythm • Without murmurs or only initial slight murmur	Normal cardiac rhythm without significant abnormalities	A	*Variation on normal:* Initial, slight murmur, usually heard at left sternal border or above apical pulse; may persist until the ductus arteriosus closes *Prematurity:* Bradycardia < 100 bpm or tachycardia > 160 bpm; murmur
Cranium/abdomen: No bruit	No arteriovenous malformation	Pa	*Definition:* Bruit—any of several generally abnormal sounds heard on auscultation
Apical pulse: At fourth or fifth intercostal space, mid-clavicular line, left anterior chest	Normal position of cardiac pulse; no shifting; without cardiomegaly		*Variation on normal*: Point of maximum impulse at the fourth intercostal space just right of the mid-clavicular line may be shifted to the right during the first few hours of life
Thrill: None present after first few hours of life	No increased cardiac activity	Pa	*Definition:* Thrill—an abnormal fine tremor or vibration in the respiratory or circulatory systems felt on palpation

Cardiopulmonary System			
Blood pressure: • Average systolic rate ◆ 28–32 weeks: 52 mm Hg ◆ 32–36 weeks: 56 mm Hg ◆ Full term: 63 mm Hg Equal in all 4 extremities	Normal cardiac output; good circulation; possibly no cardiac defect	Pa	
Tympany: Not increased over lung fields	Normal lung field borders	Pe	*Definition:* Tympany—a resonant sound heard in percussion
Skin			
Moist Warm to touch No peeling, wrinkles, edematous or shiny/taut skin	Normal, well hydrated	I	*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1 *Variation on normal* (unless other abnormal findings are also present): Nevus flammeus; meconium staining; petechiae; skin tags *Prematurity:* Gelatinous with visible veins (transparent skin and visible veins disappear with increasing gestational age) *Postmaturity:* Dry, peeling, cracked
Vernix: Present	Increases with gestational age	I	*Definition:* See above *Prematurity:* No vernix
Lanugo: Scant	Full term Decreases with gestational age	I	*Definition:* See above *Prematurity:* Abundant lanugo
Milia	Common in newborns	I	*Definition:* Milia—a small, pearly, firm, non-inflammatory elevation of the skin due to retention of keratin in an oil gland duct blocked by a thin layer of epithelium
Erythema toxicum	Newborn rash over body, usually on days 1–3	I	
Mottling	May be normal reaction to immaturity of organ systems, if infant is of normal temperature, without color changes or bradycardia	I	
Temperature: • Warm • Axillary temperature 35.5–37.0°C	Normal range	P	*Prematurity:* Temperature may be cool (< 35.5°C) or warm (> 37°C)
Head			
Normocephalic in proportion to body (head circumference for average full-term newborn is 32–38 cm)	Normal	I	
Molding: Cranial distortion lasting < 5–7 days	Excessive pressure on cranium during vaginal delivery	I	

Head			
Overriding sutures	Excessive pressure on cranium during vaginal delivery	I	
Caput succedaneum	Edematous region of scalp extending over suture lines, resulting from pressure on the presenting part during vaginal delivery	I	
Head lag: ≥ 10 degrees in full-term infant Able to support head	Head lag: Decreases with maturity	I	*Technique:* Pull newborn up, supporting the arms, from supine to sitting position; grade degree of head lag by position of head in relationship to trunk *Prematurity:* Head lag > 10 degrees; little or no support of head
Hair distribution: Over top of head, with single strands identifiable	Full term	I	*Prematurity:* Fine, fuzzy, may be over entire head
Without masses or soft areas over skull bones	Normal	Pa	*Variation on normal:* Masses or soft areas such as craniotabes over parietal bones may be normal if no abnormality is present *Definition:* Craniotabes—thinning and softening of the infantile skull in spots
Bruit: None	Normal	A	*Definition:* See above
Fontanelles			
Anterior fontanelle: • Open until 12–18 months • Diamond shaped • Approximately 5 × 4 cm • Found along the coronal and sagittal sutures	Normal	I/A	
Posterior fontanelle: • Triangle shaped • Approximately 1 × 1 cm • Found along sagittal and lambdoidal suture lines	Normal May be closed at birth	I/A	
Facies			
Eyes: • On line with ears • Normal distance between eyes (approximately 2.5 cm) • Unworried expression	Normal	I	
Nose: Midline	Normal	I	
Oral Cavity			
Mouth: • Midline on face, symmetrical without drooping or slanting unilaterally with crying or other movement of mouth • Shape and size in proportion with face	Normal	I	

Oral Cavity			
Mucous membranes: Moist, pink	Well hydrated and oxygenated	I	
Chin: Shape and size in proportion with face	Normal	I	
Lips: Completely formed, pink, moist	Normal	I	
Palate: No arching; intact	Normal	I/Pa	
Tongue: • Size in proportion with mouth • Midline	Normal No neurologic dysfunction	I	
Uvula: Midline rises with crying	Normal function of glossopharyngeal and vagus nerves	I	
Gag reflex: Present	Normal neurologic function of glossopharyngeal and vagus nerves	I	*Physiology:* Reflexes generally develop from head to toe during gestation
Sucking reflex: Present and strong	Normal maturity and intact hypoglossal nerve	I	*Technique:* Offer non-latex gloved finger or nipple to test *Prematurity:* May be absent
Rooting reflex: Present	Normal maturity and intact trigeminal nerve	I	*Technique:* When cheek is stroked, infant turns toward stroking *Prematurity:* May be absent
Salivation: Without excess	Normal	I	
Nose			
Position: Midline	Normal	I	
Nares: Bilaterally present	Intact	I	
Nares: Patent	Normal	I	*Technique:* Occlude neonate's nostrils one at a time while holding mouth closed; infant should be able to breathe through one side at a time; passing a catheter into newborn's nares, one at a time, also demonstrates patency
Nares: Grimace or cry in response to strong odors passed under nose	Normal; intact olfactory nerve	I	
Nares: Breathing detected bilaterally	Patent	A	*Technique:* With a stethoscope, auscultate for breathing one side at a time
Eyes			*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1
Sclera: Clear	Normal	I	
Conjunctiva: Clear	Normal	I	
Iris: Colored evenly, bilaterally	Normal	I	*Variation on normal:* Brushfield's spots (these gold flecks may be normal if not found with other anomalies)
Pupils: Equal bilaterally and reactive to light	Normal; intact oculomotor nerve	I	*Technique:* Examination is done in a darkened room with penlight or flashlight; if done with a newborn in an incubator or in the nursery, shield the infant's eyes as much as possible

Eyes			
Cornea: Clear	Normal; intact	I	*Prematurity:* Hazy
Retina: Transparent	Normal; intact	I	
Lacrimal duct: Patent	Normal	I	*Definition:* Lacrimal duct—any of several small ducts that carry tears from the lacrimal gland to the fornix of the conjunctiva
Blink reflex: Reactive	Intact optic nerve	I	*Technique:* Responds to bright light
Red reflex: Present	Lens intact	I	*Technique:* Hold a direct ophthalmoscope close to the examiner's eye with the ophthalmoscope lens power set at 0. In a darkened room, the ophthalmoscope light should then be projected onto both eyes of the child simultaneously from approximately 18 inches away. To be considered normal, a red reflex should emanate from both eyes and be symmetric in character. *Variation on normal:* Transient opacity from mucus in the tear film that is mobile and completely disappears with blinking
Eyelids: Without edema, ptosis, or epicanthal folds	Normal; intact oculomotor nerve	I	*Definition:* Ptosis—a drooping of the upper eyelid *Definition:* Epicanthic fold—a prolongation of a fold of the skin of the upper eyelid over the inner angle or both angles of the eye
Doll's-eye response: Present	Normal: Intact trochlear, abducens, and oculomotor nerves	I	*Technique:* With the infant in supine position, turn the head from one side to the other; eyes move to the side opposite the side to which the head is turned
Eye position: Without slant	Normal	I	
Ears			*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1
Position: In straight line with eyes; vertical angle that is greater than straight vertical line; without slant	Normal	I	
Skin tags: Absent	Normal	I	
Cartilage formation: Well-curved pinna, sturdy, stiff cartilage, instant recoil	Normal	I/Pa	*Technique:* Palpate the entire pinna of the ear for the presence of cartilage *Prematurity:* Cartilage flattened or folded with slow recoil
Hearing: Neonate startles or cries in reaction	Normal; intact auditory nerve	I	*Technique:* Use a loud noise or snapping fingers to elicit response
Otoscopic examination: • Umbo (cone) of light present, pearl-gray tympanic membrane may have vernix • Membrane is movable without bulging	Normal; intact ear without infection	I	*Technique:* This examination is often omitted because it is difficult to perform and may be potentially harmful if the examiner is not skilled; ear should be pulled down and back for examination
Neck			
Shape: Symmetrical	Normal	I	*Variation on normal:* Asymmetrical shape may be due to fetal position in utero

Neck			
Head: • Turns from side to side equally, full range of joint motion • Shape symmetrical	Normal	I	
Length: Short, without excessive skin	Normal	I	
Tonic neck reflex: Asymmetrical and present but decreases	Normal	I	*Technique:* Place the infant in supine position; turn the head to one side with the body restrained; extremities on the side to which the head is turned are extended, but extremities on the other side are flexed; newborn's attempt to right the head when turned to the side tests the accessory nerve *Prematurity:* May be asymmetrical and strongly present
Thyroid: Midline	Normal	P	
Lymph nodes: Not palpable	Normal	P	
Masses: None	Normal	P	
Carotid: Pulse rate strong and regular	Normal cardiac and circulatory function	P	*Technique:* Do not massage the carotid artery or neck, as this action can result in reflex bradycardia
Clavicles: • Even and without "lumps" along bones • Symmetrical	No fractures	P	
Abdomen and Thorax			
Chest circumference: 30–36 cm	Average for full-term neonate	I	*Prematurity:* Chest circumference < 30 cm
Diaphragm: Equal excursion	Normal	I	
Ribs: Symmetrical	Normal	I	
Breast: Nipple spacing on line without extra nipples	Normal	I	*Variation on normal:* Extra nipple
Areola: Raised and without discharge	Full term	I	*Variations on normal:* Flat areola, discharge, or hypertrophy may be due to maternal hormonal influence *Prematurity:* Flat areola and/or discharge
Abdomen: Rounded, contoured, symmetrical	Normal	I	*Technique:* To document distension accurately, measure the abdominal girth every 4 hours to detect changes *Prematurity:* Abdominal edema or distension may be present
Umbilical cord: • 3 vessels (2 arteries, 1 vein) • Bluish white color	Normal	I	*Variation on normal:* 2 vessels (1 artery, 1 vein)
Abdominal musculature: Strong	Normal	I	
Peristaltic waves: Not visible	Normal bowel activity	I	
Bowel sounds: Present	Normal	A	
Abdomen: No bruit	Normal	A	

Abdomen and Thorax			
Renal: No bruit	Normal	A	
Xiphoid process: Present	Normal; intact	Pa	
Ribs: Without masses or crepitus	Intact, without defects or "air leaks"	Pa	*Definition:* Crepitus—grating or crackling sound or sensation (like that produced by the fractured ends of a bone moving against each other)
Breast tissue: 1 cm	Normal: Full term	Pa	*Technique (NBS):* Palpate to accurately assess breast tissue *Prematurity:* Breast tissue may be < 1 cm and as little as 5 mm
Abdomen: • Soft • Nontender • Without masses	Normal	Pa	*Prematurity:* Separation of abdomino-rectus muscles (diastasis recti) is common
Kidneys: • 4–5 cm in length • Right kidney lower than left • Found in abdomen and posteriorly in lumbar or flank area	Normal	Pa	*Technique:* Palpate with the newborn's legs flexed in a fetal position to relax the infant
Liver: • Sharp edge just above right costal margin • Firm	Normal	Pa	
Spleen: 1 cm below left costal margin	Normal	Pa	
Bladder: Not distended	Normal kidney and urinary tract system	Pa	*Technique:* Be sure to evaluate immediately post void or may be falsely evaluated as distension
Groin: • Femoral pulse rate strong and regular bilaterally • No hernias or masses	Normal	Pa	
Gastric bubble: • Just below left costal margin and toward midline • Tympanic	Normal	Pe	
Abdomen: Tympanic except dull over liver, spleen, and bladder	Normal liver, spleen, bladder, no masses (indicated by dullness)	Pe	*Technique:* Be sure to examine post-void
Genitourinary Tract, Female			
Labia majora: Present and extend beyond labia minora	Full term	I	*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1 *Prematurity:* Labia majora smaller than labia minora
Labia minora: Present and well formed	Full term	I	*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1 *Prematurity:* Labia minora larger than labia majora
Clitoris: Present, may be enlarged	Full term	I	*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1 *Prematurity:* May also be enlarged

Genitourinary Tract, Female			
Urethral meatus: Present in front of vaginal orifice	Normal	I	
Vagina: Patent with or without white discharge	Normal	I	*Variation on normal:* Slight bleeding related to maternal (?) hormonal influence
Genitalia: Distinguishable as female or male	Normal	I	
Perineum: Smooth	Normal	I	
Anus: Midline, patent	Normal	I	*Technique:* Test by gently attempting to insert small non-latex gloved finger
Anal wink: Present	Normal sphincter	I	*Technique:* Light stroking of anal area produces constriction of sphincter
Genitourinary Tract, Male			
Penis: • Straight • Proportionate to body • Length: 2.8–4.3 cm	Normal	I	
Urinary meatus: Midline At tip of glans	Normal	I	*Technique:* If male neonate is uncircumcised, gently retract the foreskin. If circumcised, also check for edema or bleeding.
Urinary stream: Straight from penis	Normal urinary pattern	I	First void should occur within 24 hours after birth
Testes and scrotum: Full, numerous rugae	Full term	I	*Technique (NBS):* With the infant supine and quiet, score as indicated on Figure 37-1 *Prematurity:* Testes and scrotum may be flaccid, smooth, or with few rugae
Pigmentation: Dark	Normal	I	
Perineum: Smooth	Normal	I	
Anus: • Midline • Patent	Normal	I	*Technique:* Test by inserting small non-latex gloved finger
Anal wink: Present	Normal sphincter	I	*Technique:* Light stroking of anal area produces constriction of sphincter
Testes: Descended on at least one side	Full term	Pa	*Variation on normal:* Undescended testes *Prematurity:* Testes not palpable, though they may be found high in the inguinal canal
Upper Extremities			
Length: • In proportion to each other • Lower extremities and body symmetrical	Normal	I	
Full range of joint motion, including: • Abduction • Adduction • Internal and external rotation • Flexion • Extension as applicable to joint	Normal	I	*Physiology:* Full flexion of upper extremities comes with maturity *Prematurity:* • Limited range of joint motion • Limited range of flexion

Upper Extremities			
Shoulder: Full range of motion and flexion	Normal	I	
Clavicles: Full range of motion and flexion	Normal	I	
Elbow: Full range of motion and flexion	Normal	I	*Variation on normal:* Limited range of motion or flexion of the elbow may be related to fetal position in utero
Wrist: Square window test	Normal	I	*Technique (NBS):* Test for square window by flexing the infant's wrist on the forearm, then measure the angle according to gestational examination chart (i.e., 0-degree angle for term neonate); exert pressure sufficient to get as much flexion as possible *Variation on normal*: Limited range of motion or flexion of the wrist may be due to fetal position in utero *Prematurity:* • Limited range of motion or flexion of the wrist • Square window angle > 0 degrees
Hand–grasp reflex: Present, strong, equal bilaterally	Normal; maturity	I	*Variation on normal:* Weak, absent, or unequal grasp reflex may be due to fetal position in utero *Prematurity*: Hand–grasp reflex may be weak or absent, or unequal bilaterally
Scarf sign: Elbow short of midline	Maturity	I	*Technique (NBS):* With the infant supine, take the infant's hand and draw it across the neck and as far across the opposite shoulder as possible. Assist the elbow by lifting it across the body. Observe the position of the elbow to the chest. Grade the position according to the gestational chart. *Prematurity:* Scarf sign—elbow beyond midline
Arm recoil	Maturity	I	*Technique (NBS):* With the infant supine, fully flex the forearm for 5 seconds, then fully extend by pulling the hands, and release; recoil time is graded *Prematurity:* Arm recoil—slow
Palm: No simian creases	Normal	I	*Definition:* Simian crease—a deep crease extending across the palm that results from the fusion of the two normally occurring horizontal palmar creases and is found especially in individuals with Down syndrome
Fingers: • 10 digits and without webbing • Equal spacing	Normal	I	
Carpals and metacarpals: • Present • Equal bilaterally	No fractures; bone formation normal	I	
Nails: Extend beyond nailbeds	Normal; full term	I	
Nailbeds: • Pink • Brisk capillary refill (< 3 seconds) • Equal bilaterally	Normal peripheral perfusion, normal oxygenation	Pa	

Upper Extremities			
Clavicles: Symmetrical Without fractures or pain	Normal	Pa	
Humerus, radius, and ulna: • Present • Symmetrical • Without fractures	Normal bone formation	Pa	
Pulses: • Brachial and radial strong and equal bilaterally • Brachial and radial strong and equal in comparison with femoral pulses	Good peripheral perfusion, without obvious cardiac defects	Pa	
Lower Extremities			
Legs: • Length in proportion to body • Equal bilaterally • Limbs straight	Normal extremity length	I	
Toes: • 10 digits • Without webbing • Equal spacing	Normal	I	
Feet: Straight	Normal	I	*Variations on normal:* • Valgus (turned outward) or varus (turned inward) position of the foot may be related to fetal position in utero and usually resolves • Pedal edema may be due to pressure exerted by fetal position in utero
Ankle dorsiflexion: 0-degree angle	Maturity	I	*Technique:* Foot is flexed back on ankle, then angle between foot and ankle is measured *Prematurity:* Ankle dorsiflexion angle $>$ 0 degrees; may be up to 90 degrees in very premature neonates
Popliteal angle: $<$ 90 degrees	Maturity	I	*Technique (NBS):* With the infant supine and the pelvis flat on the examining surface, the leg is flexed on the thigh and the thigh fully flexed using one hand; with the other hand, the leg is then extended *Prematurity:* Popliteal angle: $>$ 90 degrees and $<$ 180 degrees in very immature infant
Heel-to-ear maneuver	Maturity		*Technique (NBS):* With the infant supine, hold the infant's foot with one hand and move it as near to the head as possible without forcing it; keep the pelvis flat on the examining surface. The heel will not reach the ear but only near the shoulder area in a full-term infant. *Prematurity:* The heel reaches the ear, or just short of the ear
Nails: Extend to end of nailbed	Normal; maturity	I	*Prematurity:* Nails do not extend to end of nailbed

Lower Extremities			
Nailbeds: • Pink • Brisk capillary refill (< 3 seconds)	Good peripheral perfusion	I/Pa	
Plantar creases: Cover the sole of foot	Maturity	I	*Technique (NBS)*: With the infant supine and quiet, score as indicated on Figure 37-1 *Prematurity:* Plantar creases are few or cover only the anterior third of the sole of the foot
Buttocks: Creases symmetrical	Normal hips	I	
Fibula, tibia, trochanter, and femur: • Present • Equal bilaterally	No fractures; bone formation normal	Pa	
Tarsals and metatarsals: • Present • Equal bilaterally	No fractures; bone formation normal	Pa	
Full range of joint motion (includes abduction, adduction, internal and external rotation, flexion, and extension as applicable to respective joints of legs, knees, ankles, feet, and toes)	Normal; maturity	Pa	*Prematurity:* Limited range of joint motion; flexion of legs, knees, ankles, feet, and toes is limited
Hips: Without clicks and full range of joint motion	Normal range of joint motion and no clicks	Pa	*Technique*: Ortolani's maneuver—flex the newborn's hips and knees, then abduct and adduct the hip to detect a slipping of the hip out of the acetabulum or an uneven motion unilaterally *Technique:* Barlow's maneuver—flex the newborn's hips and knees, then place a finger on the femur and trochanter, put the hip through full range of joint motion, and listen for an audible click
Knee jerk or patellar reflex: • Present • Symmetrical	Normal; mature	Pa	*Prematurity:* Knee jerk or patellar reflex absent, weak, or asymmetrical
Flexor plantar reflex: • Present • Symmetrical	Normal; mature	Pa	*Technique:* Stroking the lateral part of the sole of the foot with a fairly sharp object produces plantar flexion of the big toe; often there is also flexion and adduction of the other toes *Prematurity:* Plantar reflex absent, weak, or asymmetrical
Extensor plantar (Babinski) reflex: • Present • Symmetrical	Normal; mature	Pa	*Technique:* When the sole of the foot is firmly stroked, the big toe extends (bends back toward the top of the foot) and the other toes fan out; this is a normal reflex up to about 2 years of age

Back			
Spinal column: Straight	Normal alignment	I	*Variation on normal*: A curved spinal column should gradually resolve if it results from fetal positioning while in utero
No visible deviations or defects	Intact	I	*Definition:* Pilonidal cysts—containing hair nested in a cyst; used to describe congenitally anomalous cysts in the sacrococcygeal area that often become infected and emit discharge through a channel near the anus
Vertebrae: Present, without enlargement or pain	Normal spinal column	Pa	
Anus: See genitourinary tract		Pa	
Buttocks: See lower extremities		Pa	

Abbreviations: A, auscultation; I, inspection; MOE, method of evaluation; NBS, New Ballard Scale; Pa, palpation; Pe, percussion; RBC, red blood cell.

Based on American Academy of Pediatrics, Section on Ophthalmology, American Association for Pediatric Ophthalmology and Strabismus, et al. Red reflex examination in neonates, infants, and children. *Pediatrics.* 2008;122:1401-1404; Tappero EP, Honeyfield ME. *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination*. 5th ed. New York, NY: Springer; 2016; Boucher N, Marvicsin, Gardner SL. Physical examination, interventions, and referrals. In: Snell BJ, Gardner S, eds. *Care of the Well Newborn.* Burlington, MA: Jones & Bartlett Learning; 2017:101-134.

CHAPTER

38

Neonatal Care

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Introduction

The role of the midwife in caring for the neonate during the first month of life varies markedly. In some locales, the midwife has little formal role once the newborn leaves the birthing room. In other practices, the midwife will continue care of the woman and newborn throughout the first several weeks of life. The *Core Competencies for Basic Midwifery Practice*, revised in 2012, states that certified nurse-midwives and certified midwives "independently manage the care of the newborn immediately after birth and continue to provide care to well newborns up to 28 days of life."[1] Regardless of the formal scope of practice in specific settings, parents often call their midwife with questions related to the care and well-being of their newborn. Thus, knowledge of normal newborn behaviors and signs of newborn disorders is an important component of practice for all midwives.

During the first hour after birth, a newborn must successfully navigate the physiologic transition from an intrauterine environment to an extrauterine one. This process is described in the *Anatomy and Physiology of the Newborn* chapter. A complete newborn examination is presented in the *Examination of the Newborn* chapter. This chapter reviews newborn care through the first month of life.

The First Golden Hour

The concept of the Golden Hour originated in the care of trauma victims, for whom the first 60 minutes of care tended to predict death or survival. Today this term can also be applied to the important hours and actions immediately after birth.[2]

The initial brief examination of the newborn is performed immediately after birth. Although it is not considered a formal physical examination, many important assessments are made during this evaluation that takes place in the first minute of life before a formal Apgar score is awarded. While the newborn is placed on the mother's abdomen or remains in the midwife's hands, the midwife focuses on identifying various neonatal parameters of health such as tone, color, and heartbeat (which can be palpated through touching the umbilical cord that has not been clamped and cut), as well as any obvious birth injuries or congenital abnormalities. In addition, physiologic responses of the newborn and corresponding parental behaviors are observed, all while maintaining vigilance regarding maternal well-being. Resuscitation is initiated if required, before the 1-minute Apgar score is given.

The second assessment is assignment of the Apgar score, preferably by someone other than the person conducting the birth so as to decrease bias. The Apgar score is discussed in more detail in the *Second Stage of Labor and Birth* chapter. Depending on the birth site guidelines or clinical indication, umbilical artery blood gases may be drawn. Also, if the parents have requested cord blood banking, it is obtained at this time.

Newborn Assessment and Care in the First Hours After Birth

In past decades, umbilical cords were immediately clamped and cut and the newborn was removed to a warmer, where professionals could perform immediate care measures, including a physical examination and assignment of the Apgar score. Several well-conducted studies have called into question the need for immediate evaluation and routine interventions with the newborn separated from the mother during the first hour after birth.[3]

Skin-to-Skin Contact

In lieu of separating mother and child, placing the newborn on the mother's abdomen provides several advantages. The newborn is dried while on the mother, and her warmth decreases neonatal chilling. Early skin-to-skin contact (SSC), also known as kangaroo care, promotes longer breastfeeding and has been found to result in less crying among newborns when compared to infants who are separated from their mothers immediately after birth.[4] Other potential advantages of SSC are purported to be related to the establishment of a trusting relationship between mother and child at a sensitive period, although long-term studies of the "sensitive period" hypothesis have not proved that SSC improves maternal–infant bonding over time.

Delayed Umbilical Cord-Clamping

Approximately 5 minutes after initial SSC is initiated, the umbilical cord is clamped and cut. Often the midwife performs this task, but the woman's partner or designated support person also may cut the cord between the two clamps. Waiting for at least 3 minutes to pass before clamping and cutting the cord—a practice known as delayed cord-clamping—allows for blood in the umbilical and placental circulation to be physiologically transfused to the newborn. Delayed cord-clamping is recommended for all newborns.[5-8] Although professional association guidelines have some differences, the evidence indicates a minimum of 3 to 5 minutes is optimal for the newborn.[8-12] The benefits and procedure for delayed cord-clamping are reviewed in the appendix to the *Second Stage of Labor and Birth* chapter.

Neonatal Hypothermia

Newborns are predisposed to developing hypothermia, as they have a large surface area per unit of body weight compared to adults. As discussed in the *Anatomy and Physiology of the Newborn* chapter, brown fat is the major site for production of heat and serves as the circulatory route for heat production, or nonshivering thermogenesis. Hypothermia is associated with depression of the central nervous system, hypoglycemia, metabolic acidosis, tachypnea, respiratory distress, and decreased peripheral perfusion. The newborn is at higher risk for hypothermia following hypoxic, hypoglycemic, or other stressful episodes.

Skin-to-skin contact provides the best source of heat for the newborn and can prevent

hypothermia.[4] SSC has also been found to be at least equal to incubators for rewarming infants who already have experienced chilling,[13] and has additional benefits in facilitating breastfeeding initiation.[4] **Table 38-1** lists other methods used to prevent hypothermia.

Table 38-1	Interventions to Prevent Newborn Hypothermia
Prewarm the newborn resuscitation area.	
Set the birth room temperature at 23.9°C (75°F).	
Prewarm any blankets, hats, or clothing prior to the birth.	
Place the newborn skin to skin on the mother immediately after birth.	
Gently dry the newborn after birth, and then immediately replace wet blankets with dry ones.	
Do not evaluate the newborn on wet sheets or towels.	
Postpone the newborn's bath until newborn temperature has been stable for 2 hours.	
Place the newborn care areas away from windows, outside walls, and doorways.	
Keep the newborn's head covered and body well wrapped.	

Maintaining the temperature of the birth room or operating room at 77°F (25°C) for term newborns and at 79–82°F (26–28°C) for preterm newborns is recommended by the World Health Organization (WHO).[14] Covering the newborn's head with a stockinet cap after initial drying is conventional care because the head has a large surface area from which heat can be lost; however, the cap may interfere with maternal–newborn odor recognition and the necessity of placing a cap on the newborn has not been proven. A warm, dry blanket tented over the newborn's head while the newborn is skin to skin with the mother will assist in thermostabilization.

The newborn's temperature can be assessed at various sites with different types of thermometers. Although historically rectal temperatures were commonly performed in newborns, this technique had disadvantages: The rectal temperature can remain high after the core temperature drops, it can be affected by the presence of stool, and rectal perforation is possible. Thus, rectal temperatures are no longer recommended. Axillary temperatures are normally in the range of 36.5–37.5°C (97.7–99.0°F) but may be falsely elevated in a cold-stressed infant secondary to metabolism of brown adipose tissue. Abdominal skin temperature is normally in the range of 36–36.5°C (96.8–97.7°F). Infrared tympanic thermometers (2 seconds in the ear) can be used to measure temperature but are likely the least reliable method for a newborn.

Noncontinuous temperature assessment will be accurate only if the thermometer remains in place for an adequate time. If temperature is assessed with an abdominal skin sensor, the sensor must be in good contact with the skin and covered with a reflective shield. Skin temperatures should be slightly lower than core temperatures.

Neonatal Hypoglycemia

In the first 2 hours post birth, a newborn's blood glucose level normally falls to a nadir of

approximately 40 mg/dL. In utero, the fetus has a continuous supply of glucose. Once outside the mother's body, the newborn must adapt to the change from this continuous supply to intermittent supply via breast milk. Blood glucose levels may remain slightly low for several days and the newborn will remain asymptomatic. Because there is a great deal of individual variability in blood glucose levels associated with clinical symptoms of hypoglycemia, no specific blood glucose value that defines hypoglycemia has been published.[15,16]

Despite the definitional controversy, newborns are at increased risk for hypoglycemia and specific risk factors have been identified. Newborns born to women who have diabetes, preterm infants, and newborns who are small for gestational age are at increased risk for hypoglycemia. Common symptoms of hypoglycemia include jitteriness, irritability, lethargy, or poor feeding. Blood glucose levels obtained by heel stick can be of value in diagnosing hypoglycemia. Any glucose value of 45 to 50 mg/dL or lower should be verified immediately via a venous sample, and the pediatric provider informed of the results.

Early and frequent feeding is an important preventive measure that protects most newborns from developing clinical hypoglycemia. The exact degree of hypoglycemia that requires formula feeding or intravenous glucose depends on other clinical factors such as feeding ability, weight, and risk factors for hypoglycemia.[17] Although hypoglycemia can be easily treated, in extreme circumstances prolonged hypoglycemia is associated with neurodevelopmental delays.

Evaluation of hypoglycemia is done via capillary blood sample. It is no longer recommended to warm the heel prior to sampling, as research has not revealed any benefit from heel warming with regard to blood flow, collection time, or newborn comfort.[18,19] When performing this blood test, care should be taken to avoid puncturing sensitive structures on the back of the heel (**Figure 38-1**).[18] Use of an automated blood collection device may cause less trauma and lead to more successful sample collection results than use of manual lancets and squeezing the heel, which creates hemolysis.[20] To prevent infection, the infant's heel should be cleansed with antiseptic, followed by sterile water, and allowed to air dry. Alcohol should be avoided, as it is drying and can leave residue. Newborn discomfort may be diminished by swaddling, being held in the arms of the neonate's mother or other family member, and non-nutritive sucking.[21]

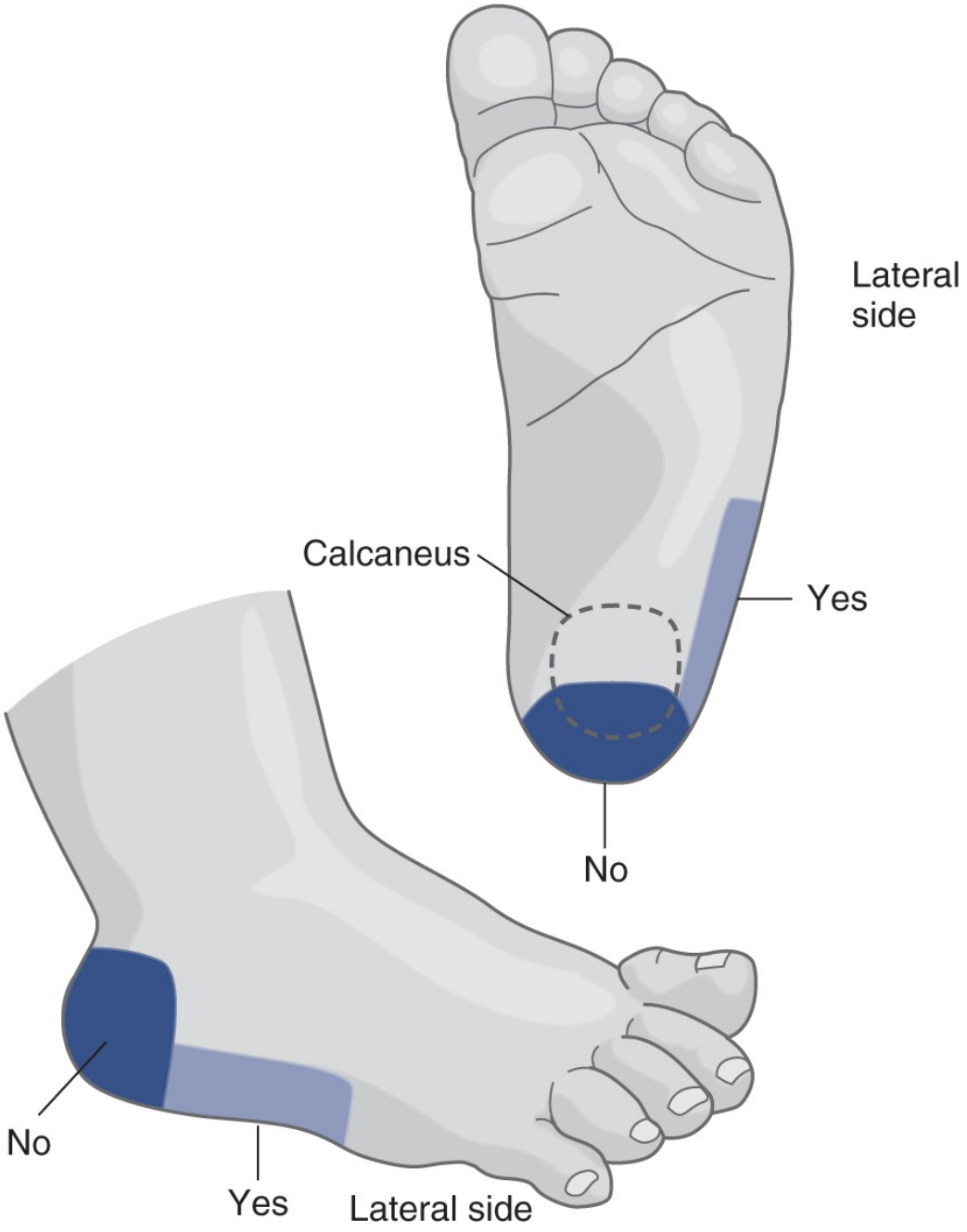


Figure 38-1 Anatomic landmarks for heel sticks.

Any glucose level below the recommended threshold for normal requires intervention. Feeding the infant is the first step. A repeat glucose test is performed 30 minutes after feeding and if the borderline level persists, a venous sample should be checked. Low values should be evaluated immediately with a venous sample drawn from a scalp vein or the antecubital fossa. If the low value is confirmed, pediatric consultation is needed and treatment is indicated.

Venous samples can be analyzed using either whole blood or plasma (serum). Samples should be chilled during transport to the laboratory. Most laboratories report the plasma result, which will be close to 15% higher than a sample run on whole blood. Thus, it is important to know the testing method and reference ranges used by a particular lab to accurately interpret the test results.

Initial Newborn Prophylaxis Treatments

Two prophylactic treatments are recommended for newborns within the first 4 hours of life: ophthalmic treatment for prevention of ophthalmia neonatorum and administration of vitamin K to prevent hemorrhagic disease of the newborn.

Ophthalmic Treatments

Eye prophylaxis is pharmacologic treatment originally intended to prevent blindness associated with undiagnosed maternal *Neisseria gonorrhoeae* infection. Today in the United States, neonatal conjunctivitis (also known as ophthalmia neonatorum) is more commonly caused by the *Chlamydia trachomatis* organism. Maternal screening and treatment during pregnancy is the first step in prevention of blindness caused by chlamydia or gonorrhea, but does not guarantee that reinfection has not occurred at the time of birth.[22]

The risk that the infant will acquire conjunctivitis is 20% to 50% if a woman is infected when she gives birth. Neonatal conjunctivitis secondary to *C. trachomatis* infection usually becomes symptomatic 5 to 14 days after birth.[22] Conjunctivitis secondary to *N. gonorrhoeae* infection becomes symptomatic 1 to 5 days after birth; the risk that the infant will acquire conjunctivitis is 30% to 40% when the mother has gonorrhea.

In years past, silver nitrate or tetracyclines were employed as treatments to prevent colonization from causing conjunctivitis or blindness. Today, erythromycin 0.5% ophthalmic ointment is the Centers for Disease Control and Prevention (CDC) recommendation for neonatal ocular prophylaxis throughout the United States. Its effectiveness against *N. gonorrhoeae* is proven, and this medication is considered the only available prophylaxis against *C. trachomatis* conjunctivitis.[22] Infants may develop a chemical conjunctivitis after eye prophylaxis, but that condition resolves spontaneously within 24 to 48 hours. In low-resource countries, small studies of the use of povidone–iodine eventually may provide an alternative treatment.[22]

The medication should be applied within 2 hours after birth. Some legal jurisdictions mandate that the eye prophylaxis be performed within the first hour of life, but in those situations many professionals wait until the end of the hour to apply this treatment so as to promote better *en face* interactions between parents and newborns. The medication is placed on each of the lower conjunctival sacs and spread with gentle massage of the eyelids. The excess ointment can be wiped away after 1 minute, but the eyes should not be irrigated.

Prophylactic Administration of Vitamin K

Newborns do not have a major store of vitamin K. In addition, the levels of vitamin K are low in breast milk, and the neonate’s gut flora, which usually includes bacteria that produce vitamin K, tends to be immature. A deficiency of this vitamin can result in the condition termed hemorrhagic disease of the newborn (HDN) or vitamin K–deficiency bleeding (VKDB).

HDN commonly is categorized as either early (within 24 hours of life), classic (days 1–7), or late (2–12 weeks). Early HDN is rare and requires immediate pediatric consultation when the infant is bleeding, especially from injection sites or the umbilicus. Early HDN is most

commonly found among infants whose mothers have taken pharmacologic agents such as anticonvulsant drugs. Classic HDN is most commonly found among infants who have not received prophylactic vitamin K injection during the first day of life, with the most common bleeding sites being gastrointestinal, cutaneous, or penile (subsequent to a circumcision). Late HDN is most common among newborns who are being exclusively breastfed and have not received prophylaxis; it is potentially the most serious type of vitamin K deficiency, as many infants affected with this variation develop intracranial bleeding.

A single injection of vitamin K, 1.0 mg given intramuscularly, has been found to prevent classic HDN.[23] For exclusively breastfed infants, the intramuscular injection also is protective, although some evidence suggests that an oral administration of 1 mg vitamin K may be effective as well.[24] Controversy exists regarding the best route of administration for prophylaxis of infants at risk of late HDN. Intramuscular injection appears effective and is the only Food and Drug Administration (FDA)–approved route, whereas the single oral dose may be subtherapeutic.[25]

The First Hours: The Newborn with an Unexpected Immediate Health Condition

Prenatal assessments of fetal morphology do not detect all congenital anomalies. When a newborn presents with an unexpected health problem, the midwife identifies deviations from normal and initiates transfer to pediatric care. A critical component of this process is communicating the degree of urgency and the midwife's assessment of how quickly pediatric assistance is needed.

Congenital Defects

Some anomalies, such as cleft lip and extra digits can be surgically corrected without long-term sequelae. Other types of congenital defects can result in rapid deterioration in the newborn and may lead to significant morbidity and mortality if not properly managed. Of these, abdominal wall defects and spinal cord defects pose particular challenges.

Abdominal wall defects are subdivided into gastroschisis and omphalocele, both of which have unclear etiologies.[26] *Gastroschisis* is a condition in which the eviscerated abdominal organs are not covered by a peritoneal membrane sac. In an *omphalocele*, the abdominal organs are external but are covered by peritoneal membrane that protects the intestines from exposure to amniotic fluid and, after birth, ambient air. In both cases, there is a high likelihood of infection, hypothermia, and dehydration because of the large surface area that is exposed.

Management of the newborn with an abdominal wall defect requires an immediate call for pediatric assistance and transport to a tertiary pediatric hospital or unit. The newborn is placed in a radiant warmer in as sterile an environment as possible. Sterile, warmed saline is applied to the eviscerated abdominal contents via sterile gauze pads. The infant's torso is then wrapped in sterile gauze to keep the saline in place. The infant is not fed via breast or a bottle; instead, a feeding tube is inserted and any stomach contents are aspirated. This neonate has an acute need for intravenous fluids.

The two most common neural tube defects are meningocele and meningomyelocele (also called spina bifida).[27] *Meningocele* is a bony defect of the spinal cord. With *meningomyelocele*, the vertebra is defective and the spinal cord and spinal roots are externally located in a meningeal sac. Meningomyeloceles are usually found in the lower spine, lumbar, and sacral areas. The principles for immediate management of spinal cord defects are similar to those described for abdominal wall defects: application of a sterile, warm saline dressing with a dry sterile overwrap; thermoregulation; and fluid maintenance. In addition, the infant is positioned prone and fecal contamination is scrupulously avoided.

Diaphragmatic hernias are surgical emergencies in the newborn because herniation of abdominal contents into the chest cavity may have caused pulmonary hypoplasia.[28] These hernias are usually unilateral on the left. The degree of respiratory distress is directly related to the amount of lung tissue that has been compromised. In some newborns, the herniation is so severe that there has been stunted, if any, lung growth on the affected side. Signs of diaphragmatic hernia include decreased left-sided breath sounds, heart sounds on the right side, and severe respiratory distress at birth secondary to persistent pulmonary hypertension.

Depending on the degree of the defect, the abdominal contents may be in the chest cavity, which causes a concave (scaphoid) abdominal contour. Skilled pediatric care must be sought immediately.

Birth Injuries

Palsies and plexus conditions generally are categorized as birth injuries and traditionally have been assumed to be associated with an event such as shoulder dystocia. Nevertheless, injury may sometimes occur in utero, such that parents and professionals are surprised when the child is born with a palsy or injury after an uneventful labor and birth.[29,30]

Facial Palsy and Brachial Plexus Injuries

Injuries to the face include bruising from forceps or facial palsy caused by either forceps or pressure from the maternal sacrum. Consultation may be needed, and use of an eye patch and lubricating eye drops are common treatments. Injuries to the brachial plexus may occur prenatally or during a birth when traction is applied to the neck.[30] Such injuries can occur during breech births, during births involving shoulder dystocia, or following an operative vaginal birth.

Management includes referral for splinting of the affected arm close to the body and consultation with the pediatric team. Parents are encouraged to minimize handling the affected extremity for the first week because of the pain involved. Parents can be reassured that in the vast majority of newborns, paralysis disappears in 3 to 6 months, with initial improvement becoming evident within a few weeks. Physical therapy is helpful after the first swelling subsides.

Injuries to nerve roots higher in the brachial plexus (C3–C5) can cause signs of significant respiratory compromise because of paralysis of the phrenic nerve and diaphragmatic compromise.[30] Newborns with this type of nerve injury take very shallow breaths with limited respiratory excursion and need aggressive respiratory support after birth. When injuries do not respond to 6 months of physical therapy, surgical repairs are often attempted.

Bone Fractures

Management of bone fractures includes obtaining a pediatric consultation and splinting. If a clavicle or arm fracture is suspected, parents can be assured that newborn bones usually heal well and quickly.

Cephalohematoma, Subgaleal Hemorrhage, Intracranial Hemorrhage, and Skull Fractures

Injuries to the head include cephalohematoma, subgaleal hematoma, intracranial hemorrhage, scalp abrasions, retinal hemorrhage, and skull fractures.[31] Cephalohematomas and small scalp abrasions occur in approximately 10% of newborns who are delivered by vacuum extraction.[31] Cephalohematomas can also occur during a spontaneous vaginal birth, although the incidence

of such injuries is much lower. More severe cranial hemorrhages are more often associated with forceps deliveries.

Pediatric support is needed urgently if symptoms of a subgaleal hemorrhage are present. Symptoms of intracranial hemorrhage generally appear a few hours after birth and include irritability, apnea, poor feeding, or lethargy, and possibly a bulging fontanel.

A clear sign of a skull fracture is a depressed area of fetal skull, particularly over the parietal bones. An area of skull depression increases the possibility that fragments of skull bone have penetrated through the dura, the covering of the brain. Management includes careful positioning of the newborn on the side opposite the affected area and consultation with the pediatric team, who will order imaging tests.

The First Golden Days: Normal Newborn Behavior

Parental advice from professionals, websites, social media, and even family often assumes that all newborns act similarly. In reality, each neonate is an individual—and all information, especially about behavior, should be viewed in that light. The midwife can help parents develop realistic expectations about normal newborn behavior during the newborn period. If this is the only aspect of newborn care in which the midwife is involved, it can be one of the most important and significant contributions offered by a healthcare professional to the new family in the first several weeks postpartum.

Sleep and Wake States

Newborns are in a period of behavioral instability. By the time parents figure out a pattern of newborn behavior, that pattern will have changed. A good rule of thumb for the first month of life is "There is no pattern."

Newborns' activities fall into two major categories of behavior: periods of waking and periods of sleeping. The waking states include crying, considerable motor activity, alert, and drowsy. The sleep states include active (light) sleep and deep sleep. **Table 38-2** summarizes these states, along with their implications for care.[32,33]

Table 38-2 **Infant Behavioral States**

State	Appearance	Eye/Facial Movements	Breathing	Response to Stimuli	Implications
Sleep States					
Quiet sleep (deep sleep)	Essentially still with occasional startle	Occasional regular sucking, otherwise still	Smooth and regular	Tolerates stimuli well; only responds to intense stimuli	Avoid trying to feed at this time; will lead to frustration
Active sleep (light sleep/REM sleep)	Some body movements	REM Fluttering of eyes with occasional smiling or crying sounds	Irregular	More responsive to hunger and handling than in quiet sleep; if stimulated, may return to active sleep to progress to quiet sleep or arousal	Highest proportion of newborn sleep Usually occurs before arousal Although may provide evidence of hunger, will not eat well in this state
Awake States					
Drowsy	Activity varies	Eyes may open and close or be slit-like in appearance Although often no facial movement, may have some smiling or sounds	Irregular	Will respond to stimuli, although response may be delayed	To promote awakening, provide some stimulation such as visual, auditory, or sucking stimulus
Quiet alert	Minimal movements	Eyes widen Face appears bright and attentive	Regular	Very responsive	Often occurs immediately after nonmedicated birth and can be positive for parent–child interactions As baby ages, spends more time in this state Good state in which to feed baby
Active alert	Smooth movements but variable activity	Eyes are open, but appearance is more dull Although often no facial movement, may have some smiling or sounds	Irregular	Will respond to stimuli, although response may be delayed	More sensitive to stimuli (e.g., hunger, handling) May move into crying state If fatigued, may move into drowsy or sleep state
Crying	Increased body movements, often with darkening skin tones	Eyes may be open or closed Grimaces	More irregular than other states	Very responsive, especially to negative stimuli	Sometimes crying infants can self-console May need swaddling, cuddling, or other measures to resolve crying

Abbreviation: REM, rapid eye movement.

Based on Blackburn S, Bakewell-Sachs S. *Understanding the Behavior of Term Infants.* White Plains, NY: March of Dimes Birth Defects Foundation. Available at: http://www.marchofdimes.com/nursing/modnemedia/othermedia/states.pdf. Accessed July 12, 2017.[33]

During the first month of life, the percentage of time spent in each of these states changes. Healthy newborns spend as much as 60% of their time sleeping. However, much of this sleeping takes the form of short naps. As the first month of life progresses, infants shift away from active (light) sleep and toward more extended periods of deep sleep. Similarly, there is a shift in the waking states toward an increase in alertness.

Newborn Reflexes

The newborn has two categories of reflexes: *proprioceptive* and *exteroceptive*. The proprioceptive reflexes include gross motor reflexes such as the Moro reflex, which can be checked at any time. Complete absence of any of these reflexes is a cause for alarm. The exteroceptive reflexes are best evoked when the infant is quiet and alert, as they are stimulated by light touch. They include the rooting, grasping, plantar, and superficial abdominal reflexes. An incomplete elicitation of a reflex may occur in some circumstances—for example, with neurologic depression secondary to medication. The loss of a previously strong reflex in the first month of life is a cause for alarm and must be reported promptly to a pediatric provider.

Sensory Capabilities

Sensory capabilities are strongly related to gestational age. Dramatic increases in the sensory stimulation that occur just after birth can lead to neonatal exhaustion, evidenced as fussiness or

aversion. Crying infants and fatigued mothers are frequently associated.[32]

At term, the healthy newborn has an ability to fix on and track objects visually. Many studies have shown a strong newborn preference for patterns of stripes. During the first month of life, however, newborns become preferentially interested in patterns with contours that resemble the human face. The ability to see in color is limited at first, so newborns respond well to black-and-white patterns or strong colors like red. Newborns in the alert behavioral state will spend some minutes staring at patterns.

The newborn has the ability to discriminate among distinctive odors.[34] A newborn can discriminate between the odor of the mother's breast pad and the odors of the breast pads of other nursing women. Newborns react strongly to variations in taste and show a strong preference for sweet liquids. They suck longer and with an increase in heart rate when presented with a sweet liquid in a bottle.

Newborns also have acute hearing and are able to localize sound in the environment. They can discriminate finely among sounds and show a preference for real voices as opposed to synthesized voices. By the end of 1 month, neonates prefer sound with a pattern similar to speech.

Prior to birth, the fetus experienced touch in the form of the shifting amniotic fluid. At birth, the newborn is dry for the first time and is subject to many and varied forms of touch. The ability of the newborn to respond to touch is well demonstrated with the elicitation of the various exteroceptive reflexes such as rooting, grasping, abdominal reflexes, and spinal curving.

Regulation of Behavior

Infants may convey their ability or inability to engage by a variety of neurobehavioral cues. For example, stability and engagement cues include sucking, smiling, feeding posture, flexion of arms and legs, searching for a sound, and facial eye-to-eye contact and normal vital signs. Distress cues include behaviors such as crying, grimace, startle or jerky movements, changes in skin color and turgor, grunting, arching, tachypnea or bradycardia, jitteriness, flaccidity or tremors, and vomiting, frequent regurgitation, or stooling.[35]

Caregivers play a critical role in infant brain development by interacting and helping the infant regulate the stress response system. The caregiver interprets behavioral cues and responds with visual, vocal, and physical interactions that provide feedback to the infant. Each infant has a unique ability to react to stimulation presented by the environment and personal bodily functions. Infants vary in their responses to these stimuli. The newborn learns best in the alert state.

On occasion, a midwife may recommend to parents that the Brazelton Neonatal Behavioral Assessment Scale (NBAS) be performed.[36] This test generates a picture of the infant's communication via responses to stimuli and neurologic reflexes. The results are used to help parents interpret their newborn's behaviors. The test is standardized and can be reliably administered only by a trained examiner. The NBAS begins with the newborn in a sleeping state. Repeated stimulations are offered, and the examiner observes how the newborn adjusts to the stimuli. The behavioral components of the NBAS assess the infant's ability to perform

the six tasks listed in **Table 38-3**.[36]

Table 38-3	Behavioral Components of the Brazelton Neonatal Behavioral Assessment Scale
Organization of behavioral state	
Decrease in motor activity in an attempt to cope with sensory input	
Ability to become alert and oriented to auditory and visual stimuli	
Interaction with a caregiver through cuddling	
Ability for self-consoling	
Habituation to repeated stimulation	

Based on Brazelton TB, Nugent JK. *Neonatal Behavioral Assessment Scale*. 4th ed. London, UK: Mac Keith Press; 2011.[36]

Several other assessment instruments also address infant behaviors and development. For example, the Denver Developmental Screening Test (Denver Scale) assesses children from birth to age 6 years. Similar to the NBAS, this test observes the actions of the child and can assist in identifying conditions suggestive of cerebral palsy. The Broussard Neonatal Perception Inventory explores how mothers perceive their infants and assesses the attachment of the mother to child.[37] A number of other assessment tests exist, but most—if not all—cannot be used as diagnostic instruments.

Psychological Tasks of Early Infancy

During the first months after birth, a profound psychodynamic develops between the infant and the principal caregiver (usually the mother). The infant is performing the psychological task differentiation: identifying as a separate being in relation to other beings. A growing body of research implicates the maternal hormone oxytocin in the initiation and maintenance of human attachment behaviors.[38] The brain is a target organ for oxytocin activity, as it is known to contain oxytocin receptors.

Although most of the literature refers to maternal–infant attachment, it is important to be cognizant of other possible “secure bases” for infants, including fathers, grandparents, or others who become primary caregivers. In discussions with the newborn’s primary caregivers, the midwife can explain the critical importance of response to infant cues.

The First Golden Days of Life: Care of the Healthy Neonate

Breastfeeding

The *Breastfeeding and the Mother–Newborn Dyad* chapter provides an overview of the evidence in favor of the importance of establishing early breastfeeding. The mother–newborn dyad is supported with knowledgeable assistance, especially during the early days when lactation is initiated and learned by both mother and newborn.

Newborn Bathing

WHO recommends delaying bathing for a minimum of 6 hours after immediate drying to prevent hypothermia and to facilitate skin-to-skin warming and early breastfeeding.[39] Several studies have found that water alone is as effective as mild soaps in cleansing infants.[40] Antimicrobial soaps tend to be harsh and are to be avoided.[41] Currently, more rigorous dermatologic studies are needed to clarify the effects that the various bathing methods have on neonatal skin integrity and hydration.[41]

Cord Treatments

In years past, treatment of the umbilical cord stump was initiated immediately or shortly after birth. Various antiseptic solutions were used, including rubbing alcohol, iodine, goldenseal, and echinacea. Today, the prevailing advice is to simply keep the area dry. A Cochrane systematic review found that newborns with cords treated with antiseptic solutions were no more or less likely to develop omphalitis when compared to neonates who received no treatment or placebo.[42] Conversely, some evidence suggests that treatment with an antiseptic solution may prolong adherence of the cord.

The Cochrane review was primarily based on studies in developed nations where births occurred in clean conditions. Few studies have explored treatments in low-resource settings where newborns frequently die from such conditions as neonatal tetanus; however, WHO recommends chlorhexidine as a cord treatment following birth in locations with poor sanitation. In those areas, additional research is needed, especially regarding the potential advantages of antiseptic solutions used to treat the umbilical cord stump.[42]

Newborn Screening and Vaccination

Technological developments in the last 60 years have increased the ability to screen newborns for diseases and conditions that are life threatening but not apparent at birth.[43-49] Routine newborn screening began in the 1960s when it became possible to detect phenylketonuria (PKU). Today newborn screening programs test for a wide variety of conditions.

Most newborn screening tests are mandated in all states within the United States. The states run the newborn screening programs, however, so there are some differences between states in

which tests are included in the screening program. The U.S. Department of Health and Human Services has published a list of tests that are recommended to be included in all state programs. Information about the Recommended Uniform Screening Panel Core Conditions and those included in the state programs can be found in the Resources section at the end of this chapter.

Parental education may be included in the state newborn screening programs; however, in some states, midwives may not be reimbursed for providing this type of health education. Some states mandate testing of only newborns who are born in hospitals and do not address neonates born in out-of-hospital settings. Midwives who practice in out-of-hospital settings may either invest in the appropriate equipment for newborn screening or refer new parents to facilities wherein they can obtain the necessary screening.

There is a general consensus that no test should be included in a state newborn screening program unless treatments—albeit not necessarily "cures"—exist.[49] Factors such as financial and family costs of false-positive results have been discussed as important considerations when a particular test is being evaluated for inclusion in a state program. Privacy of the newborn's genetic material also has been noted as a concern.[49,50]

Point-of-Care Testing

Newborn screening has expanded to include hearing tests and pulse oximetry to detect cardiac conditions. As opposed to dried-blood testing that is performed in a central laboratory, hearing tests and pulse oximetry are performed and interpreted outside of a laboratory and at, or close, to the site of direct care. This type of screening is also known as point-of-care testing.[50]

Hearing Testing

In the United States, universal hearing screening is advocated for all infants younger than the age of 1 month.[51] Approximately half of children with hearing loss have no identifiable risk factors; thus, risk-based screening for these problems has been problematic. Universal hearing screening has been found to be useful and newborns with hearing conditions identified in this manner have benefited from early intervention and ultimately fewer communication disorders.[52]

Hearing programs often follow a two-step process, beginning with otoacoustic emissions (OAE) testing in the first few days of life. If a newborn fails the OAE test, an auditory brain stem response (ABR) is conducted. In some sites, both tests are regularly performed. False-positive results occur occasionally, so confirmatory testing by pediatric providers is necessary. Midwives who conduct out-of-hospital births may find the equipment needed for such testing and its maintenance to be costly. In such a case, they may instead refer parents to sites such as hospitals that will administer the testing.

Pulse Oximetry for Congenital Heart Defects

Although a number of studies in the early twenty-first century found that pulse oximetry is a

good screening method to identify undiagnosed critical congenital heart defects (CCHDs) among otherwise healthy newborns, the initial research had few participants and a low prevalence of CCHDs.[53,54] However, by 2013, a systematic review of almost 250,000 infants found that the screening is highly specific, moderately sensitive, and likely to be cost-effective.[55]

Pulse oximetry screens for several major and secondary CCHDs, including hypoplastic left heart syndrome, pulmonary atresia with intact septum, tetralogy of Fallot, coarctation of the aorta, and transposition of the great vessels. In the normal newborn, oxygenation should be higher than 95%. Less than a 3% difference should be apparent when the pulse oximetry results for the foot and results for the hand are compared. As with hearing testing, midwives who conduct out-of-hospital births may need to find alternative sites that will perform pulse oximetry testing.

Hepatitis B Vaccine

Vaccination of the newborn against hepatitis B within the first 12 hours of life has been recommended in the United States for more than 30 years. The schedule for hepatitis B immunization of a newborn/infant varies based on maternal status. With mothers who are positive for hepatitis B surface antigen (HBsAg), the infant receives both hepatitis B vaccine (HBV) and hepatitis B immune globulin (HBIG) within the first 12 hours of birth.[56] These injections are administered in separate sites. Although the majority of states have laws requiring obstetric providers to screen women for hepatitis B, occasionally a woman presents for care and her HBsAg status is unknown. Neonates born to these women should receive HBV within the first 12 hours of life, and maternal blood should be tested as soon as possible. If the maternal HBsAg test is positive, HBIG should be immediately administered to the newborn within the first week of life.[56]

For several decades, the Centers for Disease Control and Prevention (CDC) has recommended that all newborns be immunized against hepatitis B.[56] Although there is some controversy about the need for the HBV for this age group, seroprotection following the vaccine's administration has been confirmed.[57] The current recommendation is that all infants receive the first dose of HBV within 12 hours of birth. **Table 38-4** summarizes the CDC recommendation based on whether the mother has a negative, positive, or unknown HBsAg test.[56] According to the CDC, licensed vaccines contain 10 to 40 mcg of HBsAg protein per milliliter; they do not contain thimerosal. Only a single-antigen HBV is used for the birth dose.

Table 38-4 Hepatitis B Vaccine Schedules for Term Newborn Infants, by Maternal Hepatitis B Surface Antigen Status

Single-Antigen Vaccine		Single-Antigen + Combination Vaccine	
Dose	**Age**	**Dose**	**Age**
Maternal HBsAg Positive			
1[a]	Birth (≤ 12 hrs)	1[a]	Birth (≤ 12 hrs)
HBIG[b]	Birth (≤ 12 hrs)	HBIG	Birth (≤ 12 hrs)
2	1–2 months	2	2 months
3	6 months	3	4 months
		4[c]	6 months (Pediarix) or 12–15 months (Comvax)
Maternal HBsAg Unknown[d]			
1[a]	Birth (≤ 12 hrs)	1[a]	Birth (≤ 12 hrs)
2	1–2 months	2	2 months
3[c]	6 months	3	4 months
		4[c]	6 months (Pediarix) or 12–15 months (Comvax)
Maternal HBsAg Negative			
1[a,e]	Birth (before discharge)	1[a,e]	Birth (before discharge)
2	1–2 months	2	2 months
3[c]	6 months	3	4 months
		4[c]	6 months (Pediarix) or 12–15 months (Comvax)

Abbreviations: HBIG, hepatitis B immune globulin; HBsAg, hepatitis B surface antigen.

[a] Recombivax HB or Engerix-B should be used for the birth dose. Comvax and Pediarix cannot be administered at birth or before age 6 weeks.
[b] Hepatitis B immune globulin (0.5 mL) is administered intramuscularly in a separate site from the vaccine.
[c] The final dose in the vaccine series should not be administered before age 24 weeks (164 days).
[d] Mothers should have blood drawn and tested for HBsAg as soon as possible after admission for delivery; if the mother is found to be HBsAg positive, the infant should receive HBIG as soon as possible but no later than 7 days of age.
[e] On a case-by-case basis and only in rare circumstances, the first dose may be delayed until after hospital discharge for an infant who weighs 2000 g or more and whose mother is HBsAg negative. When such a decision is made, an order to withhold the birth dose and a copy of the original laboratory report indicating that the mother was HBsAg negative during this pregnancy should be placed in the infant's medical record.

Reproduced with permission from Mast EE, Margolis HS, Fiore AE, et al.; Advisory Committee on Immunization Practices. A comprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). Part 1: immunization of infants, children, and adolescents. *MMWR Recomm Rep*. 2005;54(RR-16):1-31.[56]

The HBV series consists of either three or four injections, based on the type of HBV employed. In some parts of the world, a combination vaccine including vaccination against hepatitis B, diphtheria, tetanus, pertussis, and influenza is administered during the first year of life.[58]

Some parents decline HBV for their infants. This decision reflects maternal autonomy, although evidence supports the advantages of primary prevention of disease through immunization, as discussed in the *Health Promotion Across the Lifespan* chapter. However, for women who are HBsAg positive, there are distinct risks to the infant regarding development of chronic hepatitis B and potentially fatal hepatic carcinoma or cirrhosis. Thus, the ethical concerns in such situations are more complex, and the family can be best served with a full discussion of the risks and benefits associated with the disease and with vaccination.[59]

Circumcision

In the United States, circumcising male newborns became prevalent in the 1950s. A complex set of religious, cultural, and family traditions are contributed to the decision about circumcision. The type of practitioner who performs a circumcision varies. In many sites, the obstetrician is the clinician responsible for this procedure, while in other areas it is a pediatric provider or a family physician. If the reason for the procedure is religious, a cleric such as a rabbi or a midwife who also is a Mohel may perform the circumcision. Circumcision is not a core competency in midwifery education, but midwives practicing in the United States can learn and offer this procedure as an advanced procedure following the recommended steps outlined in the *ACNM Standards for the Practice of Midwifery*.

The circumcision procedure involves the surgical removal of adhesions and retraction of the foreskin covering the glans of the penis (prepuce). This procedure usually occurs in the hospital or within the first two weeks of life as a religious ceremony. Anesthesia is indicated for the newborn. Complications from circumcision are unusual but can be serious, with the most common being infection and bleeding. Although the foreskin of male newborns is rarely able to be retracted, within the first few years the foreskin can be spontaneously retracted for the vast majority of male children who are not circumcised.

The contemporary rationale for circumcision is a source of controversy. Arguments exist both for and against male circumcision, although true benefits and hazards are relatively rarely cited.[60] Proponents of circumcision note that circumcised males experience lower incidence of medical problems including cancer of the penis, urinary tract infection (UTI), and sexually transmitted infection (STI). Cancer of the penis is an unusual malignancy, especially in the cooler climates of the Northern Hemisphere. UTIs are similarly uncommon in infants. A number of studies have found a positive correlation between men who were uncircumcised and increased incidence of STIs, including human immunodeficiency virus (HIV), although other contributing factors such as socioeconomic class and sexual practices have clouded the association, especially in developed countries. Routinely circumcising newborns does prevent the small percentage of men with phimosis (inability to retract the foreskin) from developing problems with edema and inflammation of the glans, and male circumcision does not interfere with reproductive function of the penis.[61]

For some, this topic inspires passionate debate, in which male circumcision is likened to female circumcision or genital mutilation. Opponents of circumcision maintain that modern sanitary conditions in the United States diminish the need for this procedure and argue that the procedure is a painful violation of a nonconsenting infant that removes a functional body part. Parents who choose not to circumcise their sons are taught the normal anatomy of the penis and its development. As the child matures, he will be taught to retract the foreskin gently while bathing to clear any collection of smegma. Parents are counseled not to retract a foreskin forcefully, as the resultant irritation and edema may cause further adhesions.

Just as conflicting opinions have arisen in the general population regarding circumcision, so the American Academy of Pediatrics (AAP) has issued statements on this practice that have varied over the years. Based on research studies from 2005 to 2010, the most recent AAP statement notes that the health benefits of circumcision outweigh the risks and supports access

to the process for families who choose it.[61,62] A critical component of this statement is the informed desire of families. Information about circumcision is included in prenatal care, giving women and their families adequate time to make this decision. Some practices develop their own culturally sensitive documents to help individuals with this decision-making process.

Maternal Conditions That Influence Newborn Care

Women with HIV

HIV infection is a major perinatal threat. In the late 1990s, a major breakthrough occurred when a study found that treatment of newborns born to HIV-positive mothers with zidovudine (AZT, Retrovir) reduced the infants' risk of developing the infection.[63] In 2013, it was reported that a newborn with evidence of HIV shortly after birth was treated with a short course of multiple antiviral agents and later was found to be free of any infection.[64] Today, treatment of newborns born to women with HIV is recommended, but the appropriate drug, dosing, and follow-up have yet to be codified. A newborn whose mother has HIV is best treated by a pediatric provider with expertise in this area.

In the United States, it is recommended that HIV-positive women not breastfeed their infants because the virus is transmitted through breast milk. However, in areas where safe breast milk substitutes are lacking, the risks of HIV must be weighed against the risks of newborn diarrhea and nutritional deficits associated with bottle feeding; thus, the recommendation to breastfeed or not is a more complex decision in such locations.[65]

Colonization with Group B *Streptococcus*

Group B *Streptococcus* (GBS) infection is a leading cause of neonatal sepsis in the United States.[66] Approximately 1 in 100 to 200 newborns born to women who are colonized with GBS will develop newborn sepsis if the woman is not treated in labor. Because newborn sepsis has a significant risk of morbidity and mortality, efforts to prevent GBS-related newborn disease have been a standard part of maternity care in the United States since 1996.

The term infant whose mother received thorough intrapartum GBS prophylaxis—defined as 4 hours or more of penicillin, ampicillin (Principen), or cefazolin (Ancef) during labor in the hours immediately before the birth—should be observed for 24 to 48 hours; nevertheless, these newborns do not require special diagnostic testing unless they have signs or symptoms of illness.[67] Conversely, all infants who have signs of sepsis and all infants whose mothers have developed chorioamnionitis should have an evaluation and antimicrobial therapy. The 2010 algorithm published by the CDC can be found on their website. It is anticipated that the algorithm for neonatal care of newborns exposed to GBS will be updated in 2018 by the American Academy of Pediatrics.

Women with Diabetes

Women who have diabetes are at increased risk for having newborns with low or high birth weight, congenital anomalies, birth injuries, polycythemia, and hypoglycemia.[68] Newborns who are born to women who have diabetes may be either macrosomic or growth restricted, depending on the type and severity of the woman's diabetes. Newborn hypoglycemia may result from hyperinsulinemia that developed in response to intrauterine hyperglycemia from exogenous maternal glucose and diminished endogenous glucose production. This condition is

temporary for the newborn. Hypoglycemia is less common if the woman managed her diabetes well during pregnancy and had stable blood glucose levels.

Because of the risk of hypoglycemia, early feeding is important for these newborns. A blood glucose level should be obtained from these newborns during the first 2 hours of life.[69] A consultation with a member of the pediatric team is needed if any abnormal blood glucose levels are reported because intravenous fluids may be indicated.

Maternal Substance Use

The term *drug exposure*, when used in reference to a newborn, covers a wide variety of substances and situations. Many attempts have been made to quantify the effects of exposure on the fetus and newborn following in utero exposure to substances such as nicotine, alcohol, and illicit drugs. One major limitation of many of these studies is that many women who use drugs may be polydrug users. In addition, drugs of abuse often vary in terms of their additives and purity. Women who use drugs also may suffer from malnutrition, poverty, and chronic stress that can have an independent adverse effect on the fetus/newborn.

Newborns who have had significant exposure to a teratogenic or fetotoxic agent during pregnancy frequently will be small for gestational age and may have dysmorphic physical characteristics. Neonatal withdrawal symptoms include a wide spectrum of physiologic and neurobehavioral symptoms. Any infant suspected of being at risk for neonatal abstinence syndrome is observed carefully for the first 72 hours of life. Some infants may require hospitalization for as long as a week after birth.

Nicotine

Historically, nicotine inhaled via smoking has been termed a fetotoxic agent because of its relationship to low birth weight. Data now suggest that the delay in normal development associated with maternal smoking may begin during the embryonic period.[70] This phenomenon results from the effects of both carbon monoxide (leading to relative fetal hypoxia) and nicotine (leading to placental vasoconstriction). The effect on the fetus appears to be dose related and will cease if the woman stops smoking during pregnancy. Infants who are born to women who smoke are evaluated for gestational size and, if they are small for gestational age, treated accordingly.

Alcohol

Newborns born to women who abuse alcohol can suffer serious and permanent damage.[71,72] Ethanol readily crosses the placenta and the extent of fetal exposure to alcohol can be high. Damage is characterized as a continuum, with two terms being used to describe the damage: fetal alcohol syndrome (FAS) and fetal alcohol spectrum disorders (FASD). FASD encompasses FAS but also includes individuals with milder effects of alcohol.

Newborns exhibiting signs of FAS may have both physical and cognitive damage. Physical damage includes dysmorphic features that are congruent with a midline defect, which occurs early in fetal life. The manifestations of FASD are more subtle and will become obvious when

the child is older and has trouble with school or socialization. The signs of FAS may or may not be obvious during the newborn period. Damage varies with timing and dose of exposure. If physical or behavioral signs that are possibly indicative of FAS or FASD are noted, consultation with a pediatric team is necessary. These newborns need careful monitoring because of their risk for failure to thrive. Their behavioral characteristics may present a challenge for parents who are caring for them, rendering them vulnerable to abuse by caregivers.

Opioids and Other Illicit Drugs

Neonatal abstinence syndrome long has been associated with maternal use of opiates such as heroin and cocaine. More recently, neonatal withdrawal symptoms have been found among the children of women using various psychotropic drugs.[73-75] Maternal use of cocaine has been suspected as a cause of many fetal problems, most of them related to the intense vasoconstrictive properties of cocaine, which can cause placental and cord accidents. The known effects of cocaine exposure include behavioral problems that are secondary to central nervous system irritability. Cocaine-exposed newborns may be irritable, tremulous, and hard to comfort. They may favor a rigid, hyperextended posture. The manifestations of cocaine exposure are related to the amount, duration, and timing of fetal exposure. Third-trimester use is more likely to result in residual behavioral effects. Meconium is a particularly useful medium for drug testing because of the ease of specimen collection from the diaper and the ability to obtain accurate drug sampling of opiates and other prescriptive drugs that often are abused.[76] In some states and localities, a positive toxicology screen must be reported to child welfare officials.

Neonatal Abstinence Syndrome

In the case of opiates and potentially other drugs of abuse, the fetus becomes tolerant and can show evidence of severe withdrawal symptoms in utero if the mother stops using the drug during pregnancy. At birth, most newborns of women who are addicted to illicit drugs will exhibit some withdrawal symptoms.[73] These start in the first few days after birth and can persist for many weeks. If a woman used drugs by the intravenous route, her newborn will also be at risk of infection with the hepatitis and HIV viruses.

The symptoms of neonatal abstinence syndrome involve many neonatal systems, including central nervous system irritability, tremors, excess hunger and salivation, sweating, yawning, sneezing, fist sucking, and temperature regulation problems. Some infants progress to seizures and profuse vomiting and diarrhea. These newborns need to be managed by the pediatric team during an inpatient stay. Management considerations include providing a quiet, nonstimulating environment, a possibly increased need for calories and fluids, and potential need for pharmacotherapy to control symptoms. Traditionally methadone (Dolophine) has been the drug used to support withdrawal; however, buprenorphine (Buprenex) now is gaining popularity for this indication.

Newborns experiencing withdrawal need an environment that is not too stimulating. The

mother can be taught the swaddling and calming techniques. Healthcare providers have a particular obligation to help this mother identify resources that she can rely upon if she has difficulty coping with the newborn's behaviors.

Newborn Disorders

Several newborn conditions may adversely affect newborn health. One of the great challenges facing providers who care for newborns is the nonspecificity of signs and symptoms of some serious conditions. Because of the nonspecific nature, a careful history and physical examination to gather the maximum amount of data to report to pediatric caregivers is needed. The midwife practicing far from a pediatric inpatient facility should become familiar with techniques for starting a peripheral intravenous line in the neonate. If the neonate's condition deteriorates, it will be more difficult to find a vein.

Hyperbilirubinemia

As many as 50% of newborns develop clinically evident hyperbilirubinemia or jaundice.[77,78] Management of jaundice depends on whether the jaundice is determined to be physiologic or hyperbilirubinemia that is indicative of a pathophysiologic process. **Table 38-5** provides a list to help distinguish physiologic jaundice from hyperbilirubinemia.

Table 38-5 Newborn Hyperbilirubinemia

Type of Hyperbilirubinemia	Signs and Symptoms
Physiologic jaundice	Jaundice appears on days 2–4 after birth
	Total bilirubin rises slowly and peaks at day 3 or 4 of life < 13 mg/dL
Hyperbilirubinemia	Jaundice is visible during first 24 hours after birth
	Total bilirubin rises quickly, up to > 5 mg/dL /24 hours; it peaks at > 13 mg/dL
	Visible jaundice persists after one week of life
	Newborn may have risk factors such as cephalohematoma or ABO blood group or Rh (D) incompatibility, polycythemia
Breast milk failure jaundice	Jaundice appears in first week after birth
	Total bilirubin levels may rise to > 13 mg/dL
	Exclusively breastfed: feeds poorly, weight loss and signs of dehydration, infrequent urine output
	Delayed passage of meconium
Breast milk jaundice	Jaundice appears after first week of life and persists for up to 12 weeks
	Mildly elevated bilirubin levels that do not rise
	Feeds well, normal weight gain, normal urine output

Newborn physiologic jaundice is the result of bilirubin deposits in the dermis. Bilirubin levels rise during the first few days after birth, secondary to three aspects of newborn physiology:

- The newborn has more red blood cells than does an adult, and these red blood cells have a shorter lifespan. As fetal red blood cells break down so that fetal hemoglobin can be replaced with adult hemoglobin, heme catabolism produces bilirubin as a by-product.

- Bilirubin must be converted from an unconjugated (indirect) non-water-soluble form to a conjugated (direct) water-soluble form so that it can be excreted through urine and stool. The hepatic metabolism pathway that conjugates bilirubin is immature in the newborn and is not effective for a few weeks.
- Conjugated bilirubin must be broken down further by intestinal bacteria so that it can be excreted. Because the newborn has an essentially sterile gastrointestinal tract, conjugated bilirubin is reabsorbed and recirculated via the enterohepatic system more than it would be in an adult.

Thus, physiologic jaundice is the result of increased production of bilirubin at a time when elimination is delayed.

All newborns develop rising total bilirubin levels for the first few days after birth, and it is not uncommon for some bilirubin deposited in the skin to be evident as newborn jaundice. The mean peak of total bilirubin plasma levels occurs between 48 and 92 hours after birth and the average value is between 7 mg/dL and 9 mg/dL.

Some hospitals screen all newborns with serum total bilirubin levels at 1 day of age, and those infants with a level less than 5 mg/dL are not rescreened unless the clinical picture changes. Infant care practices that enhance clearance of bilirubin, such as early and frequent feedings to promote the passage of meconium, should be advocated. Delayed passage of meconium promotes reabsorption of bilirubin as part of the enterohepatic circulation. All parents should be educated about newborn jaundice and taught to assess the newborn in a well-lighted room or near a sunny window by blanching the skin to reveal the underlying color.

Hyperbilirubinemia in newborns who are equal to or more than 35 weeks' gestation at birth is defined as a total bilirubin level that is higher than the 95% on the hour-specific Bhutani nomogram. This nomogram is used in a standardized bilirubin calculator that accounts for gestational age at birth, risk factors, number of hours since birth, and bilirubin values. The bilirubin calculator is available on the Internet or as a medical app for smartphones or personal computers. The calculator determines the risk for hyperbilirubinemia and presents recommended management based on the degree of risk.

A total bilirubin level that is higher than 25 to 30 mg/dL is associated with a risk for bilirubin-induced neurologic dysfunction (BIND). This disorder occurs when bilirubin crosses the blood–brain barrier and binds to brain tissue. Unconjugated bilirubin is highly neurotoxic, and the term *acute bilirubin encephalopathy (ABE)* refers to cerebral symptoms associated with hyperbilirubinemia such as irritability, high-pitched cry, hypertonia or hypotonia, seizures, and possibly death if left untreated. The term *kernicterus* is used to describe the chronic and permanent sequelae of ABE in infants who sustain permanent cerebral damage as the result of hyperbilirubinemia. The incidence of kernicterus in full-term newborns is exceedingly rare, even in those who develop ABE. Because of the persistence of kernicterus, in 2004 the American Academy of Pediatrics recommended that all newborns be screened for elevated bilirubin prior to hospital discharge. Those with hyperbilirubinemia could have close follow-up planned. Screening for bilirubin concentration was added to the recommended elements of the newborn visit in 2017.[78]

Newborns at increased risk for hyperbilirubinemia include those who have a hemolytic

process, such as an ABO blood-type incompatibility, or accelerated red blood cells breakdown from a large cephalohematoma. Other risk factors include inherited red blood cell membrane defects, enzyme defects such as glucose-6-phosphate dehydrogenase (G6PD) deficiency, newborn sepsis, and maternal diabetes.

Breast milk jaundice and breastfeeding failure jaundice are two additional forms of newborn jaundice. Infants diagnosed with *breast milk jaundice* have mild hyperbilirubinemia that persists beyond the first week of life and slowly declines between 3 and 12 weeks of life. These infants need to be monitored but rarely need treatment; continued breastfeeding should be encouraged. Although the etiology is not clearly known, it appears some women have a substance in their breast milk that stimulates reabsorption of bilirubin from the neonate's intestine. Conversely, *breastfeeding failure jaundice* occurs secondary to exclusive breastfeeding but inadequate intake. In this case, the infant loses weight and has slower bilirubin elimination and higher levels of reabsorption via enterohepatic circulation. Breastfeeding failure jaundice is not the result of breast milk per se; rather, it is the result of too little fluid and dehydration. This form of newborn jaundice appears in the first week of life and may need specific treatment for the jaundice in addition to interventions designed to establish lactation.

Bilirubin can be measured via a central venous sample or a point-of-care transcutaneous meter, or can be estimated via physical examination. Jaundice appears first in the face and has a cephalocaudal progression. Although bilirubin levels can be visually estimated based on the level of jaundice in the newborn, visual estimation is not sufficiently predictive to be relied upon for management decisions.

The treatment for newborn jaundice is phototherapy, and in severe cases exchange transfusion. The light from the phototherapy converts bilirubin to a water-soluble form that can be excreted. The American Academy of Pediatrics has issued guidelines for newborn jaundice that, although not all evidence based, are generally followed throughout the United States, especially regarding indications for phototherapy.[79] The following symptoms may indicate that the jaundice is not physiologic and the newborn needs a more extensive medical evaluation: vomiting, lethargy, poor feeding, weight loss, hepatosplenomegaly, apnea, temperature instability, tachypnea, dark urine or urine positive for bilirubin, light-colored stools, and jaundice that persists for more than 3 weeks.

Phototherapy is an effective treatment for newborn jaundice and can be performed at home. Home phototherapy is not as effective as the technology available in the hospital, however, and one recommendation is to initiate therapy at home only when a newborn has a serum total bilirubin level 2 to 3 mg/dL lower than when phototherapy is initiated in a hospital.[80] Home therapy is less expensive than hospitalization and allows mothers and neonates to stay together, especially for breastfeeding. Occasionally a woman is unable to manage the process at home. Outside the United States, a cholesterol-lowering agent, clofibrate, has been used in conjunction with phototherapy to reduce unconjugated bilirubin more rapidly, but additional studies are needed to assess its effectiveness.[79,80]

Respiratory Conditions

Respiratory signs are the most common abnormal finding in newborns. The most common pulmonary causes of neonatal respiratory distress in term infants are pneumothorax, aspiration of meconium, neonatal pneumonia, and transient tachypnea of the newborn (**Table 38-6**). Signs of respiratory distress include tachypnea (> 60 breaths/min), nasal flaring, retractions of intercostal muscles or sternum if severe, or grunting. Cyanosis, rales, or rhonchi are possible. Central cyanosis (of the trunk, lips, tongue, and oral mucous membranes) is a sign of serious compromise of the neonate's hemoglobin oxygen saturation and requires supplemental oxygen.

Table 38-6 **Common Neonatal Respiratory Conditions**

Associated Conditions	Comments
TTN due to longer period of time transitioning from fluid-filled to air-filled lungs	TTN lasts 48–72 hours with spontaneous resolution, although the newborn may need assistance with feedings and extra oxygen. Generally these infants are monitored in the hospital until TTN resolves.
Meconium aspiration that can result in pneumonitis, hypoxia, respiratory failure, and persistent pulmonary hypertension	Requires support from a respirator and occasionally ECMO
Neonatal pneumonia caused by bacteria, viruses, or other microorganisms	Anti-inflammatory agents and supportive therapy
Pneumothorax (unilateral or bilateral), often after mechanical ventilation and overdistension	A large pneumothorax is a pediatric emergency
Congenital anomalies causing airway obstruction or pulmonary obstruction	May require surgical intervention
Sepsis	Evaluate other signs of sepsis such as temperature variations

Abbreviations: ECMO, extracorporeal membrane oxygenation; TTN, transient tachypnea of newborn.

If the newborn is having respiratory distress, continued monitoring of heart rate and respiratory rate is necessary while awaiting pediatric consultation or transport. If possible, check the newborn's blood pressure. To assess the oxygen level, use a continuous pulse oximetry sensor attached to one of the neonate's fingers or toes. This method computes arterial oxygen saturation (SaO_2), with an acceptable level being between 92% and 95%.

Newborn Infection

The newborn may have a congenital infection or may become infected with a virus or bacterium during labor, birth, or the immediate postpartum period. The viruses most likely to cause congenital infection are toxoplasmosis, parvovirus, varicella, syphilis, rubella, cytomegalovirus (CMV), and herpes. Infants with such infections are often small for gestational age. They may present with signs that include early jaundice, hepatosplenomegaly, petechiae, and palpable lymph nodes. Some viruses cause cataracts, limb or cardiac defects, microcephaly, rash, or vesicles.[81] Thrombocytopenia may be evident on a complete blood count (CBC). The visible effects of infection will vary according to the timing of fetal exposure and the severity of the infection.

Management may include isolation of the newborn from other newborns and from pregnant

healthcare workers until the virus is identified. Usually the mother and infant can be isolated together. Cord blood is saved for further testing. Historically, TORCH testing has been a routine component of the assessment of infants who are small for gestational age. TORCH is an acronym for “toxoplasmosis, other, rubella, CMV, and herpes”; the “other” category includes tests for syphilis, varicella, and parvovirus as indicated.[82] Although the presence of immunoglobulin M (IgM) antibodies in the cord blood will help to confirm a suspected viral infection, it is rare that IgM antibodies are present to aid diagnosis. Often the diagnosis of congenital infection does not become clear until the infant is older. Treatment in the newborn period is symptomatic.

Infections caused by *Staphylococcus aureus* and *Staphylococcus epidermidis* are usually acquired after birth in hospital settings. Signs of neonatal sepsis include respiratory distress, vomiting, lethargy, apnea, and irritability. Common causes of bacterial infections acquired by newborns during labor are group B beta-hemolytic *Streptococcus* and *Escherichia coli*.

Cardiac Conditions

Cardiac problems in newborns result from a variety of causes and mimic a number of other problems, especially respiratory disease and sepsis. Pulse oximetry is advocated for all infants, as described earlier, and can be used to identify such problems. Some newborns will quickly demonstrate signs of an underlying congenital defect. These defects are usually structural in nature and may affect the heart valves, the walls between the atrium and the ventricles, or the heart muscle itself.

The primary signs of heart disease include central cyanosis, murmurs, and other cardiac sounds.[83] Depending on the defect, pulses may be either bounding or diminished. Pulses can vary in intensity from the upper to lower extremities. Similarly, based on the defect, a loud murmur may be present at birth or may not be heard until 1 to 2 weeks after birth. The cyanosis of heart disease may be present over the entire body, which differentiates this condition from the peripheral cyanosis often found among normal newborns. For infants with a ventral septal defect, oxygenated blood crosses over the opening in the septum and recirculates through the lungs. In this case, cyanosis often is not present and a murmur may not be heard until the infant reaches 2 or more weeks of age as the compensatory measure of increased pulmonary vascular resistance diminishes and more blood flows from left to right. This shift is characteristic of a large defect and may be associated with a subtle onset of congestive heart failure evidenced by tachypnea, feeding problems, and failure to thrive. A parent may describe troubling symptoms during a postpartum visit or during calls about breastfeeding. In smaller ventricular septal defects, the systolic murmur is heard soon after birth but will not lead to congestive heart failure.

Some newborns will not make the transition from intrauterine to extrauterine circulation. In these neonates, the pulmonary resistance remains so high that blood flow through the newborn lungs is decreased. This relatively rare phenomenon results in persistent pulmonary hypertension (formerly called persistent fetal circulation). Signs include tachypnea, nasal flaring, and intercostal retractions. Because of the high level of resistance from the lungs, the ductus arteriosus and the foramen ovale may stay open to provide right-to-left shunting. In this

case, peripheral pulses will be bounding, the precordium will be active, and a murmur will be heard.[84]

Midwifery management of newborns with acute signs of heart disease includes immediate consultation with the pediatric team and supportive care for the resulting respiratory distress. The newborn's heart rate and respiratory rate are constantly monitored. If heart disease is suspected, the blood arterial oxygen saturation of an upper extremity can be compared to that of a lower extremity. In persistent pulmonary hypertension, the saturation is lower in the descending aorta. Warm, humidified oxygen is provided via hood at high concentrations. When there is a cardiac problem, however, even high concentrations of oxygen may not yield normal results. If the newborn is tachypneic, oral feedings are withheld. The metabolic demands of respiratory distress will predispose the newborn to hypoglycemia.

Signs of Neurologic Disease

The most common sign of neurologic compromise in the newborn period is seizure activity. There are many causes of neonatal seizures, ranging from hypoglycemia to neonatal encephalopathy.[85] Treatment is based on the etiology of the seizure.[85] Prognosis is also related to the etiology more than the seizure activity itself, unless the seizures are prolonged or frequent.[85] In rare cases, a congenital abnormality in the brain may be present. In the older newborn, seizures can be an indicator of an inborn error of metabolism.[86]

Newborns may evidence various types of seizure activity. The most common is the subtle seizure. Signs of subtle seizures include sucking motions, chewing, bicycling of limbs, drooling, apnea, deviation of the eyes, and eyelid fluttering. These seizures are evidenced as short, repetitive bursts of activity. Such repetitive behavioral patterns can be observed during a newborn examination. Because there is an association between hypoglycemia and seizure activity, it is recommended to perform a point-of-care glucose screening when seizure is suspected. Because of the risk of apnea, the newborn with possible seizure activity is carefully monitored by professionals with expertise in this area.

Current treatment for a newborn with seizures secondary to hypoxia–ischemic encephalopathy includes therapeutic hypothermia or brain cooling.[85,87] Other types of seizures (multifocal, focal) are less common in newborns. The signs of these seizures include movement of one limb with progression to other limbs or movement of both limbs on one side of the body.

Nonvisible Congenital Defects

Tracheoesophageal fistula and esophageal atresia are not visible on physical examination. However, signs such as excess salivation, respiratory distress, swallowing problems, and abdominal distension will be present. If esophageal fistula is suspected, a sterile feeding tube can be introduced into the newborn's esophagus but will not be able to be passed more than 10 to 12 centimeters.[88] In such a case, the newborn is positioned in a prone position with the head elevated and oral feedings withheld. Aspiration of the esophageal contents by feeding tube attached to a syringe helps to minimize the chance of aspiration into the lungs.

Tracheoesophageal fistula and esophageal atresia often occur together.[89]

A variety of congenital defects can lead to intestinal obstruction in the newborn. Among these are malrotation and midgut volvulus, meconium plugs, meconium ileus, Hirschsprung's disease (megacolon), and imperforate anus. The major diagnostic signs of all these anomalies are bile-stained emesis and failure to pass stool. Significant abdominal distension will be present with meconium ileus, meconium plug, and Hirschsprung's disease.[90] Newborns with these signs must be evaluated immediately by a pediatric caregiver. No oral feedings should be given if bile-stained emesis is observed. If neglected, any of these defects can lead to intestinal perforation.

Discharging the Newborn from Early Newborn Care

In hospitals, a pediatrician is usually responsible for assessing newborn readiness for care by its parents at home; however, when the birth occurs in a birth center or a home setting, the midwife makes this decision. In any setting, the midwife should be confident that the parents understand the basics of newborn care and can feed the newborn comfortably. Many hospitals have specific criteria for discharge, such as passage of urine and stool, completed physical examination, or two successful feeds.[91,92]

When the newborn is discharged from the hospital early or is leaving a birth center, follow-up assessment is recommended for 48 hours later. Parents will need assistance with arranging age-appropriate testing such as metabolic screening after discharge. Transition between providers is facilitated if new parents know that the midwife remains available for questions.

The First Golden Month: Primary Care of the Newborn

The role of the midwife during the newborn's first month of life varies markedly. In some locales, the midwife has little formal role once the newborn leaves a birthing room. In other practices, the midwife will continue care of the mother and newborn throughout the first 4 to 6 weeks after birth. These midwives may work in a collegial relationship with pediatric providers, with the well-child care gradually shifting to pediatric or family healthcare providers.

Regardless of any formal expectation regarding the role of the midwife, the reality is that many parents will call the midwife with questions related to the care and well-being of their newborn. The midwife, conscious of the special connection between mother and child, promotes family well-being through involvement in care and provision of evidence-based advice.

Well-Neonate Surveillance: Physical Care of the Newborn

Parents have many concerns regarding the physical care of their newborn. Overall, they should be reassured that there is more than one right way to approach many physical aspects of their child's care. Many midwives help mothers learn about their infants by working with the mother–newborn dyad during a home visit (**Figure 38-2**). Various common conditions about which parents may have concerns are summarized in **Table 38-7**.



Figure 38-2 Midwife illustrating care of a child with a mother.

Courtesy of Helen Varney Burst.

Table 38-7 Common Neonatal Conditions and Parental Concerns in the First Month of Life

Conditions/Questions	Comments	Treatments/Advice
Skin Conditions		
Diaper simple dermatitis	Flat, reddened areas Minimal skinfold involvement Neonate may demonstrate distress during cleaning/diaper	Gentle cleaning with water Frequent diaper changes Use of protectants (e.g., zinc oxide)

	changes	
Diaper dermatitis secondary to fungus	Usually due to *Candida albicans* Confluent erythematous lesions Often lesions remote from the main area (satellite lesions) Painful to the neonate, even when not touched	Topical antifungals (e.g., miconazole) Topical steroids (e.g., hydrocortisone)
Diaper dermatitis with secondary infection	Usually due to *Staphylococcus* or *Streptococcus* microorganisms Peeling skin Vesicles Exudates	Treatment based on type of microorganism involved
Cradle cap	Seborrheic exudates	Gently comb hair/scalp with oil (e.g., vegetable, olive) Remove exudate with shampoo Regular shampooing usually resolves issue
Erythema toxicum neonatorum and milia	Various eruptions, small vesicles or generalized rash	These dermatologic conditions are common in the first week of life and will spontaneously resolve. No treatment is necessary, and the condition does not appear to be painful to the newborn. Parents can be reassured that this is a normal transient finding in newborns.
Oral Conditions		
Thrush	Adherent white plaques on tongue, gums, and/or palate	Oral antifungals Also rule out mastitis if the mother is breastfeeding
Nasal discharge	Discharge may be apparent Occasional periods of irregular breathing Crusting around nares may or may not be present	Counsel parents to avoid bulb syringes; they can cause trauma and edema Saline drops Differentiate between nasal congestion and apnea (apnea is associated with low heart rate, pauses of 20 seconds or more)
Behavior		
“Fussiness”	Crying “Colicky” behavior Difficult to console Reasons unknown and likely to be unique to individual parent–newborn dyad	Multiple suggestions to console a baby— although no research that one method is better than another: Swaddling Talk with baby *en face* Cuddle, reposition, walk, or move with baby Attempt to feed or give pacifier Reduce environment sensory stimulation *Never shake a baby!*
Sleep		
Timing of sleep	Usually sleep 16–18 hours in first few days Variation more profound as baby grows, ranging from as few as 8 hours or as much as almost 20 hours by 4 weeks	Reassure parents that newborns do not necessarily have days and nights confused Most likely will take 12 weeks at least to start sleeping more during nighttime and may take 3–5 months based on melatonin and cortisol production

	Newborns are less governed by circadian rhythms than adults Sleep is associated with feeding; more likely to sleep during day	
Safe sleep	Newborns who sleep on their backs are less likely to experience sudden infant death syndrome (SIDS) Originally part of the national campaign “Back to Sleep”; now termed “Safe Sleep”	Newborns should be placed on their backs for sleep Should have “tummy time” or interactions when on abdomen only when the newborn is awake
Feeding[a]		
Adequate feeding	Newborn should feed multiple times daily (e.g., approximately every 2–4 hours) May lose 5–7% of birth weight in first few days of life	Proxy measures for adequate intake include: Weight gain at end of first week or beginning of the second week (usually 150–200 g per week) Stools and urine in diapers at least 1–3 times during each of the first few days of life Once lactation established, 3–4 stools and 5–6 wet diapers every day If difficult to ascertain if diaper is wet, place a soft tissue inside the absorbent diaper If no bowel movement or urine within 24–48 hours, consult pediatric provider
Car Safety		
Car seats	Newborns should travel in rear-facing car seats	Never leave a baby alone in a car Choose a car seat that meets U.S. safety standards (will be marked on box and tag). Because multiple car seats and models of cars exist, have the car seat installed by or verified by a car seat technician.

[a] More details can be found in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

For example, an infant does not need a full bath daily. Newborns need to have their heads and diaper areas sponged whenever appropriate using a mild, nondeodorized soap, with the area then being completely dried. A full bath can be given occasionally when the parent has the time to make it a relaxing event for the newborn. Parents should be urged never to leave a newborn unattended in the bath. The soap, towels, shampoo, and clean clothes can all be organized before starting the bath. Slip-proof bath mats are inexpensive and helpful.

Parents may have preferences for which type of diaper to use—paper disposable or cloth—and the best type to use has not been determined. A few parents will choose to wash their child’s diapers themselves. If so, recommend that dirty diapers be presoaked using bleach and detergent and washed twice in very hot water without packing the washer too full, to ensure that bacteria are destroyed.

Family members may encourage new parents to dress infants in excessive layers of clothing. A general rule of thumb is to clothe the infant with as many layers as other persons in the room are wearing, plus one layer. Because infants cannot sweat effectively, symptoms of overheating are mainly a red skin color, irritability, and body warmth. Eventually the overheated infant will

appear lethargic. Infants also cool rapidly through inspired cold air. When taken outside on windy cold days, newborns should have a nonrestrictive covering near their faces.

From the earliest moments at home with a newborn, parents should evaluate the environment for safety risks. In the first months of life, these risks are primarily related to falling or becoming stuck in small places such as between the bars on the sides of cribs. Parents should also consider removing soft pillows, mobiles, and toys that may lead to suffocation from the site in which the newborn sleeps.

If parents choose to use a pacifier, the pacifier should be cleaned regularly and checked for signs of dirt or mold. Most pacifiers today have a design that promotes proper development of the muscles of the mouth. Pacifiers should never be placed on a cord around the baby's neck because of the risk of strangulation. Some newborns do not accept a pacifier, so parents devise other methods to help the newborn become calm (walking the baby, distracting the baby, encouraging thumb sucking).

When an infant sucks on a bottle for a prolonged period of time, the residual sugars from the milk or juice may contribute to childhood dental caries, also known as "baby bottle mouth." The accumulation of milk in the back of the oropharynx may lead to ear infections as well. Therefore, prolonged bottle feeding, especially by propping a bottle, should never be used to calm an infant.

Parents also need to recognize situations in which immediate pediatric care is needed for the newborn. **Table 38-8** lists some situations in which a pediatric provider should be consulted promptly.

Table 38-8 **Neonatal Signs and Symptoms Requiring Prompt Parental Reporting/Pediatric Referral**[a]

Danger Signs	Description
Temperature instability	Axillary temperature > 37.4°C (99.3°F) or < 36.5°C (97.5°F)
	Tachypnea (> 60 breaths/min) for > 60 seconds
	Pauses > 20 seconds (periodic apnea)
Abnormal respirations	Regular retractions, wheezing, grunting
Infection of umbilicus	Odor, drainage, or bleeding from umbilical stump
Infection of eyes	Purulent discharge from eyes
Jaundice	Icteric changes of eyes, chest, or extremities
Gastrointestinal signs	Projectile vomiting
Diarrhea	Bile-stained vomit
	No stools/no urination since birth
	Taut, swollen abdomen
	> 6 stools per 24 hours/bloody or very watery stools
General signs of illness	Cough
	Excessive number of stools or watery stools
	Poor muscle tone
	Sleepiness/refusal to feed/inability to rouse
	Poor suck
	Neurologic signs such as bulging fontanelle, irritability, abnormal reflexes

[a] This list is not comprehensive. Midwives who provide well-baby care develop relationships with local pediatric providers to learn preferred consultation routines.

Well-Neonate Care Visits

When a midwife is the provider for a newborn during the first 28 days of life, the neonate may be seen in an ambulatory setting, often at the same time the woman is seen for a postpartum examination. The purpose of this visit is fourfold: (1) to monitor normal growth and development, (2) to identify symptoms of disease, (3) to offer screening measures, and (4) to educate and support the parents. When a parent brings a newborn in for a well-child visit, every effort should be made to keep the infant from exposure to others, especially anyone who is ill. Whenever possible, the parents should be escorted into an examination room as soon as they arrive with their child.

The visit starts with a brief interview of the parent(s) or primary caregiver. Particular attention should be paid to unresolved problems related to the birth or the immediate care of the newborn following birth. The midwife should assess the well-being of the parents and look for signs of depression or inability to cope with the demands of the newborn. Inquiries should be made about help in the home, sibling reactions, and reaction of other relatives to the newborn. Particular emphasis should be paid to the infant's feeding patterns, levels of alertness, bowel and bladder patterns, and crying patterns.

A complete physical examination is performed. During the physical examination, the midwife has an opportunity to observe the parent–newborn relationship. Observing a feeding can provide valuable insight into the parental–newborn relationship and parenting knowledge.

The greatest part of the initial well-child visit should be spent soliciting parental concerns and offering guidance and anticipatory advice. Any midwife providing well-child care should have a clear relationship established for pediatric consultation or referral of any infant presenting with signs or symptoms of illness.

Conclusion

Although midwives are not commonly viewed as primary pediatric providers, they provide the first neonatal assessment as they give the newborn to its mother. Ongoing assessment during the third stage of labor gives the midwife an important role during the first Golden Hour. Even in areas where pediatric care quickly is assumed by other professionals, the relationship between a midwife and a woman and her family often means that as questions arise about the infant, the midwife may be the first person to be called. Through their acceptance of this responsibility, midwives have an integral role in caring for the health of society's future generations.

Resources

Organization	Description	Webpage
American Academy of Pediatrics (AAP)	This site contains policy statements and practice guidelines from the American Academy of Pediatrics, such as guidelines for delayed cord-clamping and breastfeeding.	http://www.aappublications.org/search/toc_section%3AFrom%20the%20American%2 date?see_more_page=1&see_more_page_title=AAP%20Policy
Office on Women's Health (OWH)	This website contains information on newborn care for new parents, including bathing, feeding, car seat safety, circumcision care, and SIDS prevention. It may also be useful for students who have recently cared for newborns.	https://www.womenshealth.gov/pregnancy/childbirth-and-beyond/newborn-care-and-
U.S. Department of Health and Human Services (DHHS)	Newborn Screening Programs: Recommended uniform screening panel.	https://www.hrsa.gov/advisorycommittees/mchbadvisory/heritabledisorders/recomm
Global Health Media	Training videos on neonatal breathing difficulties and umbilical cord infections. These training videos are based on standards of care described in *Care of the Newborn Reference*	https://youtu.be/m6MBM0pylpA?list=PLXeu9kWMOoFLMXFrdo67Xx06HiCa3Q6Vv

	Manual, Save the Children, 2004; *Managing Newborn Problems*, World Health Organization, 2003; and *Integrated Management of Childhood Illnesses Chart Booklet*, World Health Organization, 2011.	
National Newborn Screening and Global Resource Center	This agency has information on all the state newborn screening programs, including contact information and details about each state's program.	http://genes-r-us.uthscsa.edu
YouTube	This site contains a half-dozen videos showing infant car seats and their safe use. It is a resource for parents and midwives unfamiliar with infant car carriers.	https://www.youtube.com/watch?v=87wknW5t2Zk

References

1. American College of Nurse-Midwives. *Core Competencies for Basic Midwifery Practice*. Silver Spring, MD: American College of Nurse-Midwives; 2012.
2. Sharma D, Pradeep Sharma P, Sweta Shastri S. Golden 60 minutes of newborn's life: Part 2: term neonate. *J Matern Fet Neonatal Med*. 2016;29:1-6.
3. Mercer JS, Erickson-Owens DA, Graves B, Haley MM. Evidence-based practices for the fetal to newborn transition. *J Midwifery Womens Health*. 2007;52(3): 262-272.
4. Moore ER, Bergman N, Anderson G, Medley N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;5:CD003519. doi:10.1002/14651858.CD003519.pub4.
5. American Academy of Pediatrics. Delayed umbilical cord clamping after birth. *Pediatrics*. 2017;139(6). doi:10.1542/peds.2017-0957.
6. World Health Organization. *Guideline: Delayed Umbilical Cord Clamping for Improved Maternal and Infant Health and Nutrition Outcomes*. Geneva, Switzerland: World Health Organization; 2014.
7. Committee on Obstetric Practice. Committee Opinion No. 684: delayed umbilical cord clamping after birth. *Obstet Gynec* 2017;129(1):e5-e10.
8. American College of Nurse-Midwives. *Position Statement: Delayed Umbilical Cord Clamping*. Washington, DC: American College of Nurse Midwives; 2014. Available at: http://www.midwife.org/ACNM/files/ACNMLibraryData/UPLOADFILENAME/000000000290/Delayed-Umbilical-Cord-Clamping-May-2014.pdf Accessed September 28, 2017.
9. McDonald SJ, Middleton P, Dowsell T, Morris P. Effect of timing of umbilical cord clamping of term infants on maternal a neonatal outcomes. *Cochrane Database Syst Rev*. 2013;2:CD004074. doi:10.1002/14651858.CD004074.pub3.
10. Mercer JS, Erickson-Owens DA, Collins J, Barcelos MO, Parker AB, Padbury JF. Effects of delayed cord clamping on residual placental blood volume, hemoglobin and bilirubin levels in term infants: a randomized controlled trial. *J Perinatol*. 2017;37(3):260-264.
11. Katheria AC, Lakshminrusimha S, Rabe H, McAdams R, Mercer JS. Placental transfusion: a review. *J Perinatol*. 2017;37(2):105-111.
12. Rabe H, Diaz-Rossello JL, Duley L, Dowswell T. Effect of timing of umbilical cord clamping and other strategies to influence placental transfusion at preterm birth on maternal and infant outcomes. *Cochrane Database Syst Rev*. 2012;8:CD003248. doi:10.1002/14651858.CD003248.pub3.
13. Lunze K, Blood D, Jamison D, Hamer D. The global burden of neonatal hypothermia: systemic review of a major challeng for newborn survival. *BMC Med*. 2013;11(24). Available at: http://www.biomedcentral.com/1741-7015/11/24. Accessed July 10, 2017.
14. World Health Organization. *Thermal Control of the Newborn: A Practical Guide*. Geneva, Switzerland: Maternal and Safe Motherhood Programme, Division of Family Health, World Health Organization; 1993.
15. Haninger NC, Farley CI. Screening for hypoglycemia in healthy term neonates. *J Midwifery Womens Health*. 2001;46:292-301.
16. Hay WW Jr, Raju TN, Higgins RD, Kalhan SC, Devaskar SU. Knowledge gaps and research needs for understanding an treating neonatal hypoglycemia: workshop report from Eunice Kennedy Shriver National Institute of Child Health and Human Development. *J Pediatr*. 2009;155:612-617.
17. Thompson-Branch A, Havranek T. Neonatal hypoglycemia. *Pediatr Rev*. 2017;38(4):147-157.
18. Folk L. Guide to capillary heelstick blood sampling in infants. *Adv Neonatal Care*. 2007;7(4):171-178.
19. Janes M, Pinelli J, Landry S, Downey S, Paes B. Comparison of capillary blood sampling using an automated incision device with and without warming the heel. *J Perinatol*. 2002;22(2):154-158.
20. Yin T, Yang L, Lee TY, Li CC, Hua YN, Liaw HH. Development of atraumatic heel-stick procedures by combined treatment with non-nutritive sucking, oral sucrose, and facilitated tucking: a randomized, controlled trial. *Int J Nurs Stud*. 2015;52(8):1288-1299.
21. Morrow C, Hidinger A, Wilkinson-Faulk D. Reducing neonatal pain during routine heel lance procedures. *Am J Matern Child Nurs*. 2010;35(6):346-354.
22. Centers for Disease Control and Prevention. Sexually transmitted infection treatment guidelines, 2015. *MMWR*. 2015;24(16):55-56, 66-67.
23. Puckett RM, Offringa M. Prophylactic vitamin K for vitamin K deficiency bleeding in neonates. *Cochrane Database Sys Rev*. 2000;4:CD002776. doi:10.1002/14651858.CD002776.
24. Van Winckel M, De Bruyne R, Van De Velde S, Van Biervliet S. Vitamin K: an update for the paediatrician. *Eur J Pediat* 2009;168(2):127-134.
25. Phillippi J, Holley S, Morad A, Collins M. Prevention of vitamin K deficiency bleeding. *J Midwifery Womens Health*. 2016; 61(5):632-636.
26. Skarsgard ED. Management of gastroschisis. *Curr Opin Pediatr*. 2016;28(3):363-369.

27. DeJong P, Adams N, Mann R, Polley J, Girotto J. Management of lumbosacral myelomeningocele. *Eplasty*. 2016;16:ic51.
28. Tovar J. Congenital diaphragmatic hernia. *Ophanet J Rare Dis*. 2012;7(1). doi:10.1186/1750-1172-7-18.
29. Karahanoglu E, Kasapoglu T, Ozdemirci S, et al. Risk factors for clavicle fracture concurrent with brachial plexus injury. *Arch Gynecol Obstet*. 2016;293(4):783-787.
30. Yan LJ. Neonatal brachial plexus palsy: management and prognostic features. *Semin Perinatol*. 2014;38(4):222-234.
31. Doumouchtsis SK, Arulkumaran S. Head injuries after instrumental vaginal deliveries. *Curr Opin Obstet Gynecol*. 2006;18(2):129-134.
32. Kurth E, Kennedy HP, Spichiger E, Hösli I, Stutz EZ. Crying babies, tired mothers: what do we know? A systematic review. *Midwifery*. 2011;27(2):187-194.
33. Blackburn S, Bakewell-Sachs S. *Understanding the Behavior of Term Infants*. White Plains, NY: March of Dimes Birth Defects Foundation. Available at: http://www.marchofdimes.com/nursing/modnemedia/othermedia/states.pdf. Accessed July 12, 2017.
34. Winberg J. Mother and newborn baby: mutual regulation of physiology and behavior: a selective review. *Dev Psychobiol*. 2005;47(3):217-222.
35. Hotelling BA. Newborn capabilities: parent teaching is a necessity. *J Perinat Educ*. 2004;13(4):43-49.
36. Brazelton TB, Nugent JK. *Neonatal Behavioral Assessment Scale*. 4th ed. London, UK: Mac Keith Press; 2011.
37. Povedrano MCA, Noto IS, Pinhiero MDS, Guinsburg R. Mothers' perceptions and expectations regarding their newborn infants: the use of Broussard's Neonatal Perception Inventory. *Rev Paul Pediatr*. 2011;29(2):239-244.
38. Szymanska M, Schneider M, Chateau-Smith C, Nezelof S, Vuillez-Coady L. Psychophysiological effects of oxytocin on parent–child interactions: a literature review on oxytocin and parent–child interactions. *Psychiatry Clin Neurosci*. June 2, 2017. [Epub ahead of print]. doi:10.1111/pcn.12544.
39. Lavender T, Bedwell C, Roberts SA, et al. Randomized, controlled trial evaluating a baby wash product on skin barrier function in healthy, term neonates. *J Obstet Gynecol Neonatal Nurs*. 2013;42(2):203-214.
40. Ness MJ, Davis DMR, Care WA. Neonatal skin care: a concise review. *Int J Dermatol*. 2013;52:14-22.
41. Blume-Peytavi U, Hauser M, Stamatas GN, Pathirana D, Garcia Bartels N. Skin care practices for newborns and infants: review of the clinical evidence for best practices. *Pediatr Dermatol*. 2012;29(1):1-14.
42. Sinha A, Sazawal S, Pradhan A, Ramji S, Opiyo N. Chlorhexidine skin or cord care for prevention of mortality and infections in neonates. *Cochrane Database Syst Rev*. 2015;3:CD007835. doi:10.1002/14651858.CD007835.pub2.
43. DeLuca J, Zanni KL, Bonhomme N, Kemper AR. Implications of newborn screening for nurses. *J Nurs Scholar*. 2013;45(1):25-33.
44. Centers for Disease Control and Prevention. CDC grand rounds: newborn screening and improved outcomes. *MMWR*. 2012;61(21):390-393.
45. Sweetman L, Millington DS, Therrell BL, et al. Naming and counting disorders (conditions) included in newborn screening panels. *Pediatrics*. 2006;117(5 pt 2):S308-S314.
46. Berry S. Newborn screening. *Clin Perinatol*. 2015;42(2): 441-453.
47. Ross L. Ethical and policy issues in newborn screening of children for neurologic and developments disorders. *Pediatr Clin North Am*. 2015;62(3):787-798.
48. Andermann A, Blancquaert I, Beauchamp S, Costea I. Guiding policy decisions for genetic screening: developing a systematic and transparent approach. *Public Health Genomics*. 2011;14(1):9-16.
49. Lewis MH, Goldenberg A, Anderson R, Rothwell E, Botkin J. State laws regarding the retention and use of residual newborn screening blood samples. *Pediatrics*. 2011;127(4):703-712.
50. Kemper AR, Kus CA, Ostrander RJ, et al.; U.S. Secretary of Health and Human Services' Advisory Committee on Heritable Disorders in Newborns and Children. Implementing point-of-care newborn screening. 2012. Available at: http://www.hrsa.gov/advisorycommittees/mchbadvisory/heritabledisorders/recommendations/correspondence/implementpo Accessed March 24, 2013.
51. Wolff Melnyk BM, Grossman DC, Chou R, et al. USPSTF perspective on evidence-based preventive recommendations fo children. *Pediatrics*. 2012;130(2):e399-e407.
52. Wolff R, Hommerich J, Riemsma R, Antes G, Lange S, Kleijnen J. Hearing screening in newborns: systematic review of accuracy, effectiveness, and effects of interventions after screening. *Arch Dis Child*. 2010;95(2):130-135.
53. Ewer AK. Review of pulse oximetry screening for critical congenital heart defects in newborn infants. *Curr Opin Cardiol*. 2013;28(2):92-96.
54. Ewer AK, Middleton LF, Furnston AT, et al. Pulse oximetry screening for congenital heart defects in newborn infants. *Lancet*. 2011;378:785-794.
55. Thangaratinam S, Brown K, Zamora J, Khan KS, Ewer AK. Pulse oximetry screening for critical congenital heart defects in asymptomatic newborn babies: a systematic review and meta-analysis. *Lancet*. 2012;379(9835):2459-2464.
56. Mast EE, Margolis HS, Fiore AE, et al.; Advisory Committee on Immunization Practices. A comprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP). Part 1: immunization of infants, children, and adolescents. *MMWR*

Recomm Rep. 2005;54(RR-16):1-31.
57. Schillie SF, Murphy TV. Seroprotection after recombinant hepatitis B vaccination among newborn infants: a review. *Vaccine*. 2013;31(21):2506-2516.
58. Bar-On ES, Goldberg E, Hellmann S, Leibovici L. Combined DTP-HBV-HIB vaccine versus separately administered DTP-HBV and HIB vaccines for primary prevention of diphtheria, tetanus, pertussis, hepatitis B and *Haemophilus influenzae* B (HIB). *Cochrane Database Syst Rev*. 2012;4:CD005530. doi:10.1002/14651858.CD005530.pub3.
59. Isaacs D, Kilham HA, Alexander S, Wood N, Buckmaster A, Royle J. Ethical issues in preventing mother-to-child transmission of hepatitis B by immunisation. *Vaccine*. 2011;29(37):6159-6162.
60. Mielke R. Counseling parents who are considering newborn male circumcision [erratum, *J Midwifery Womens Health*. 2014;59(2):225]. *J Midwifery Womens Health*. 2013;58(6):671-682.
61. American Academy of Pediatrics, Task Force on Circumcision. Male circumcision. *Pediatrics*. 2012; 130(3):e756-e785.
62. American Academy of Pediatrics, Task Force on Circumcision. Circumcision policy statement. *Pediatrics*. 2012;130(3):585-586.
63. Sperling RS, Shapiro DE, Coombs RW, et al. Maternal viral load, zidovudine treatment, and the risk of transmission of human immunodeficiency virus type 1 from mother to infant. Pediatric AIDS Clinical Trials Group Protocol 076 Study Group. *N Engl J Med*. 1996;335(22):1621-1629.
64. Cohen J. HIV/AIDS: early treatment may have cured infant of HIV infection. *Science*. 2013;339(6124):1134.
65. World Health Organization. *HIV and Infant Feeding*. Geneva, Switzerland: World Health Organization; 2010.
66. Verani JR, Schrag SJ. Group B streptococcal disease in infants: progress in prevention and continued challenges. *Clin Perinatol*. 2010;37(2):375-392.
67. Verani JR, McGee L, Schrag SJ; Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention. Prevention of perinatal group B streptococcal disease: revised guidelines from CDC, 2010. *MMWR Recomm Rep*. 2010;59(RR-10):1-36.
68. Mitanchez D, Yzydorczyk C, Siddeek B, Boubred F, Benahmed M, Simeoni U. The offspring of the diabetic mother: short· and long-term implications. *Best Pract Res Clin Obstet Gynecol*. 2015;29(2):256-269.
69. Maayan-Metzger A, Lubin D, Kuint J. Hypoglycemia rates in the first days of life among term infants born to diabetic mothers. *Neonatology*. 2009;96(2):80-85.
70. Fréour T, Dessolle L, Lammers J, Lattes S, Barrière P. Comparison of embryo morphokinetics after in vitro fertilization–intracytoplasmic sperm injection in smoking and nonsmoking women. *Fertil Steril*. 2013;99(7):1944-1950.
71. Pruett D, Waterman EH, Caughey AB. Fetal alcohol exposure: consequences, diagnosis, and treatment. *Obstet Gynecol Surv*. 2013;68(1):62-69.
72. Warren KR, Hewitt BG, Thomas JD. Fetal alcohol spectrum disorders: research challenges and opportunities. National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism. Available at: http://pubs.niaaa.nih.gov/publications/arh341/4-14.htm. Accessed March 29, 2013.
73. McQueen K, Murphy-Oikonen J. Neonatal abstinence syndrome. *N Engl J Med*. 2016;375(25):2468-2479.
74. Källén B, Reis M. Neonatal complications after maternal concomitant use of SSRI and other central nervous system activ drugs during the second or third trimester of pregnancy. *J Clin Psychopharmacol*. 2012;32(5):608-614.
75. Jevitt C, Rowe W. Perinatal drug dependency disorders. In: Csiernik R, Rowe W, eds. *Responding to the Oppression of Addiction: Canadian Social Work Perspectives*. 3rd ed. Toronto, Canada: Canadian Scholars' Press; 2017:95-108.
76. Launiainen T, Nupponen I, Halmesmäki E, Ojanperä I. Meconium drug testing reveals maternal misuse of medicinal opioid among addicted mothers. *Drug Test Anal*. 2013;5(7):529-533.
77. Muchowski KE. Evaluation and treatment of neonatal hyperbilirubinemia. *Am Fam Physician*. 2014;89(11):873-878.
78. American Academy of Pediatrics, Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newbor infant 35 or more weeks of gestation. *Pediatrics*. 2004;114:297-316.
79. Malwade US, Jardine LA. Home versus hospital-based phototherapy for the treatment of non-haemolytic jaundice in infants at more than 37 weeks' gestation. *Cochrane Database Syst Rev*. 2014;10(6):CD010212. doi:10.1002/14651858.CD010212.pub2.
80. Gholitabar M, McGuire H, Rennie J, Manning D, Lai R. Clofibrate in combination with phototherapy for unconjugated neonatal hyperbilirubinaemia. *Cochrane Database Syst Rev*. 2012;12:CD009017. doi:10.1002/14651858.CD009017.pub2.
81. de Jong EP, Vossen AC, Walther FJ, Lopriore E. How to use neonatal TORCH testing. *Arch Dis Child Educ Pract Ed*. 2013;98(3):93-98.
82. Neu N, Duchon J, Zachariah P. TORCH infections. *Clin Perinatol*. 2015;42(1):77-103.
83. Fillips DJ, Bucciarelli RL. Cardiac evaluation of the newborn. *Pediatr Clin North Am*. 2015;62(2):471-489.
84. Jain A, McNamara PJ. Persistent pulmonary hypertension of the newborn: advances in diagnosis and treatment. *Semin Fetal Neonatal Med*. 2015;20(4):262-271.
85. American College of Obstetricians and Gynecologists, Task Force on Neonatal Encephalopathy. *Neonatal Encephalopathy and Neurologic Outcome*. 2nd ed. Washington, DC: American College of Obstetricians and Gynecologists; 2014.

86. Campistol J, Plecko B. Treatable newborn and infant seizures due to inborn errors of metabolism. *Epileptic Disord*. 2015;17(3):229-242.
87. Jacobs SE, Berg M, Hunt R, Tarnow-Mordi WO, Inder TE, Davis PG. Cooling for newborns with hypoxic ischaemic encephalopathy. *Cochrane Database Syst Rev*. 2013;1:CD003311. doi:10.1002/14651858.CD003311.pub3.
88. Wessel LM, Fuchs J, Rolle U. The surgical correction of congenital deformities: the treatment of diaphragmatic hernia, esophageal atresia and small bowel atresia. *Dtsch Arztebl Int*. 2015;112(20):357-364.
89. Pinheiro PF, Simões e Silva AC, Pereira RM. Current knowledge on esophageal atresia. *World J Gastroenterol*. 2012;28;18(28):3662-3672.
90. Bergeron KF, Silversides DW, Pilon N. The developmental genetics of Hirschsprung's disease. *Clin Genet*. 2013;3(1):15-22.
91. Bentz W. Hospital stay for healthy term newborn infants: American Academy of Pediatrics policy statement. *Pediatrics*. 2015;135(5):948-953.
92. American Academy of Pediatrics, Committee on Practice and Ambulatory Medicine; Academic Pediatric Association, Bright Futures Periodicity Schedule Workgroup. 2017 recommendations for preventative pediatric health care. *Pediatrics*. 2017;139(4):e20170254.

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